

Salomone Di Saverio · Fausto Catena · Luca Ansaloni
Federico Coccolini · George Velmahos *Editors*

Acute Care Surgery Handbook

Volume 2

Common Gastrointestinal
and Abdominal Emergencies

Foreword by Kenneth Mattox



WORLD SOCIETY OF
EMERGENCY SURGERY

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Springer

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With deep gratitude I'd like to dedicate this book to the memory of my father, Tito, who recognized my inclinations early on, and who encouraged and supported me in my pursuit of a medical and surgical career. His constant presence in my youth is for me still a model for how to lead my own life. I also wish to thank my mother Gabriella, who has always been the beacon of light guiding me morally and culturally. She remains my mentor in logic and the humanities, sharing the wisdom of her beloved Greek and Latin masters. Last but not least, I am grateful to and dedicate this book to my devoted wife Omeshnie, who is constantly supporting me with patience and love

Salomone Di Saverio

To my parents who served as my life's springboard and to my wife and children who serve as my life's compass

George Velmahos

To my family that tolerates me and my job every day...

Fausto Catena

To my wife Anna

Federico Coccolini

Foreword

This section is called a “fore” “word” meaning editorial comments made about the book, usually by someone who is *not* an editor or author, to give their impression as to what this book is all about. Finally, this honor (and responsibility) is often given to a more senior person in the same field of the editors. In the case of this foreword, each of these traditional historical tasks is fulfilled. And, the author of this foreword considers this to be a unique and honored request, and one which I am humbled to be fill the responsibility to read the whole book, all of its chapters, and to make meaningful reflections on the purpose of this book, make subtle observations, and reflect on what might be missing from the specific chapters included in this book.

This is a fascinating collection of chapters ranging from emergencies of the esophagus, the gastrointestinal tract, including the rectum, and mesenteric ischemia. This book reflects what is undoubtedly the modern thinking in regard to the surgical, GI endoscopic, and radiologist approach to the diagnosis and management of acute surgical conditions of the above organs. In this book, and in today’s best surgical practice, this management is not always operative, and often such standard therapy is more often than not expectant and nonoperative. Such a modern contemporary approach is the antithesis of the training and practice of many of the surgeons of the 1950–1990 time frames, prior to the advent of CT, MRI, ultrasound, IR, advanced procedural endoscopy, minimally invasive laparoscopic surgery, endoluminal suturing, stents, and NOTES. As I

read the chapters, I reflected upon my own medical student and residency training during the years 1960–1973, where the emphasis was on plain abdominal x-rays, palpation, auscultation, percussion, and repeated abdominal examination. Textbooks were composed of very little statistical analysis of treatment modes and contained multiple drawings of the authors' favored procedure, often bearing that surgeons named operation. This book, happily, justifies many of the alternate approaches by presenting evidence-based comparative studies for the reader to analyze.

Observe the title of this book – *Acute Care Surgery*. Sixty years ago, most “surgical” textbooks were very similar and included the width and breath of a surgical practice. The body of knowledge in surgery be it acute, cancer, infections, trauma, or anatomy and physiology was relatively small. Regardless of the modifying words around the word, “surgery,” most surgery textbooks read and looked quite identical. Surgical textbooks began to focus on either specific organs, etiologies, regions of the body, or techniques of surgery, such as breast, colon, cancer, critical care, trauma, burns, cardiac, thoracic, foregut, colon and rectal, pediatric, and on and on. Beginning about 10–15 years ago, surgeons around the world were attempting to define the body of knowledge which requires emergency, urgent, and acute intervention. Turf wars over the exact wording of this product line broke out. Initially “trauma” and to some extent, “emergency medicine” filled the need for textbooks which focused on the areas where acute care was most concentrated. In Europe, the term, “emergency surgery” emerged, while in the United States, the term “acute care surgeon” was selected, although the curricula of these seemingly similar approaches were basically identical. In Japan and in other Asian countries, the terms, both acute and emergency and trauma surgery were often used almost interchangeably or in combination. In each of these areas, both textbooks and specialty societies were created with the favored

descriptive words were those preferred by the organizing leaders of those societies. This lack of international or even regional agreement contributed to the ongoing confusion of just what was the best vernacular to be used for what used to be merely, “general surgery,” has led to some difficulty for “acute care surgeons” to construct an exact “body of knowledge” which encompasses this emerging discipline. For this Volume 2, the authors and editors have focused on the gastrointestinal tract. One might consider that the acute vascular emergencies in the vena cava, iliac veins, abdominal aorta, renal arteries, and portal vein will also be seen emergently by the acute care surgeon. In many locations, the acute care surgeon serves as the diagnostic and operative gatekeeper of the vascular surgeon. Likewise, the acute care surgeon often could benefit from a more detailed description of both laparoscopic and open common duct surgical procedures than appear in these chapters.

The field of “acute care surgery” is rapidly developing a critical mass of surgeons who have claimed this to be their area of practice. Textbooks and surgical journals are appearing to be the written resource of the fundamentals and the research reporting archives of the knowledge and the craft of this surgical discipline. This textbook is one of those resources. Both the trainee and the practitioner of acute care surgery will find this textbook useful and a ready resource for current approaches to surgical emergencies.

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Preface

The World Society of Emergency Surgery (WSES) was established in 2007 and its aim was clearly declared: “The overall goals include the promotion of the specialty of emergency surgery as part of the emerging discipline of acute care surgery via academic exchange in an effort to further training and education as well as translational research in the specialty.”

Since 2011, the core group of Acute Care and Trauma Surgeons, founder members of World Society of Emergency Surgery, had the feeling of a strong need for improving education in the field of Acute Care and Trauma surgery, especially for the younger surgeons or any doctors and professionals approaching for the first time this discipline and the complex management of trauma and Acute Care (nontrauma) patients.

We have therefore had the idea of writing initially a book of *Trauma Surgery*, aiming to offer a practical manual of procedures, techniques, and operative strategies, which was published 2 years ago.

Following this preliminary and successful project, we have decided to proceed further with the project of a comprehensive *Acute Care Surgery* manual, covering the whole aspects of the treatment of Acute Surgical patients, with a worldwide perspective. In different nations and continents the emergency surgical care may vary widely. Being a group of World Emergency Surgeon, we provide suggestions and skills that are valid and therefore can be used everywhere, as well as give a picture of several different options and perspective in Acute Care surgery.

After more than a year of hard work, it is now with great pleasure that we are announcing the completion of our further ambitious project of an *Acute Care Surgery* manual, where most of the renowned Acute Care surgeons and physicians from all over the world have made an appreciated and highly valuable contribution, with the intent not to merely describe in academic fashion the most recent surgical techniques, but rather to suggest the best surgical and/or endoscopic and/or interventional radiology strategies, with the final of keeping the things simple but effective when in treating a patient in Acute Care setting. The contributing professionals are herewith sharing their expertise for achieving a wise clinical judgment and good common sense. This manual represents a real “vademecum,” especially for young physicians and trainees, with the specific aim of giving a fresh view and practical suggestions for best managing Acute patients and improving the skills of their treating surgeons and physicians.

This Volume two of the manual is focused on abdominal and GI emergencies in Acute Care surgery covering the most common diseases of the whole area and offering a careful description of diagnostic procedures, surgical techniques, and nonoperative management. This practical and complete guide stems from the partnership and collaboration between the members of World Society of Emergency Surgery (WSES) and other internationally recognized experts in the field; its aim is to provide general surgeons as well as emergency physicians, gastroenterologists, professionals from many other specialties, residents, and trainees with a complete and up-to-date overview of the most relevant operative techniques and with useful “tips and tricks” for their daily clinical practice.

Once again I thankfully acknowledge the excellent level of scientific quality and educational value of the content that each chapter’s author has contributed. The material received is extremely extensive in terms of quantity and quality that the contents have been apportioned between two volumes.

We are moreover very glad that this project, conducted in cooperation with our World Society of Emergency Surgery and its journal, has truly joined together not only Acute Care surgeons but also surgeons and physicians from other surgical specialties, such as thoracic and vascular surgery, ObGyn, urology, pediatrics, and ENT, as well as gastroenterology, gastrointestinal endoscopy, and interventional radiology, from all over the world sharing our experiences in the management of the acutely ill patients. The multidisciplinary board of authors, editors, and foreword writers of this book is truly international with contributors from the Americas, Europe, Africa, Australasia, and Asia. This is the most heartening and promising signal for a worldwide collaboration.

This is the second of the planned WSES book series, starting the WSES educational program for the next future years. This project aims to link together WSES courses, WSES guidelines, and WSES books to give complete educational tools to the next generation of emergency and trauma surgeons.

WSES is demonstrating to act as the first scientific world society capable to develop a systematic scientific and education program with the aim of science progress according to evidence-based medicine and experience-sharing program among professionals.

I acknowledge the invaluable foreword contributions from two masters Dr. Kenneth Mattox MD FACS and Dr. David Feliciano MD FACS, emanating from their extensive experiences.

Last but not least, I am deeply grateful to the board of Directors of AUSL Bologna for their continuing commitment in improving public health and the care of Acute Surgical patients. Special mention to the Director General of AUSL Bologna Dr. Chiara Gibertoni, the Health Director Dr. Angelo Fioritti, the Administrative Director Dr. AM. Petrini, the Directors of the Department of Emergency Dr. Giovanni Gordini and Department of Surgery Prof. Elio Jovine, and the chief of the Trauma

Surgery Unit Dr. Gregorio Tugnoli. With the contribution and cooperation of all these professionals, an outstanding model of Acute Care Surgery and Trauma Center for a modern and multidisciplinary care of the Acute Surgical patients has been developed in the Province of Bologna, including a functional model of “Hub and Spoke” and a convenient system of tertiary referral care. I am sincerely proud to be part of this exciting multidisciplinary team of AUSL Bologna dedicated to the improvement of Acute Care Surgery model, within a northern Italian province of Emilia Romagna region.

We look forward to a successful and worldwide ongoing cooperation within our international family of enthusiastic Acute Care and Emergency surgeons, aiming to provide a better care for the acutely ill surgical patients.

Bologna, Italy Salomone Di Saverio MD, FACS, FRCS

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Chapter 1

Acute Abdomen: Clinical Assessment and Decision-Making

Fernando Turégano

1.1 Introduction

An acute abdomen is usually defined as an acute abdominal pain of short duration which requires a decision on whether to proceed or not with urgent intervention [1]. All abdominal crises present with one or more of five main symptoms or signs: pain, vomiting, abdominal distension, muscular rigidity, or shock. The severity and the order of occurrence of the symptoms are important for diagnosis, together with the presence or absence of fever, diarrhea, constipation, and others [2, 3]. The presence of tenderness on palpation is a hallmark of potential acute abdominal problem of surgical importance, and it generally implies inflammation of the visceral peritoneum. This tenderness may be accompanied or not by muscular rigidity (*defense guarding* or *guarding*). There are several grades of muscular rigidity, and its elucidation is not always easy on clinical exam, with the exception of the board-like

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rigidity typical of perforated ulcer. This guarding usually implies inflammation of the parietal peritoneum. Sometimes it takes a great deal of clinical acuity and experience to differentiate between voluntary and involuntary guarding. Modern abdominal imaging, interventional radiology, a better understanding of the natural history of many acute conditions, and more effective antibiotic treatments have revolutionized emergency abdominal surgery and certainly improved our decision-making capabilities. CT scan imaging has considerably decreased the challenge of differential diagnosis of the acute abdomen, decreasing also the rate of negative or nontherapeutic abdominal explorations. It has also reduced hospital admission rates and the duration of hospital stays; its overuse, though, should be avoided, especially in the pediatric population [4].

Decision-making should always involve discriminating between urgent and nonurgent causes; patients suspected of nonurgent diagnoses can be safely reevaluated the next day. Antibiotics should be started as soon as sepsis is recognized. Opioid analgesics should not be withheld for fear of affecting the accuracy of physical examination [5].

1.2 Acute Generalized Abdominal Pain with Tenderness

Generalized peritonitis consists of diffuse severe abdominal pain in a patient who looks sick and toxic. The patient typically lies motionless and has a tender abdomen with “peritoneal signs” (rebound tenderness and defense guarding). Surgical management is warranted. The three most common causes of generalized peritonitis in adults are perforated appendicitis, colonic perforation, and a perforated ulcer [1].

The most common causes of colonic perforation are malignancy and acute sigmoid diverticulitis. Colorectal tumors can

perforate in the tumor itself, usually at the rectosigmoid, or at the cecum, after several days of unrelieved complete obstruction in a patient with a competent ileocecal valve. In the latter case, tenderness of the abdomen on the right side may be a sign of impending perforation. Regarding acute diverticulitis, a small number of patients present from the start with diffuse peritonitis, with free intraperitoneal gas on CT scan.

The pain caused by a peptic perforation usually develops very suddenly in the upper abdomen, and most patients present with signs of diffuse peritoneal irritation and tenderness. Spillage of gastroduodenal contents along the right gutter into the RLQ may mimic acute appendicitis and, although very uncommon, can occur in clinical practice (*Valentino's syndrome*). There is free gas under the diaphragm in about two-thirds of perforated patients. Differential diagnosis should also be made at times with diffuse peritonitis from other causes (perforated gallbladder with bile peritonitis and others). An occasional patient with acute pancreatitis may present with a clinical picture mimicking diffuse peritonitis [1], but lab tests and CT scan can help with the diagnosis. In the elderly or not so elderly patient with a known arrhythmia, advanced mesenteric ischemia should be considered; the patient will almost always be clammy, a sign of poor peripheral perfusion typical of the condition at this stage of bowel necrosis.

A CT scan, although not mandatory, is invaluable in confirming the diagnosis and helping decide on the type and extent of laparotomy or whether a laparoscopic approach is warranted.

1.3 Localized Abdominal Pain with Tenderness

The importance of the character of the pain, whether cramping, steady, sharp, etc., cannot be overemphasized. The golden rule is to examine the patient again within 2 or 3 h. In nearly every serious case, there will by that time be some other symptoms,

such as vomiting, fever, or local tenderness, which may point more definitely to the nature of the lesion.

1.3.1 Pain Around the Umbilicus and Epigastrium

It is uncommon in the absence of incarcerated umbilical hernia and omphalitis. Severe epigastric or central abdominal pain with some tenderness may be due to simple intestinal colic, to the initial stage of obstruction of the small intestine, to acute pancreatitis, or even to the initial stages of acute cholecystitis or biliary colic.

1.3.2 RUQ Pain

If the chest is clear (no right basal pneumonia), this is usually due to acalculous acute cholecystitis (AC). RUQ pain and tenderness (*Murphy's sign*) are accompanied by systemic evidence of inflammation (fever, leukocytosis) and usually by a mild or moderate elevation of bilirubin or liver enzymes. There can also be a mild elevation of the serum amylase. Diagnosis is usually confirmed with US. The presence of intramural gas, and gas within the gallbladder lumen (*acute emphysematous cholecystitis*), can be present in AC of diabetic patients. This latter condition should prompt urgent surgical intervention. A laparoscopic cholecystectomy is warranted in most cases within the first few hours or days after diagnosis.

Acalculous acute cholecystitis is a manifestation of the disturbed microcirculation in critically ill patients. Clinical diagnosis can be extremely difficult in that context, and early diagnosis requires a high degree of suspicion, excluding it as a cause of an otherwise unexplained septic state or SIRS. Urgent cholecystectomy or cholecystostomy is warranted.

RUQ tenderness may be also due to acute cholangitis, characterized by *Charcot's triad* (RUQ pain, fever, and jaundice). Disproportionate pain may be due to coexisting AC. In the elderly patient, or when medical intervention is delayed, the syndrome can progress to include confusion and septic shock (*Reynolds pentad*). A typical biochemical panel shows mildly elevated transaminase, variably elevated total bilirubin with a direct preponderance, and a disproportionately elevated alkaline phosphatase and glutamyl transferase. Diagnosis is usually confirmed by US. Interventional biliary decompression by means of ERCP or the transhepatic route should be preferred to surgery and performed as soon as available.

1.3.3 LUQ Pain

The left upper quadrant of the abdomen is least often the site of origin of local peritonitis. Acute pancreatitis is one of the most common causes of pain in the LUQ, often occurring without any epigastric component at all. Vomiting and retching are frequent. A carcinoma or stricture of the splenic flexure of the colon may rarely cause severe localized pain, and constipation will be a common symptom. A left perinephric abscess, the rupture of an inflamed jejunal diverticulum, and the spontaneous rupture of the spleen, all can cause LUQ pain with tenderness [2].

1.3.4 Pain in the Hypogastrium

Hypogastric pain and rigidity in a young or middle-aged man are usually due to appendicitis, while in an older man, an alternative diagnosis would be acute diverticulitis or, infrequently, a rectosigmoid cancer with localized perforation. The same

symptoms in a young woman might be due either to appendicitis or to a gynecological condition.

Acute urinary bladder retention should always be considered in an elderly patient with a history of advanced prostatic, and a tumor mass effect will be felt on palpation. In the pre-US and CT scan era, this condition has been known to lead to an occasional misdiagnosis and abdominal exploration.

1.3.5 RLQ Pain

The most common cause is, of course, acute appendicitis (AA). The different anatomic positions of the appendix may make symptoms and signs variable. Vomiting before the onset of pain should lead to suspicion of a different diagnosis. The occurrence of diarrhea, especially in children, is occasionally misleading. This diarrhea can be caused by a pelvic appendix irritating the rectum by contiguity or irritation by a pelvic abscess. Sometimes, with a perforated iliac appendix lying behind the end of the ileum, the subsequent symptoms of small bowel obstruction may be misleading.

McBurney's point of tenderness corresponds roughly to the position of the base of the appendix, and it is common to find no local muscular rigidity in a case of appendicitis without any peritonitis. Pressure over the LLQ will sometimes cause pain in the appendicular region (*Rovsing's sign*). Fever almost never precedes the onset of pain. A slight tachycardia is a common and helpful sign in cases of doubtful clinical diagnosis. Laparoscopic or open appendectomy is indicated, taking into consideration that perforation in AA is not strictly a time-dependent phenomenon [6]. Conservative management with antibiotics is being advocated now by some groups in Europe. Occasionally, palpation of a mass over the RLQ, together with a clinical picture consistent with appendicitis of several days' duration, should prompt the diagnosis of an *appendiceal phlegmon*. An US or CT scan should rule out an abscess within the phlegmon, in which

case drainage by interventional radiology is indicated. Otherwise, surgery should be contraindicated at this stage.

In the female, acute salpingitis, hydrosalpinx with a twisted pedicle, a ruptured follicular cyst (*mittelschmerz* or pain at mid-cycle), a ruptured *corpus luteum* cyst (pain with the menses), a ruptured pyosalpinx, and a ruptured ovarian endometrioma can be misdiagnosed as AA on clinical grounds. An US is usually of help, and the decision-making process will depend on the specific condition.

1.3.6 LLQ Pain

Acute diverticulitis (AD) of the sigmoid colon is the most frequent cause, and there is sometimes rigidity of the overlying muscular abdominal wall. There may be slight fever, but neither epigastric initial pain nor vomiting. There are signs of systemic inflammation with fever, increased CRP (C-reactive protein), and leukocytosis with left shift. The patient usually has a simple phlegmonous AD or a pericolic abscess, and conservative management is indicated, with interventional percutaneous drainage in large abscesses. When the sigmoid colon is redundant and lies well to the right, confusion with AA can be considerable.

Pain and tenderness in the LLQ may also be due to inflammation around a cancer of the sigmoid colon.

1.4 Acute Abdominal Pain Without Tenderness

1.4.1 Early Acute Mesenteric Ischemia (AMI)

In early AMI the clinical exam is remarkably nonspecific. The patient usually complains of severe abdominal pain, with very

little findings on physical examination, leading the clinician to frequent misdiagnoses of very dire consequences for the patient. *Any patient with an arrhythmia such as auricular fibrillation who complains of severe abdominal pain of sudden onset should be highly suspected of having embolization to the superior mesenteric artery (SMA) until proved otherwise [1].*

In thrombosis, there is usually a history indicating previous abdominal angina, and the patient complains initially of mild central cramping abdominal pain. Frequent bowel movements are common and usually contain either grossly or microscopically detectable blood. It will never be too overemphasized that, in the early stages, physical examination of the process is treacherously benign; peritoneal irritation appears too late, when the bowel is already dead. Laboratory studies usually are normal until the bowel loses viability, when leukocytosis, hyperamylasemia, and lactic acidosis develop. A CT angio is usually very helpful in ruling out embolism or thrombosis to the SMA.

Nonocclusive mesenteric ischemia (NOMI) is due to a low-flow state, in the absence of documented arterial thrombosis or embolus. The low-flow state is often due to a combination of low cardiac output, reduced mesenteric flow, or mesenteric vasoconstriction in the setting of a preexisting critical illness. The condition may involve the entire small intestine and colon, often in a patchy distribution. The clinical picture may be indistinguishable from that of organic occlusion of the mesenteric vessels. *Any patient who takes digitalis and diuretics and who complains of abdominal pain must be considered to have NOMI until proven otherwise.* Chronic renal insufficiency patients on hemodialysis are also prone to this condition.

Mesenteric venous thrombosis is much less common than the previously discussed, and there is commonly an underlying hypercoagulable state or sluggish portal flow due to hepatic cirrhosis. The use of contraceptive pills has been implicated as a pathogenetic factor. The clinical presentation is nonspecific.

Abdominal pain and varying gastrointestinal symptoms may last a few days until eventually the intestines are compromised, and peritoneal signs develop.

Decision-making in AMI may involve interventional procedures, surgical treatment and anticoagulation, alone or combined, and also palliative care.

1.4.2 Pain Radiating to the Back

In a dissecting aneurysm of the aorta, sometimes the patient will come in complaining of an unbearable pain through the back, extending down to the abdomen, and, initially, without any tenderness nor rigidity on palpation. On careful questioning, the pain would have started in the thorax, radiating through to the back. Significant arterial hypertension of prolonged duration is usually a forerunner, and there will almost certainly be differences between an upper- and a lower-limb pulse according to the position of the lesion. Clinical misdiagnosis with a renal colic has not been uncommon in the pre-CT scan era, with dire consequences for the patient.

Leakage or rupture of an abdominal aneurysm is by far a more common cause of abdominal pain than is a dissecting thoracic aneurysm. Any patient with a known aneurysm and recent abdominal pain should be regarded as being in imminent danger of rupture. When present, the pain prior to rupture is of a throbbing (pulsatile) or aching nature, and it is located in the epigastrium or the back. Collapse in a patient with a known aneurysm almost always indicates rupture. Abdominal and flank examination usually reveals a mass representing the extravasated hematoma, and the left flank is the most common site. Time to surgery or endograft is of the essence here, and insistence on a preop CT angio in every case, although invaluable to plan management, still contributes in many deaths.

A postmetecic transmural rent in the distal esophagus (Boerhaave's syndrome) usually presents with pain radiating to the back and no epigastric tenderness. The sequence of vomiting first followed by sharp pain in the back should help in making an early clinical diagnosis and saving the patient's life. Surgical management is usually warranted, but endoscopic procedures do have a role in early cases.

1.5 Nonspecific Abdominal Pain (NSAP)

NSAP is defined as pain lasting a maximum of 7 days for which no immediate cause can be found during the acute admission and specifically does not require surgical intervention. It is a presenting symptom of a large number of minor and self-limiting conditions. It is a diagnosis by exclusion, and up to 10% of patients with NSAP over the age of 50 years have subsequently been found to have an intra-abdominal malignancy. An association between NSAP and irritable bowel syndrome or celiac disease has been described. Women account for about 75% of admissions with NSAP. Compared with active clinical observation, early laparoscopy has not shown a clear benefit in women with NSAP [7].

1.6 Painful Abdominal Wall Swelling

An incarcerated hernia is one of the most common forms of intestinal obstruction, and it is often difficult to make certain whether an hernia is merely incarcerated or whether it is strangulated (with advanced ischemia or necrosis of its content), for pain and constipation are usually present in both cases. With simple incarceration of short duration, though, the pain tends to

be milder than with strangulation. In certain cases there may be little local tenderness to call attention to the hernia.

A strangulated femoral hernia gives rise to more mistakes in diagnosis than a strangulated inguinal hernia. Sometimes only a small knuckle of gut comprising a small portion of the circumference of the bowel may be caught in the femoral canal (*Richter's hernia*), and scarcely any projection may be felt in the thigh. Some of these patients, usually elderly ladies, will be worked up with a presumed diagnosis of intestinal pseudo-obstruction, and a CT scan can confirm an accurate preoperative diagnosis. Inflamed and enlarged inguinal glands produce a more diffuse and fixed swelling, and fever is not uncommon. Vomiting will be absent. They result from a primary cause that may be detected on the corresponding thigh, the penis, or anoperineal region. Ultrasound may be helpful, but, ultimately, only surgical intervention will differentiate between both conditions in some patients. An inflamed appendix in a femoral hernia sac (*Littre's hernia*) cannot be distinguished definitely from a strangulated femoral hernia before operation.

An obturator hernia is very uncommon and most frequently found in wasted, elderly women. Symptoms of obstruction of unknown cause will predominate. The only local symptom may be some pain radiating down the inner side of the thigh along the distribution of the obturator nerve. If the diagnosis is suspected on clinical grounds, something very unusual, rotation of the thigh (*Romberg's sign*) will elicit pain. CT scan is diagnostic.

A rectus sheath hematoma usually manifests itself as a painful abdominal swelling of moderate size and imprecise limits and used to be confused with other acute abdominal conditions of surgical importance in the pre-CT scan era. If discoloration of the skin is already present, together with the typical history of bouts of coughing in a patient on anticoagulation medication, the diagnosis is more straightforward.

1.7 The Postoperative Acute Abdomen

Few clinical situations are as diagnostically demanding as the evaluation of the abdomen in a patient who has undergone an abdominal operation [2]. The key to an early diagnosis of a serious abdominal complication is a frequent daily assessment of the patient. The passage of stool and gas, and also at times resumption of an oral diet, is not always a guarantee that all is well within the peritoneal cavity. Peritonitis that occurs from the fourth to the eighth or ninth postoperative day is almost always caused by an anastomotic disruption. Pain is usually present, and any new pain should be regarded with suspicion. Tenderness and rigidity are usually present but may be so mild as to be misleading. At times signs and symptoms can be so subtle as to represent a real clinical challenge. Occasionally, only the presence of oliguria leads to the suspicion of anastomotic disruption in an otherwise asymptomatic patient. The presence of an unexplained tachycardia, in the absence of fever, or tachypnea, in the absence of atelectasis or pneumonia, should also raise the suspicion of anastomotic disruption. In the latter, many patients are thought to have pulmonary embolus and undergo CT scan and other tests, when in fact the tachypnea is due to the post-op abdominal condition. The decision-making process should always be individualized and surgical revision warranted in many cases, but interventional radiology and therapeutic endoscopy should be considered when deemed appropriate.

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Chapter 2

Diagnostic Tools in ACS: CT Scan, Diagnostic Laparoscopy, and Exploratory Laparotomy

Ning Lu and Walter L. Biffl

2.1 Introduction

The abdomen is a black box of diagnostic uncertainty. There is an old surgical adage that goes, “Never let the skin come between you and the diagnosis.” However, it is just that: an old adage. The surgeon has many alternatives to employ in situations in which the clinical diagnosis, or decision to operate, is not straightforward. In this chapter, three primary modalities are discussed: computed tomography (CT) scanning, diagnostic laparoscopy (DL), and exploratory laparotomy (LAP).

2.2 CT Scanning

The CT scan is an exceedingly valuable tool for the diagnosis of essentially any abdominal surgical problem. A CT scan can quickly

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and accurately demonstrate any number of pathologies while ruling out others, allowing the surgeon to narrow the list of differential diagnoses and plan definitive management strategies. It is noninvasive, rapid, and nearly universally available and has been insinuated into myriad clinical care guidelines for surgical problems. The ability to grade the severity of pathology prior to operating allows the surgeon to tailor the approach to the situation and to counsel the patient regarding expectations more accurately.

2.2.1 Perforated Gastroduodenal Ulcers

CT is 95 % sensitive and 93 % specific for diagnosing gastroduodenal perforation. In addition to identifying free air, signs of periduodenal fat stranding, wall defect/ulcer, and wall thickening can be seen 72–89 % of the time [1]. However, these other signs may not be visible before at least 6 h of symptomatology [2].

2.2.2 Cholecystitis

Ultrasonography is the accepted standard for detecting cholelithiasis and diagnosing acute calculous cholecystitis. CT can detect gallstones only 50 % of the time, but in patients with equivocal ultrasounds, CT can demonstrate wall thickening, pericholecystic stranding, and pericholecystic fluid [3–5]. CT is also valuable in identifying complications of cholecystitis, including emphysematous, hemorrhagic, or perforated cholecystitis [6].

2.2.3 Choledocholithiasis

CT has a diagnostic sensitivity ranging from 56.5 to 81 % and a specificity ranging from 72.8 to 96 %. Thus, it is not the initial imaging study of choice for patients suspected of

choledocholithiasis [7, 8]. On the other hand, CT can accurately and reliably identify common bile duct dilation.

2.2.4 Pancreatitis

CT has a 92 % sensitivity and 100 % specificity in identifying acute pancreatitis. It is 80–90 % accurate with a 90 % sensitivity and 33 % specificity in identifying pancreatic necrosis [9–11]. In addition, CT imaging allows classification of pancreatitis per Atlanta and revised Atlanta classification [12, 13].

2.2.5 Small Bowel Obstruction

CT is able to diagnose complete bowel obstruction with a sensitivity of 92 % (81–100 %) and a specificity of 93 % (68–100 %). CT is able to diagnose intestinal ischemia with 83 % (63–100 %) sensitivity and 92 % (61–100 %) specificity [14, 15]. CT has great value in patients with inconclusive plain films and can be helpful in determining the likely etiology of the obstruction, whether it is due to hernias, adhesions, or malignancy [16].

2.2.6 Mesenteric Ischemia

CT angiography is rapid and noninvasive for diagnosis of acute mesenteric ischemia and its multiple etiologies (arterial thrombosis, arterial embolism, mesenteric vein thrombosis, and non-occlusive ischemia), with a sensitivity and specificity of 96 % and 94 %, respectively [17]. CT angiography in nonocclusive mesenteric ischemia will demonstrate no signs of arterial or venous occlusion, may demonstrate vascular spasm, and may demonstrate more diffuse nonconsecutive segments of bowel with signs of ischemia. However, after ruling out vascular

occlusive disease, diagnosing nonocclusive mesenteric ischemia still requires a high clinical suspicion [18]. The ability to differentiate the multiple etiologies of mesenteric ischemia is critical as the treatment for each can vary.

2.2.7 *Appendicitis*

CT is 91% sensitive and 90% specific for diagnosing acute appendicitis. For those suspected of having appendicitis, there is clear benefit to the use of IV, but not oral contrast [19]. In addition, CT can grade the severity of appendicitis (inflamed, perforated with localized free fluid, perforated with regional abscess, perforated with diffuse peritonitis) [20]. The grading of appendicitis can allow for appropriate treatment plans, which may be operative or via IR drainage.

2.2.8 *Diverticulitis*

CT is 94% sensitive and 99% specific in the diagnosis of acute diverticulitis [21]. In addition to identifying the absence or presence of perforation, CT allows for Hinchey classification of perforated diverticulitis. This facilitates determination of whether hospitalization is required and selection of patients for medical vs. surgical therapy [22] (Table 2.1).

CT is, of course, not without risks. The average CT abdomen/pelvis with contrast has an estimated radiation dose of 10–30 mSv and 3–10 mSv in pediatric patients. The average CT angiogram of the abdomen has an estimated radiation dose of 1–10 mSv and 0.3–3 mSv in pediatric patients. When possible, the risks of radiation exposure are minimized in the pregnant and pediatric populations. Depending on the pathology, ultrasound and MRI are viable options with similar accuracy. In the pediatric population, ultrasound approaches the accuracy of

CT in diagnosing appendicitis with a sensitivity of 88% and specificity of 94% [23]. In the pregnant population suspected of appendicitis, MRI has a sensitivity of 97% and specificity of 95% [24].

Most non-trauma patients are candidates for CT for diagnosis. It is not recommended for patients who are unstable and in extremis. For those with renal dysfunction, exposure to contrast agents should be minimized. There is a well-known risk of contrast-induced nephropathy.

2.3 Diagnostic Laparoscopy

Laparoscopy is increasingly used in the diagnosis and treatment of many intra-abdominal pathologies. Traditionally, patients admitted with acute abdominal pain of unclear origin

Table 2.1 Summary of CT in diagnosing intra-abdominal pathologies

Pathology	Sensitivity	Specificity	Grading/ classification capability
Perforated gastroduodenal ulcers	95%	93%	
Cholecystitis	–	–	
Choledocholithiasis	56.5–81%	72.8–96%	
Pancreatitis	92%	100%	X
Pancreatic necrosis	90%	33%	
Small bowel obstruction	92% (81–100%)	93% (68–100%)	
Intestinal ischemia	83% (63–100%)	92% (61–100%)	
Mesenteric ischemia	96%	94%	X
Appendicitis	91%	90%	X
Diverticulitis	94%	99%	X

are managed with observation (serial abdominal exams, laboratory tests, and/or repeat imaging), progressing to surgery only if signs of peritonitis develop. However, this can lead to delays in diagnosis. In certain populations (immunocompromised, morbidly obese, paraplegic/quadruplegic, sedated, comatose), the abdominal exam is not always reliable. In patients with a suspected acute abdomen or unexplained unrelenting acute abdominal pain, especially those with an unreliable exam, diagnostic laparoscopy may be invaluable. The diagnostic accuracy of laparoscopy is 90–99.5% [25–30].

After a diagnosis is made, treatment can also be achieved laparoscopically in many instances with safety and efficacy. By avoiding laparotomy, the relatively higher morbidity can be avoided as well. In cases of acute cholecystitis and acute appendicitis, laparoscopic cholecystectomy and appendectomy are safe and effective, now becoming the standard of care (level I). For patients with Hinchey I–IV perforated diverticulitis, when colectomy is performed, laparoscopic colectomy (with or without Hartmann's procedure) has been performed successfully by expert laparoscopic groups. For patients with Hinchey III perforated diverticulitis, laparoscopic exploration with peritoneal lavage and drainage is an emerging therapeutic modality. Current recommendation for laparoscopic management of diverticulitis is level III. For gastroduodenal perforations, laparoscopic management has been demonstrated to be safe and effective (level I) [31]. In the case of adhesive small bowel obstruction, laparoscopy is an emerging therapy, which may be successful in the hands of an experienced laparoscopic surgeon on a hemodynamically stable patient, in the absence of peritonitis or severe intra-abdominal sepsis, in patients with localized distention on imaging, the absence of severe abdominal distention, an anticipated single band, and a low peritoneal adhesion index. The etiology of the obstruction can be determined with 96.9% accuracy, and treatment can be provided without conversion to laparotomy in more than 50% of patients [16, 32,

33]. Minimally invasive necrosectomy is an emerging therapeutic option with less morbidity and mortality than open necrosectomy in the hands of experienced laparoscopic surgeons [34, 35].

Laparoscopy is contraindicated with patients known to have a “frozen abdomen,” massive bowel distention, inability to tolerate pneumoperitoneum, uncorrectable coagulopathy, uncorrectable hypercapnia >50 Torr, or hemodynamic instability [36]. Historically, laparoscopy was delayed until the second trimester to reduce the likelihood of complications including spontaneous abortions and preterm labor. However, recent studies show that it may be safe to perform laparoscopy during any trimester of pregnancy without increased risk to the mother or fetus. However, data on long-term effects to children is lacking [37].

Given the safety, efficacy, and accuracy of diagnostic laparoscopy, with the added ability to treat most diagnosed pathologies, laparoscopy should be considered in the majority of patients with an acute abdomen.

2.4 Exploratory Laparotomy

For those with suspected intra-abdominal pathologies, and certainly those with evidence of peritonitis, laparotomy is still the gold standard. Patients with an acute abdomen and a contraindication to laparoscopy require laparotomy. Especially critical in the decompensating patient, laparotomy has the ability to diagnose with absolute certainty and provide treatment of the disease. However, exploratory laparotomy has significantly higher morbidity (5–22%) compared to diagnostic laparoscopy [38]. Thus, in stable patients without contraindications, a minimally invasive approach should be considered.

2.5 Conclusions

The three modalities – CT, DL, and LAP – are individually very accurate and thus frequently employed. Rather than consider them competitive, they are complementary tests that have major roles in Acute Care Surgery.

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Chapter 3

Laparoscopy: A Diagnostic and Therapeutic Tool for Acute Care Surgery

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Of the current global burden of disease, more than 15% of the total disability-adjusted life years (DALYs) lost are due to diseases requiring surgical management [1, 2]. Much of this burden is attributable to immediately life- or limb-threatening conditions that necessitate emergency surgery [1]. The resuscitation and management of these very sick patients require physicians who are in tune with complex human physiology and critical care needs [3]. According to the definition of the American Association for the Surgery of Trauma formulated in international collaboration with many other societies, the scope of acute care surgery includes most surgical emergencies of traumatic or nontraumatic etiology [4].

Due to continuing advances, mainly developed for, or well adapted to, minimal access techniques, laparoscopy today has a

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major role not only in elective but also in emergency surgery, not just as a technical variation of the approach, but also because of other added benefits that include better vision, less parietal insult, and potentially less morbidity, which in turn might contribute to better outcomes [2, 5].

The most prevalent surgical diseases worldwide that can be treated by laparoscopy are in increasing order of frequency, peptic ulcer disease, bowel obstruction, biliary disease, hernia, and appendiceal disease [1–5].

The main diagnostic modalities (abdominal sonography, computer tomography [CT], and endoscopy) have their specific indications and play a major role in detecting the cause of an emergency. In contrast, an advantage of laparoscopy is that the entire abdomen can be seen and can lead to therapy almost at the same time as diagnosis.

According to the OPTIMA study, sensitivity and specificity of abdominal sonography are 63 % and 99 %, respectively, for intestinal obstruction and 76 % and 95 %, respectively, for appendicitis [6]. Sensitivity and specificity of CT are 69 % and 99 %, respectively, for intestinal obstruction and 95 % and 95 % for appendicitis [6].

Magnetic resonance tomography (MRI) plays a subordinate role in acute diagnostics; its accuracy is similar to that of CT, but it is time-consuming and more expensive.

3.1 Patient Position and Trocar Setup

For emergency laparoscopic exploration of the abdomen, the patient should be positioned supine and adequately fixed to the table with shoulder supports so that the table can be turned in any position, including extreme Trendelenburg or anti-Trendelenburg. The surgeon should stand on the side or between

the legs of the patient, opposite to the anticipated pathology. The patient (table and equipment) should be positioned so that the surgeon has access to whichever side of the patient is involved and can move around to gain access to all four quadrants of the abdomen as required.

Trocar positioning is especially important in acute surgery since surgery on the organs involved is more difficult than in elective surgery. Optimal triangulation is essential, and the optic should ideally be placed midway between the working trocars.

For diagnostic purposes with an unclear abdomen, the optic trocar should be in the center of the abdomen, i.e., at the navel. Ideally two trocars should be inserted along the anterior axillary line on both flanks so that both the upper and lower abdomen can be accessed (Fig. 3.1). In other indications, the trocars are located according to the organ involved as determined by appropriate imaging modalities.

The initial trocar layout for acute nonspecific abdominal pain (NSAP) depends on clinical findings and diagnostic probabilities: emergency explorative laparoscopy for acute abdominal pain predominating in the lower abdomen should allow complete exploration of the pelvis and the genital organs as well as the appendix. In the case of intestinal dilation (intestinal obstruction or ileus secondary to peritonitis or abscess), trocars should be inserted as far as possible from the expected site of obstruction and if needed in the flank to best view the middle of the abdomen. Previous scars (incisions or drainage sites) should be avoided. Additional trocars can be added as needed.

It is strongly recommended to insert the first trocar with the open technique. While this does not always avoid intestinal injuries, it does prevent the more serious vascular injuries, and should an incidental enterotomy occur, the diagnosis and repair can be undertaken immediately.

The following indications for diagnostic and therapeutic laparoscopy will be dealt with in more detail:



Fig. 3.1 Trocar setting in diagnostic emergency laparoscopy

Nonspecific abdominal pain
Appendicitis
Perforated ulcer
Small intestinal obstruction/incarcerated hernia
Acute cholecystitis
Diverticulitis
Trauma

3.2 Nonspecific Abdominal Pain (NSAP)

Defined as acute abdominal pain in the right iliac or hypogastric area lasting more than 6 h and less than 8 days, without fever, leukocytosis, or obvious peritoneal signs and with uncertain diagnosis after physical examination and baseline investigations including abdominal sonography [7], NSAP represents 25–40% of admissions. While most authors still advise abdominal and pelvic CT (LE1b) [8], final diagnosis can be established by diagnostic laparoscopy (GoR A) [2]. The diagnostic precision of laparoscopy is 90–100% (LE2b), and in 36–95% of patients, a laparotomy can be avoided (LE3b) [9]. In experienced hands, the morbidity is below 8%; no mortality has been reported in connection with laparoscopy for NSAP (LE2b) [10, 11]. According to Decadt, early laparoscopy can even provide higher diagnostic accuracy and improved quality of life for patients with NSAP [12].

3.3 Appendicitis

While the SAGES guidelines state that the indications for laparoscopic appendectomy should be the same as for open appendectomy [7], the reality is quite different as rates have increased 3.5-fold from 1998 to 2002 (20.6–70.8%), with laparoscopy becoming the most prevalent approach in many institutions as the treatment of choice for appendicitis [13]. However, even if there might be marginal benefits for the patient, widespread use of laparoscopic appendectomy is fraught with increased costs for hospitals and health care systems [13, 14]

Laparoscopy has been lauded as a diagnostic modality in the evaluation of a patient suspected of having acute appendicitis, but it is an invasive procedure requiring general anesthesia and

with a risk similar to appendectomy. Even though it may be helpful in equivocal cases or in women of childbearing age, it should not be advocated as a routine diagnostic procedure to replace the classical preoperative workup. Moreover, no correlation has ever been shown between the macroscopic aspect of the appendix and the presence of appendicitis [15], raising the unanswered question of whether a “normal” appendix should be removed during laparoscopy for abdominal pain when no other obvious disease is present. Several studies have shown that clinical examination is superior, complemented by laboratory and imaging studies (sonography and especially CT scan) (sensitivity, 94 % and specificity, 95 %). The EAES recommends laparoscopy for a perforated appendix. Laparoscopic appendectomy may offer particular advantages for perforated appendicitis, the obese, the elderly, and patients anxious to return to normal activities sooner or for cosmetic reasons [16, 17].

In selected cases, antibiotics as first-line management for uncomplicated appendicitis might not be inferior to surgery, but this treatment modality cannot yet be recommended since the results of the available trials are methodologically not strong enough to support widespread application [18].

3.4 Perforated Duodenal Ulcer

While the prevalence of ulcer disease has decreased in recent years, perforation remains a life-threatening complication [1, 19], especially if diagnosis is delayed [2, 20]. Correct diagnosis relies on clinical examination and imaging techniques, the most reliable being CT, both for positive diagnosis (sensitivity nearly 100 %, specificity 86 %) and the site of perforation. Laparoscopy also performs well in this respect [21, 22]. Once the diagnosis has been made, laparoscopic treatment can be initiated and has been shown to be effective in low-risk patients [23], obviating

the need for a midline laparotomy [24]. The techniques for closure include suture, with or without omental reinforcement (a vascularized omentoplasty is better than the classic, free omental Graham patch), with or without fibrin adhesive. Problems with the laparoscopic approach are related to difficulty in locating the ulcer and choosing how to treat large or giant perforations (>10 mm) and friable edges that do not lend themselves to suturing. Success rates are not inferior to open surgery [19]

3.5 Small Intestinal Obstruction/Incarcerated Hernia

Postoperative adhesions are the most frequent cause of small intestinal obstruction (84.9%), much more common than incarcerated hernia or tumors (3.2 and 2.5%, respectively) [25, 26]. Intravenous contrast-enhanced CT imaging is essential to recognize intestinal ischemia (precision 73–80%, sensitivity 85–100%) and to eliminate other causes of mechanical or paralytic ileus [27]. Complete obstruction mandates surgery as soon as possible, while partial obstruction can undergo the trial of time or contrast flush. Laparoscopy is particularly indicated for patients fit for surgery, who ideally have not undergone more than two previous operations and when single-band obstruction and thin adhesions (often secondary to appendectomy by a McBurney incision) are expected. The diameter of the dilated intestinal loop should not be more than 4–5 cm, the distension of the abdomen should not be extensive, and the surgeon should be experienced in advanced laparoscopic techniques [28]. Adequate exploration is facilitated by tilting the operating table and gentle and atraumatic running of the intestines, care being taken not to grasp the dilated bowel, but rather the adjacent mesentery. Contraindications are essentially the same as the general contraindications for laparoscopic surgery, i.e., massive intestinal

obstruction with pronounced distension, hemodynamic instability, and perforated viscus (free air in the abdomen). During laparoscopy, if a bowel resection is needed in a patient with extensively dilated loops, conversion to open surgery or resection with establishment of the anastomosis through a small incision via minilaparotomy should be seen as a wise decision.

Intestinal obstruction due to any kind of hernia should be treated without delay (GoR C), and laparoscopy is a viable option (GoR B). Care must be exercised not to perforate or rupture the incarcerated bowel, and the use of a mesh is not contraindicated [29, 30]. Incarcerated hernia repair is readily applicable, safe, and beneficial, shortening hospital stay, reducing morbidity, and allowing quicker return of intestinal activity than after open repair.

3.6 Acute Cholecystitis

Open or laparoscopic cholecystectomy is the treatment of choice for acute cholecystitis. Acute cholecystitis was long thought to be a contraindication for the laparoscopic approach, but today most authors agree that if performed early, laparoscopy is the preferred approach in grade A and B disease (Tokyo Guidelines, Table 3.1) (GoR A). Routine intraoperative cholangiography is an important tool to reduce the risk of iatrogenic bile duct injury during acute cholecystitis [31–33]. In the future, identification of the bile duct structures before dissection, especially utilizing fluorescent illumination under near-infrared light after injection of indocyanine green, might have an important place in this setting [34, 35].

The ideal timing of cholecystectomy for acute cholecystitis is an important issue that remains controversial today. Notwithstanding the variety of conclusions, essentially due to variations in the delays before surgery, the opinion of experts depends on whether there is an increased risk for bile duct injury

Table 3.1 Updated Tokyo classification for acute cholecystitis

The grade of severity for acute cholecystitis	Optimal treatment
Grade I (mild)	Early laparoscopic cholecystectomy is the preferred procedure
Grade II (moderate)	Early cholecystectomy is recommended in experienced centers. However, early gallbladder drainage (percutaneous or surgical) may be indicated in cases of severe local inflammation. Since early cholecystectomy may be difficult, medical treatment and delayed cholecystectomy are mandatory
Grade III (severe)	Immediate management of organ dysfunction and urgent intervention for severe local inflammation through gallbladder drainage should be accomplished. In case of cholecystectomy indication, delayed elective cholecystectomy should be implemented

in acute cholecystitis. Early laparoscopic cholecystectomy (<24 h or at the latest <72 h from onset) is recommended in grades A and B acute cholecystitis [36, 37]. For grade C, delayed cholecystectomy is recommended, but there is no evidence that late cholecystectomy is easier or that it entails fewer bile duct injuries. The fundus-down dissection technique should be avoided in acute cholecystitis as the risk of combined biliovascular injury seems to be increased according to the Tokyo Guidelines Experts' Report in 2013 [37].

3.7 Diverticulitis

Laparoscopic management of diverticular disease requiring surgery is feasible and safe, and physical and intestinal recovery is faster, with lower morbidity and costs than with open surgery

Table 3.2 Hinchey classification

Hinchey I – localized abscess (paracolic)
Hinchey II – pelvic abscess
Hinchey III – purulent peritonitis (the presence of pus in the abdominal cavity)
Hinchey IV – feculent peritonitis

Although the Hinchey classification (Table 3.2) was developed before CT came into routine use, it is still used to categorize the therapeutic indications for complicated diverticular disease. While most if not all patients with Hinchey I and IIa disease can be managed conservatively or with interventional radiology, some patients with Hinchey IIb as well as cases with persisting signs of sepsis in spite of percutaneous drainage require surgery. Laparoscopic surgery is appropriate and has been found to be just as effective and safe as open surgery [38, 39]. In Hinchey III, when lavage and drainage do not suffice, or in Hinchey IV, laparoscopic colectomy with or without a protective stoma is a possible option (GoR C). When peritonitis is present, the risk of anastomotic failure is 5–30 % higher, and morbidity increases from 10 to 50 % [40]. Elderly patients and those with immunosuppression or severe systemic comorbidity are at risk of re-intervention after simple laparoscopic lavage [41].

3.8 Trauma

In the abdominal or thoracic hemodynamically stable trauma victim, laparoscopy has a place as a diagnostic (and potentially therapeutic) option. Most cases involve injuries to the mesentery, intestines, pancreas, or diaphragm [42]. Laparoscopy should allow a rapid workup of injuries that require surgical

attention and therefore decrease the rate of missed injuries and morbidity associated with delayed operations on hollow organs and the diaphragm [43]. Laparoscopy in penetrating abdominal trauma shows a sensitivity ranging from 66.7 to 100 %, specificity from 33.3 to 100 %, and accuracy from 50 to 100 %. It is safe and more cost-effective than a negative laparotomy for stable patients with penetrating abdominal trauma [44]. The advantages highlighted are reduction of morbidity, accuracy in detecting diaphragmatic and intestinal injuries, and elimination of prolonged hospitalization just for observation.

Diagnostic laparoscopy seems particularly interesting in patients with free fluid of unknown origin, suspected intestinal injury with blunt trauma, mesenteric injury, pancreatic injury, and penetrating trauma in hemodynamically stable patients [42]. In these settings, laparoscopy is particularly well suited to detect the injury and in some cases allows repair. Potential risks with laparoscopy for trauma patients are gas embolism, increased intracranial pressure with head injuries, and tension pneumothorax when the diaphragm is interrupted.

3.9 Conclusion

Laparoscopy is an established diagnostic and potentially therapeutic procedure in emergency settings. Laparoscopy is very effective when a diagnosis can be obtained and therapy undertaken during the same procedure. This is especially the case in patients with acute cholecystitis, appendicitis, and perforated ulcers. The role of laparoscopy is still debated and poorly defined in patients with nonspecific abdominal pain or complicated diverticular disease. In experienced hands, it has specific indications in intestinal obstruction, incarcerated hernia, and trauma.

The advantages of the minimal access approach to emergency surgery reside in its combined diagnostic and therapeutic capacity, as well as in the avoidance of formal laparotomy, which reduces morbidity and length of hospital stay and in some instances contributes to cost-effectiveness.

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Chapter 4

Esophageal Non-bleeding Emergencies: Minimally Invasive and Endoscopic Management of Esophageal Perforations

Stefano Siboni and Luigi Bonavina

4.1 Introduction

Despite significant advances in diagnosis and treatment, esophageal perforation remains a life-threatening event and an emergency condition particularly challenging for the surgeon. Iatrogenic injury is the most frequent cause of esophageal perforation, accounting for 59 % of all patients, followed by spontaneous rupture (15 %), foreign body ingestion (12 %), trauma (9 %), and esophageal cancer (1 %) [1].

Depending on comorbidity, etiology, location, extent, and time to treatment of the injury, mortality can be considerably high, reaching the rate of 36 % in some published series [1–3]. Early diagnosis is of paramount importance to improve outcomes. CT scan is a quickly available, first-line diagnostic tool. The role of endoscopy has been controversial for years but is accepted today in most referral centers. Initial manage-

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ment of esophageal perforation has traditionally relied upon open surgical procedures such as primary suture repair with or without tissue reinforcement, esophageal diversion, or esophageal resection. More recently, treatment strategies have increasingly shifted toward the use of minimally invasive procedures, including laparoscopic surgery, endoscopic stenting, and CT scan-guided drainage.

4.2 Clinical Features and Diagnosis

4.2.1 *Iatrogenic Injury*

Endoscopy accounts for at least one third of esophageal perforations [4]. The procedure most commonly complicated by perforation is pneumatic dilation for achalasia (2–6%), followed by sclerotherapy for varices (1–5%) and stricture dilation (0.09–2.2%) [5, 6]. Conversely, the event is rare during diagnostic endoscopy (0.03%). The majority of perforations occur at Killian's triangle, particularly in the presence of an unknown Zenker's diverticulum. Other predisposing factors are cervical osteophytes, strictures, and malignancies [7].

Esophageal perforation can also occur during esophageal or non-esophageal surgery. Cervical injury can occur during osteosynthesis for cervical spine fracture with a reported rate of 3.4% [8]. Thoracic and abdominal perforations have been reported following esophageal and pulmonary surgery [5, 9]. Often, the esophageal injury is detected during the endoscopic or surgical procedure, but occasionally it may go unrecognized and lead to significant treatment delay. Leakage of the esophago-visceral anastomoses after esophagectomy is discussed elsewhere in this book.

4.2.2 *Boerhaave's Syndrome*

Spontaneous perforation of the esophagus is caused by sudden increase in intraesophageal pressure that usually occurs during retching or vomiting. The distal part of the intrathoracic esophagus, mainly the left side, is the most common site of injury, but even cervical perforations have been reported [10]. The main symptom is severe retrosternal pain, and in 75% of the cases there is a history of vomiting [11]. Other symptoms that can develop later are odynophagia, dyspnea, and signs of sepsis. Patients with cervical perforation may present with cervical pain, dysphagia, dysphonia, and subcutaneous emphysema. The most fearful complications are mediastinitis, pneumothorax, and pleural empyema.

Patients presenting with severe chest pain and recent history of vomiting should be promptly investigated with contrast esophagram and/or CT scan [12]. Cervical and chest radiograph are far less sensitive because mediastinal emphysema and widening may not be visible radiologically in the first hour after the perforation [13]. CT scan is mandatory in order to detect mediastinal or abdominal collections and to plan the treatment [14]. Upper endoscopy should be performed only by an experienced surgeon/gastroenterologist who may also be able to manage the perforation [15, 16].

4.2.3 *Caustic Injury*

Ingestion of caustic agents may cause severe damage to the esophagus. The extent of the injury depends on the volume, the pH, the concentration of the substance ingested, and the duration of contact with the esophageal mucosa. Esophageal damage usually follows alkali ingestion that may cause transmural

colliquative necrosis. In contrast, acid ingestion passes quickly into the stomach, thus causing less injury to the esophagus [17, 18]. The clinical presentation of caustic ingestion includes dysphagia/odynophagia and oropharyngeal, retrosternal, or epigastric pain. Abdominal tenderness, leukocytosis, elevated CRP, and $\text{pH} < 7.2$ indicate a severe injury [19]. Tracheal intubation is mandatory in case of dyspnea and severe airway involvement. Plain chest radiograph is useful to detect early signs of perforation, such as pneumomediastinum, pneumoperitoneum, or pleural effusion. Endoscopy should be performed 3–6 h after ingestion in order to assess the extent of the injury [20]. The Zargar classification is widely used to identify patients who need emergency surgery [17]. Recent studies have demonstrated that CT scan is better than endoscopy in selecting patients for surgery or nonoperative management [21].

4.2.4 Foreign Body

Foreign body ingestions occur accidentally in children and edentulous adults and intentionally in prisoners or mentally impaired patients. Accidental ingestions are usually treated conservatively, while operative treatment is required in patients who ingested dangerous objects such as batteries, blades, or needles. These objects can impact the esophagus at the physiologic narrowing areas causing ulcers or perforations and possible aorto-esophageal and tracheoesophageal fistula. The most common site of foreign body impact is the cervical esophagus (57%) [22]. The main symptoms are dysphagia, odynophagia, hyper-salivation, and neck tenderness [23]. Neck, chest, and abdominal radiograph should be performed in all patients to detect perforation and to locate the site of impact. Impaction of food and plastic, wood, and glass objects is usually not visible at the plain radiograph, and therefore a CT scan is required [24].

Endoscopy performed within a few hours from the ingestion may allow to retrieve some foreign bodies [25].

4.2.5 External Trauma

Esophageal perforation following trauma is rare [26]. The majority of the esophageal injuries are penetrating, while perforation secondary to blunt trauma is uncommon, although possible after motor vehicle accident [27]. Cervical and upper thoracic esophagus are the most common sites of injury (82 %) [26]. The trachea is involved in 75 % of the patients. Symptoms and signs of esophageal perforation can be subtle; therefore these patients need to be thoroughly investigated. In clinically stable patients, the most accurate test is the esophagoscopy, with a reported sensitivity and specificity of 100 % and 92.4 %, respectively [28], whereas Gastrografin swallow and CT scan have a high false-positive rate [29]. In unstable patients the esophagoscopy should be performed in the operating room during the emergency operation [26].

4.3 Treatment

Esophageal perforations are very heterogeneous entities. Surgical exploration has been traditionally recommended as it allows the surgeon to take care of the esophageal perforation, to provide adequate drainage, and to establish enteral nutrition support. However, no treatment has been conclusively demonstrated to be superior to others, in part because of patient selection bias and the lack of standardized therapeutic algorithms. Over the past decades, new management algorithms based on minimally invasive techniques have been proposed.

4.3.1 Esophageal Stenting

Endoscopic stenting has been proposed as temporary or definitive treatment for early esophageal perforation. Most stents are available with a plastic coverage that helps to seal the perforation and uncovered ends to allow integration into the esophageal wall. Particularly suitable for this procedure are patients unfit to surgery, such as those with severe comorbidities, and patients with iatrogenic and post-traumatic perforations, where an early diagnosis is likely [30–32]. Patients with spontaneous perforation usually have a greater delay in diagnosis and treatment, and therefore surgical management may be more appropriate. When the delay in treatment exceeds 12 h, a CT scan and a multidisciplinary approach are mandatory to detect any mediastinal or abdominal collections that can be managed with drainage or minimally invasive surgical technique [32, 33]. In the case of external trauma, priority has to be given to life-threatening associated injuries. If hard signs of vascular or laryngotracheal trauma are present, emergent surgical exploration is mandatory, and primary repair is usually performed. An endoscopic esophageal stent could be placed as a damage control procedure during the emergent operation.

The reported success rates for esophageal stent insertion range from 85 to 100 %, but the procedure may be challenging even for an experienced endoscopist. Factors associated with failure are cervical or gastroesophageal injuries, long perforations, and the emergency setting [34]. The most common complications are stent migration with plastic stents and obstruction with metal stents (6–35 %) [4, 35].

4.3.2 Endoscopic Clipping

Endoscopic clipping has been traditionally used as treatment for upper gastrointestinal bleeding, but in the last two decades, several cases of perforation repair with clips have been reported. A

Careful endoscopic evaluation is critical to decide whether or not the perforation is manageable endoscopically. Indications for this techniques are small (<1 cm) and clean perforations with a normal aspect of the surrounding mucosa, usually occurring after iatrogenic endoscopic perforation or foreign body ingestion [36, 37]. Delay in treatment seems to be the main predictive factor of failure [38]. A limitation of the conventional endoscopic clips is the difficulty to approximate the thickened mucosal borders, especially in the case of significant gap. Lately, the over-the-scope clip (Ovesco) has been introduced as an effective treatment for small spontaneous perforations [15, 16]. A recent series reported a success rate of 65 % and the reduced need for emergent operations [39, 40]. More recently, a novel endoscopic suturing system (OverStitch, Apollo Endosurgery) has been launched that may overcome the limitations of the currently available clips [41].

4.3.3 Endoluminal Vacuum Therapy

Large perforations not amenable to primary endoscopic stenting/clipping in high-risk patients can be treated by endoluminal vacuum (VAC) therapy. A recent study has shown complete closure of perforation/leak after an average of 35 days with multiple VAC changes [42].

4.3.4 Minimally Invasive Surgery

In the last two decades, minimally invasive surgery has emerged as a safe and effective approach in esophageal surgery. Video-assisted thoracoscopy and laparoscopy have been demonstrated to be better tolerated than open surgery. The goals of the minimally invasive approach are identification and closure of the perforation, debridement of necrotic tissue, and drainage of the

mediastinum [43]. The outcomes are strongly affected by several factors, such as length and the vitality of the margins of the perforation. Thoracoscopy has also been proposed as the technique of choice to remove foreign bodies impacted in the esophagus below the cricopharyngeal sphincter [44]. It is logical to assume that the minimally invasive surgical approach has the best chance of success in patients with early diagnosis of perforation.

4.4 Conclusions

Esophageal perforation is a rare but serious condition which is traditionally treated by an open surgical approach. More recent therapeutic algorithms based on minimally invasive and hybrid techniques have been proposed and applied in high-volume referral center [45, 46]. In expert hands, laparoscopy, thoracoscopy, endoscopic stenting, and clipping combined with percutaneous drainage of collections are safe and effective techniques to treat early perforations with clean margins.

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Chapter 5

Management of Postoperative Complications in Esophagogastric Surgery

Emanuele Asti and Luigi Bonavina

5.1 Introduction

Recent advancements in minimally invasive surgery have changed the scenario of the management of cancer of the esophagus and stomach compared to the past century. The evolution of surgical technique has been paralleled by an increasing tendency, at least in high-volume centers, to apply “fast-track” protocols according to the enhanced recovery after surgery (ERAS) principles in patients undergoing esophagogastrectomy for cancer [1, 2]. Nonetheless, these methodological changes have not yet definitely impacted on postoperative morbidity. Complications continue to occur despite a steady decline of operative mortality [3]. Postoperative morbidity and mortality after upper gastrointestinal surgery still depend on the preoperative physiological patient status, comorbidity, use of neoadjuvant therapy, and also on the anesthetic and intensive care approach. Therefore, good case selection, appropriate choice of

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the operative strategy, and careful postoperative monitoring continue to play a critical role in preventing the potential catastrophic consequences of surgical complications [4]. Finally, it has been shown that technical complications of esophagogastric surgery also have a profound negative impact on patient survival, and, as a consequence, strategies to optimize surgical techniques and minimize complications have the potential to improve long-term outcomes [5].

Postoperative complications include adverse events that are common to any major surgical operation and those specific to foregut resective surgery. Complications related to the surgical procedure can be treated conservatively or may need an active intervention (endoscopic, percutaneous, surgical). It is important to recognize that the Dindo-Clavien classification has represented a major advance by providing a platform and a common language for reporting of surgical complications worldwide [6].

5.2 Esophageal Surgery

Most esophageal resections are currently performed for squamous cell or adenocarcinoma of the esophagus. Neoadjuvant chemoradiation protocols are employed in significant proportions of these patients worldwide to downstage the tumor and increase the chance of radical resection.

5.2.1 *Respiratory Complications*

Respiratory complications occur in up to one out of four patients undergoing esophagectomy without a statistically significant difference between the transthoracic and the transhiatal approach [7]. Over the past years, with the advent of minimally invasive

thoracoscopic and hybrid techniques of esophagectomy, a decreased incidence of respiratory complications has been observed [8–10]. Pain from extensive incisions is the major contributor to decreased ventilation and atelectasis from mucous plugs which lead to bronchopneumonia and respiratory failure. An additional factor is represented by the mediastinal lymphadenectomy that impairs the drainage from the alveoli and leads to pulmonary fluid retention and edema.

5.2.2 Surgical Site Infections and Cardiovascular Complications

The incidence of surgical site infections remains low after esophageal resection. Conversely, thromboembolic complications, cerebrovascular accidents, and myocardial ischemia are not uncommon in the elderly patient population [11, 12].

5.2.3 Anastomotic Leakage

The rate of anastomotic leak between the esophagus and the conduit used for reconstruction, most commonly the stomach, is the highest among any surgical anastomosis. Currently, even in specialized centers, the leakage rate is around 10% [13]. Early dehiscence of an anastomosis may occur within 48–72 h as a result of a technical error or, more rarely, a failure of the stapler [14, 15]. Diagnosis is confirmed by Gastrografin swallow study, CT scan with oral contrast, and upper gastrointestinal endoscopy. New-onset atrial fibrillation is an ominous clinical sign that should raise the suspicion of anastomotic leak [16]. Depending on the extent of the lesion, the first-line treatment for an intrathoracic anastomotic dehiscence can consist of surgical exploration or endoscopic stenting or clipping [17]. If the endoscopic approach is chosen, concomitant ultrasound or CT

scan-assisted percutaneous drainage of fluid collections is mandatory. Dehiscence of a cervical anastomosis does always require opening of the wound and drainage to prevent a mediastinal abscess. In large disruptions, the use of a T-tube is helpful to create a controlled external fistula.

Later dehiscence of an anastomosis, generally manifesting between the 5th and the 10th postoperative day, may be the result of ischemia of the esophageal substitute or is caused by a perianastomotic abscess. Conservative treatment is recommended in such circumstances, especially in subclinical leaks which consist of antibiotics, nasogastric suction, percutaneous drainage of fluid collections, early enteral nutrition via naso-enteral tube, or jejunostomy. Larger anastomotic defects may require endoscopic stenting. Stents should be of large caliber to ensure complete sealing of the defect and prevent an endoleak. Partially covered stents are preferable and should be removed within 3–4 weeks [18]. Cervical anastomotic leaks require early external drainage to prevent mediastinitis. Enteral nutrition by a nasojejunal tube or via jejunostomy should be provided to these patients for at least 3 weeks, which represent the minimum time to safe fistula healing.

5.2.4 Gastric Resection Line Leakage

This is a rather infrequent event, but when it occurs the defect is usually large. The leakage may be secondary to ischemia of the gastric tube in the area between the uppermost linear staple line and the circular anastomosis. Another possible site of leakage is at the intersection of the staple lines along the gastric tube. Surgical re-exploration is mandatory in these patients.

5.2.5 Ischemia of the Gastric/Colonic Conduit

Gangrene of the conduit is a catastrophic complication requiring prompt resuscitation and immediate takedown of the conduit

followed by cervical esophagostomy, stapling of the conduit remnant, and a feeding jejunostomy. The continuity of the alimentary tract can be restored at a later date by a colonic interposition.

Subtle ischemic changes of the mucosa of the proximal conduit may be asymptomatic. However, otherwise unexplainable tachycardia accompanied by hypoxia and low-grade fever should alert the surgeon. Endoscopy and CT scan are recommended to diagnose the condition at an early stage and to follow up the patient with conservative treatment and enteral feeding [19].

5.2.6 Gastric Outlet Obstruction

It should be differentiated from gastroparesis. The latter is usually a transient phenomenon and can be treated with nasogastric aspiration and intravenous erythromycin. Mechanical obstruction may be due to stricture at the level of the crura or twisting of the stomach that occurs during pull-up in the chest or in the neck. Both conditions require a reoperation.

5.2.7 Chylothorax

Damage to the thoracic duct may occur either after transthoracic or transhiatal esophagectomy. The diagnosis is straightforward when a high-volume output and/or milky fluid is seen in the chest drain. Sometimes, a chyle leak is recognized only after resuming oral feeding when a thoracentesis is required to drain a massive pleural effusion. Conservative treatment with octreotide and low-fat diet may be successful. However, if a sustained chyle leak persists for more than 2 weeks, surgical treatment is recommended. Administration of cream through a nasogastric tube 6 h before surgery is useful to identify the duct and the

leakage site. The operation can be performed through a right thoracotomy/thoracoscopy [20] or, in some circumstances, through a transhiatal approach [21].

5.3 Gastric Surgery

Most anatomical gastric resections are currently performed for gastric carcinoma. Today, morbid obesity is also a common indication for bariatric surgical procedures such as gastric bypass and sleeve gastrectomy. The overall morbidity rate in gastric surgery is higher after total gastrectomy and esophagojejunostomy, especially in the case of combined splenectomy and distal pancreatectomy.

5.3.1 Duodenal Stump Leakage

It may be related to a technical error, ischemia of the transection margins, or closed-loop obstruction of the afferent jejunal limb. In patients with early leaks, the appearance of high-output bile-stained fluid in the drain is an indication for immediate open surgical revision or relaparoscopy. Delayed leaks can be treated conservatively until a fistulous tract is established, and no fluid collections are detected on CT scan. An elemental type of diet is allowed and subcutaneous octreotide should be administered. If undrained collections are present and there is evidence of intra-abdominal sepsis, either a pigtail drain can be inserted percutaneously or a reoperation may be required to drain the subhepatic space with silastic tubes and place a Petzer tube in the duodenal stump. Inspection of the enteroenterostomy is mandatory to verify patency. As an alternative to reoperation, percutaneous

transhepatic duodenal drainage has been reported as an effective method to externally drain biliopancreatic secretions [22].

5.3.2 Anastomotic Leakage

It is often due to a technical error after a total or subtotal gastrectomy or after a gastric bypass operation, and it may occur with either circular or semimechanical linear anastomoses. In morbid obese patients, the only clinical symptom of leakage from a staple line may be an otherwise unexplained tachycardia, abdominal pain, and/or low-grade fever. Esophagojejunal anastomotic leaks occur in less than 10% of patients. A reoperation is indicated in case of early leak (<72 h). The management of late leaks is more controversial. Endoscopic assessment should always follow the Gastrografin swallow study to evaluate the extent of the dehiscence and to differentiate a true anastomotic leakage from a jejunal stump leak. In an asymptomatic patient with a contained leak, enteral feeding through a nasojejunal tube or via jejunostomy is recommended for at least 3 weeks. The role of endoscopic stents remains controversial although effective in selected patients [23, 24].

5.3.3 Gastric Staple-Line Leakage After Sleeve Gastrectomy

Leaks appearing at the angle of His are not uncommon after sleeve gastrectomy [25]. These leaks, both immediate and delayed, can safely be treated by endoscopic stent placement. Persistent leaks after previous restrictive surgery are more difficult to handle and can require reoperation and Roux-en-Y gastrojejunostomy [26].

5.3.4 *Intra-abdominal Sepsis*

It can be related to anastomotic or duodenal leakage, pancreatic stump leakage, or pancreatic necrosis. It carries a significant risk of life-threatening secondary hemorrhage. Percutaneous drainage may be a reasonable approach unless there are bowel loops interposed between the abdominal wall and the abscess cavity or there is evidence of significant necrotic tissue at CT scan that invariably requires surgical debridement.

5.4 Conclusions

Prevention, early recognition, and a proactive attitude before sepsis develops are the keys for successful treatment of complications after esophagogastrrectomy. Changing the algorithm from a “wait and see” to a “look and see” policy with a more liberal use of radiological and endoscopic imaging has contributed to better patient outcomes over the past 25 years. Actually, the integrated use of interventional endoscopy and radiology has significantly reduced the need for reoperation and the overall mortality rate for these complications.

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Chapter 6

Management of Complicated and Strangulated Hiatal Hernias

Smita Sihag and David W. Rattner

6.1 Classification and Pathophysiology of Hiatal Hernia

6.1.1 Classification

The prevalence of hiatal hernias is estimated to be somewhere between 10 and 50 % in the population, with greater frequency in patients over the age of 50. Many are discovered incidentally by radiologists or gastroenterologists, as symptoms caused by the hernia or gastroesophageal reflux occur in half of patients. The lifetime risk of gastric volvulus or strangulation with ischemia to the stomach is not known precisely, but mortality of emergency surgery in this scenario has historically reported to be greater than 50 % [1].

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The classification system of hiatal hernias is based on the relative positions of the gastroesophageal junction, the stomach, and the esophageal hiatus [2].

- Type I hernias are sliding hiatal hernias, where the gastroesophageal junction migrates freely above the diaphragm due to weakness predominantly in the posterolateral phrenoesophageal attachments. The stomach, however, remains in its normal alignment.
- Type II hernias occur when the gastroesophageal junction remains fixed in its normal anatomic position, but a portion of the fundus herniates through the diaphragmatic hiatus adjacent to the esophagus. The anterior phrenoesophageal attachments are usually disrupted in this case, while the posterolateral attachments may be preserved.
- Type III hernias represent a combination of types I and II, whereby the gastroesophageal junction has migrated above its normal anatomic position, and the fundus and body have herniated through the hiatus, lying cephalad to the intrathoracic gastroesophageal junction.
- Type IV hernias are characterized by the herniation of other intra-abdominal organs, such as the spleen, colon, small bowel, and/or omentum through the esophageal hiatus. It is often an extension of a type III hernia, as the gastroesophageal junction and some or all of the stomach have already herniated through the hiatus as well. These are associated with a very large hiatal defect.

Type I hiatal hernias are, by far, the most common type, representing up to 95 % of all hiatal hernias. Acute presentation of sliding hiatal hernias is exceedingly rare, however, and they tend to be associated with gastroesophageal reflux disease (GERD) and obesity. As there is almost never an indication to address type I hernias on an emergent basis, the focus of this chapter will be on type II–IV hiatal hernias, referred to as paraesophageal hernias (PEH), which comprise the residual 5 % of hiatal hernias. Almost 90 % of PEH are type III, and the least common

is a type II at less than 2%. The term “giant paraesophageal hernia” typically refers to type III and IV hernias, where greater than 50% of the stomach is in the chest [3]. With respect to giant paraesophageal hernias, distinguishing between subtypes is more of a theoretical than practical exercise, as the surgical approach and management are rarely affected.

6.1.2 Pathophysiology

The pathophysiology of hiatal hernias is not entirely understood, but widening of the esophageal hiatus and cephalad migration of the gastroesophageal junction are likely related to the following factors: [4]

- Laxity of the phrenoesophageal membrane as a result of decreased elastin and collagen fibers in the context of connective tissue dysfunction or advanced age
- Increased intra-abdominal pressure due to obesity, pregnancy, or possibly repetitive straining (i.e., vomiting, heavy lifting, constipation)
- Esophageal shortening as a consequence of GERD with chronic inflammation and fibrosis

While genetics may play a role to some degree, the above are primarily acquired risk factors. Of note, other diaphragmatic hernias, such as congenital or traumatic hernias, are beyond the scope of this discussion, though some principles of management and surgical repair may overlap. Because the phrenoesophageal membrane and its attachments to the muscular wall of the lower esophagus constitute a key anatomic component of the lower esophageal sphincter (LES), there is a close relationship in the evolution of both hiatal hernias and gastroesophageal reflux. Thus, GERD is a frequent early symptom of hiatal hernia in the initial non-acute presentation and represents an indication for elective repair if proton pump inhibitor therapy proves insufficient.

6.2 Clinical Manifestations in the Acute Setting

6.2.1 Presentation

6.2.1.1 Gastric Volvulus

The clinical presentation of PEH in the acute setting includes obstruction, bleeding, perforation, or strangulation. Nearly 50% are thought to be symptomatic (though minor symptoms may be incorrectly attributed to other etiologies), and the literature suggests that the annual risk of developing symptoms in the setting of a known PEH is approximately 14% [5]. The risk of developing acute symptoms, however, that mandates an emergent operation is likely to be less than 2% per year. Gastric volvulus with migration of the stomach into the chest is categorized as either mesenteroaxial or organoaxial, based on the axis of rotation of the stomach. Organoaxial rotation is more common (approximately 60% of cases) and occurs when the stomach rotates horizontally along the long axis, connecting the pylorus and gastroesophageal junction. Strangulation and necrosis occurs in up to 30% of cases with organoaxial gastric volvulus [6]. Mesenteroaxial refers to vertical rotation along the short axis of the stomach, bisecting the greater and lesser curves of the stomach. Mesenteroaxial rotation is less frequent and less likely to lead to vascular compromise of the stomach. Combined organoaxial and mesenteroaxial rotation is also possible, but occurs in less than 10% of cases. Borchardt's triad of epigastric pain, retching without vomiting, and inability to pass a nasogastric tube represents the acute clinical manifestation of gastric volvulus that has progressed to complete obstruction. Of note, volvulization of the stomach with organoaxial or mesenteroaxial rotation can be chronic and may be seen on imaging in the absence complete obstruction, strangulation, or perforation, though significant symptoms are usually apparent and risk of

progression to either of these endpoints is presumably higher in these patients.

6.2.1.2 Obstruction

Obstructive symptoms may occur intermittently, usually following oral intake. These symptoms range from nausea, vomiting, dysphagia, heartburn, and regurgitation to severe postprandial pain related to gastric distension and transient ischemia with or without volvulus. At times, the clinical picture can be confused with angina or other cardiopulmonary etiologies given that substernal chest pain radiating to the back, palpitations, and dyspnea are quite common. Respiratory symptoms frequently occur with giant paraesophageal hernias, though repair does not necessarily relieve these symptoms. On average, patients experience a 10–20% improvement in pulmonary function values [7]. Giant paraesophageal hernias can also cause compression of the inferior pulmonary vein or right atrium, which leads to rhythm disturbances, such as supraventricular tachycardia. This often triggers a full battery of cardiac testing, including cardiac catheterization, all of which usually turn up negative. Patients may also present to the emergency department with a more chronic history of reflux, recurrent aspiration events with pneumonia, early satiety, worsening food intolerance, and weight loss. Only paraesophageal hernias where part (i.e., fundus) or all of the stomach has ascended into the chest are at risk of acute gastric volvulus and subsequent obstruction.

6.2.1.3 Bleeding

Upper gastrointestinal (GI) bleeding in the setting of PEH is almost always a consequence of Cameron's ulcers, which are thought to arise from mechanical friction of the gastric mucosa

in the sliding hernia sac. They are typically described as superficial, linear erosions of the stomach at the level of herniation where the stomach is constricted by the diaphragm. While slow, occult bleeding resulting in iron deficiency (microcytic) anemia has been reported in up to 47% of patients with giant paraesophageal hernias, acute symptomatic hemorrhage from Cameron's lesions, accompanied by melena or hematemesis, is only seen rarely. Initial treatment of upper GI bleeding from Cameron's ulcers involves acid suppression with intravenous proton pump inhibitors and supportive measures, such as iron supplementation or transfusion if necessary. Definitive management, however, is not amenable to endoscopic interventions and relies on surgical repair of the hernia. Occult bleeding with iron deficiency anemia resolves in 90% of patients following surgical repair [8].

6.2.1.4 Strangulation

Strangulation represents the most catastrophic endpoint of gastric volvulus within a PEH and is defined by acute vascular compromise of the stomach and possibly other organs. Patients may present in various degrees of extremis, and symptoms tend to escalate from intermittent to constant, severe substernal and epigastric pain. Frequently, obstructive symptoms will exacerbate the clinical picture. Signs of sepsis may also be present with hypotension requiring vasopressors, respiratory distress, and evidence of inadequate end-organ perfusion. Laboratory studies may reveal a lactic acidosis and a leukocytosis, though the elderly septic patient may be leukopenic instead.

6.2.1.5 Perforation

Perforation is a much less common endpoint of acute PEH, but has been described in various case reports where incarceration

of the fundus of the stomach within the hernia sac leads to perforation. Linear tears of the gastric corpus have also been described in the setting of organoaxial volvulus. This complication is more common in the immunosuppressed patient on steroids. Typically, other symptoms of obstruction and strangulation precede perforation, as this represents a downstream finding likely secondary to ischemia. The perforation may be contained within the hernia sac or may extend freely into the peritoneal or pleural cavities. Pleural effusions or fulminant mediastinitis may result, and signs of systemic sepsis, leukocytosis with bandemia, and respiratory failure are to be expected in this scenario.

6.2.2 *Diagnosis*

6.2.2.1 **Computed Tomography**

Spiral computed tomography (CT) scan is the primary diagnostic tool of choice in the patient that presents to the emergency department with acute symptoms and suspicion of PEH [6]. Abdominal plain films are insufficient to map out the anatomy and plan for the appropriate surgical approach. CT scan clearly shows the anatomy of the esophagus and stomach and allows for a complete assessment of the hernia including (1) percentage of the stomach that has herniated into the chest, (2) whether other organs are contained in the hernia sac, (3) complete or partial obstruction if there is no passage of contrast distally, (4) organoaxial vs. mesenteroaxial gastric volvulus with swirling of the fat of the lesser or greater omentum, (5) ischemia if there is stranding or pneumatosis of the stomach wall, (6) and perforation with free air and fluid. Figure 6.1 shows a CT image of a giant type IV PEH containing small bowel and stomach with organoaxial rotation. Plain films can certainly provide clues with findings such as a retro-cardiac air fluid level on lateral view or an



Fig. 6.1 Axial CT image of a giant type IV paraesophageal hernia

intrathoracic stomach with compressive atelectasis of the lung. The presence of pneumomediastinum or pneumoperitoneum signifies perforation.

6.2.2.2 Upper GI Series

In the stable patient with lower concern for complete obstruction or strangulation, esophagram plus upper GI series with either water-soluble contrast or barium is the first-line diagnostic study and can be a helpful adjunct to CT in further delineating the anatomy of the esophagus and stomach, the position of the gastroesophageal junction, and whether any partial obstruction may exist. A contained perforation may

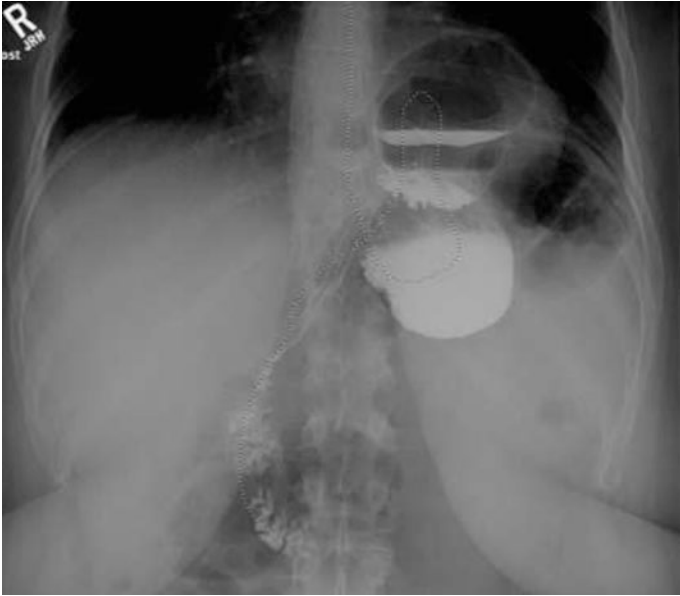


Fig. 6.2 Upper GI series of incarcerated intrathoracic stomach with near-total obstruction

also be identified. Gastric volvulus is best recognized on CT scan, but can also be seen on a barium contrast study. Figure 6.2 demonstrates an incarcerated intrathoracic stomach with near-total obstruction and only minimal passage of contrast to the small bowel, as seen on an upper GI series contrast study. In the patient that presents with systemic sepsis or gastric volvulus with concern for vascular compromise of the stomach, barium studies should be forgone as they simply delay surgical intervention. Moreover, they should be avoided if there is imminent risk of aspiration of contrast material. Lastly, obtaining an esophagram in the acute setting may not

even be an option, as many centers do not have a 24-h on-site radiologist or gastrointestinal fluoroscopy team readily available to perform emergent exams.

6.2.2.3 Manometry and pH Testing

While manometry and pH probe monitoring can also occasionally provide useful information with respect to the degree of symptomatic reflux and motility pattern of the esophagus and stomach, there is absolutely no role for these studies in the acute presentation of PEH. In the elective setting, the function and location of the LES may be more accurately assessed by manometry, and esophageal shortening may be apparent if the distance between the upper and lower esophageal sphincters is less than expected. Evaluation of peristaltic function, combined with data on symptomatic acid reflux, may assist the surgeon in determining what type of anti-reflux procedure to offer the patient prior to an elective repair. However, many surgeons would argue that these tests are of little value even in the elective setting, since the need for an esophageal lengthening procedure or fundoplication in an attempt to restore LES function is usually determined intraoperatively. In addition, both manometry and 24-h pH probe monitoring may be technically impossible to execute in patients with giant paraesophageal hernias.

6.2.2.4 Endoscopy

Endoscopic evaluation of the esophagus and stomach should be performed in the operating room prior to any surgical intervention, acute or elective, for PEH. Findings of erosive esophagitis, Barrett's dysplasia, mass, or ulcer disease can guide operative planning, as resection may be indicated rather than simple reduction of the hernia. Viability of the gastric mucosa

and torsion of the stomach is also critical to assess in the case of gastric volvulus and may also guide the surgeon toward resection or not. Endoscopic decompression of the stomach may facilitate further operative management, and nasogastric tube placement may require direct visualization if the tube does not pass easily or if there is uncertainty about the effectiveness of gastric decompression. Endoscopic detorsion of the stomach with percutaneous endoscopic gastrostomy placement in the setting of acute gastric volvulus is rarely feasible, though this approach has been described by some surgeons in cases where most of the stomach is below the diaphragm.

6.3 Indications for Surgical Repair

6.3.1 *Elective Indications*

As the morbidity and mortality associated with emergent repair of PEH has been historically high, some surgeons insist that all should be repaired on an elective basis regardless of symptoms if the patient is an appropriate surgical candidate. In 1967, Skinner and Belsey published a rate of nearly 30% of patients who did not undergo operative repair that progressed from only minimal symptoms to death from acute strangulation [1]. The rationale for repair upon diagnosis is also related to the fact that many surgeons believe that paraesophageal hernias tend to enlarge over time and become more and more technically difficult to reduce and repair, and the patient's operative risk will only increase with advancing age, though there is little actual published data describing the natural history of hiatal hernia. Other, more recent data suggests a much lower rate (less than 2% per patient per year) of asymptomatic patients that go on to develop life-threatening complications of an incarcerated

PEH. In particular, Stylopoulos and colleagues use an extensive mathematical model that incorporates the results of five different studies to estimate the risk of acute complications to be approximately 1.2% per patient per year [5]. The lifetime risk of acute complications of PEH in a 65-year-old patient is, therefore, predicted to be on the order of 18%. Thus, repairing an asymptomatic or minimally symptomatic PEH has become increasingly controversial. Morbidity and mortality following emergent or urgent repair has significantly decreased as well over time in the era of laparoscopic reduction, to a range of 5–20% in more recent studies [9]. As a result, the current recommendation is to follow asymptomatic patients, especially in the absence of a large hernia (greater than 30% of the stomach above the diaphragm) or evidence organoaxial rotation of the stomach. In the author's experience, giant paraesophageal hernias are very rarely completely asymptomatic, and symptoms may be more insidious and long standing or slow to evolve. They may include postprandial bloating, weight loss, a change in eating habits to small portions, or avoidance of certain foods. If the patient is symptomatic, the guidelines universally support pursuing elective repair at whatever age if the patient is of reasonable surgical risk [10]. Larusson et al. have reported a significant quality of life improvement in elderly patients over the age of 70 who underwent laparoscopic repair [11]. Prophylactic repair is considered acceptable in patients younger than age 65 of low surgical risk based on patient preference, though recommendations for prophylactic repair need to be tempered by the realization that radiographic evidence of recurrent herniation is seen in 40% of patients within 5 years of surgery in some series [12]. Prophylactic repair in patients over the age of 80 is not recommended. Undeniably, patients that undergo elective repair of giant paraesophageal hernia early upon onset of symptoms have the best outcomes. Mortality following elective repair is less than 1–2% [13].

6.3.2 Urgent Indications

Estimates of the morbidity and mortality of emergency surgery for acute presentation of complicated PEH vary widely, and therefore, the timing of when to operate is not well defined. The majority of patients that present to the emergency department with acute symptoms of giant paraesophageal hernia can be treated conservatively in the initial phase, as most commonly, their complaints are related to either acute or chronic worsening obstruction secondary to volvulus. These are patients with stable hemodynamics and no signs of systemic sepsis or imminent ischemia or perforation of the stomach. First steps of management rely on nasogastric tube decompression of the stomach, electrolyte repletion, and fluid resuscitation with correction of any base deficit. Many patients will improve with these preliminary measures, and in that case, they may be watched closely and either repaired during the same hospitalization or semi-electively if their condition improves adequately. Even mild to moderate epigastric pain due to low-grade ischemia and partial strangulation may resolve with decompression, since the redundant blood supply to the stomach makes gastric necrosis a rare event. These patients should, however, undergo surgical intervention within days of initial presentation. Bawahab et al. propose a useful algorithm based on their study of 20 patients that were repaired laparoscopically [14]. Their study suggests repeating a contrast study after nasogastric decompression and fluid resuscitation. If the patient remains obstructed, repair is performed urgently. If there is passage of contrast, surgery is delayed to the semi-elective setting. Though only six patients were included in the semi-elective repair group, a study from Kohler et al. also shows that delayed or semi-elective operative management yields better outcomes than emergency surgery as none of the patients in this arm experienced a perioperative complication [15].

6.3.3 Emergent Indications

Emergency surgery for complicated or strangulated PEH is inherently high risk. As mentioned above, mortality was historically reported to be as high as 56%. Early experiences of emergent laparoscopic repair of giant paraesophageal hernia suggested an average mortality rate of 17%. More recently, mortality has been reported to be as low as 5.4% with laparoscopic reduction [5]. The high mortality rates overall, though, are mostly attributable to the patient population that presents with incarcerated PEH (i.e., the elderly and frail), as well as the difficulties of treating mediastinitis. Patients that present with evidence of incarcerated intrathoracic stomach complicated by strangulation or perforation require emergent surgical intervention. Systemic sepsis and lactic acidosis should be treated with intravenous fluids and a nasogastric tube to decompress the stomach since degree of vascular compromise to the stomach may be mitigated with these maneuvers. However, while preoperative decompression and resuscitation is critical, unstable patients presenting in extremis should be taken to the operating room within hours of arrival in the emergency department. Bhayani and colleagues examined the outcomes of 224 patients from the National Surgical Quality Improvement Database who underwent early vs. interval repair following acute presentation [16]. Early repair within 24 h of admission was associated with better outcomes in terms of lower rates of postoperative sepsis and shorter length of hospital stay. Perhaps as a testament to the quality of critical care and nutritional support available in the current era, mortality was not different between early and delayed treatment groups, however. Thus, absolute indications for emergent operation include hemodynamic instability with evidence of gastric necrosis or perforation. The remainder of cases must be taken on an individual basis to determine optimal timing for repair. If there is any suspicion for ischemia, even if

transient, PEH repair should be undertaken during the sentinel hospitalization. We recommend that all paraesophageal hernias presenting in an acute manner be repaired as soon as possible, unless age and comorbidities are prohibitive.

6.4 Surgical Principles and Techniques

6.4.1 *Laparoscopic Versus Open*

Laparoscopic repair is currently the standard approach in both elective and emergent cases of PEH repair. Many studies now demonstrate that a laparoscopic approach is not only safe but less morbid overall especially in elderly patients. Postoperative respiratory complications, pain, wound infections, and length of hospital stay have all been found to be reduced with laparoscopic repair [17]. Many surgeons also argue that visualization of the hiatus and even into the mediastinum is superior with greater mobilization of the esophagus and less need for esophageal lengthening procedures, though skeptics suggest that pneumoperitoneum may distort the hiatus and perhaps make the intra-abdominal esophagus appear longer. However, the first caveat of laparoscopic paraesophageal hernia repair is that the surgeon must be experienced and comfortable with complex laparoscopy of the foregut, routinely performing anti-reflux and other benign esophageal procedures. The second is that the patient must be able to tolerate pneumoperitoneum for approximately 2–4 h, which is the average duration of this procedure in most hands. Entry into the pleural cavity does occasionally occur during laparoscopic repair when the hernia sac is scarred into the pleural surface and difficult to mobilize. If airway pressures increase, the diaphragm becomes floppy or the patient becomes hypotensive – all signs of a

clinically significant pneumothorax – a pigtail catheter may be placed mid-operation with resolution of symptoms. Typically, the case may proceed laparoscopically, as pneumoperitoneum may still be maintained without respiratory or circulatory compromise. Of note, if there is pleural entry with no clinical consequence during the procedure, pigtail placement is not necessary. Postoperatively, the lung usually re-expands quickly with reabsorption of any residual carbon dioxide.

For surgeons without advanced laparoscopic training, open laparotomy is an acceptable alternative. Efficacy of hernia repair is adequate and has a similar recurrence rate as minimally invasive surgery based on single-center, retrospective comparisons [18]. An open approach is also recommended in the unstable or hypotensive patient that will not tolerate pneumoperitoneum and may have frank gastric necrosis or perforation with gross peritoneal contamination. After the stomach is untwisted and reduced into the abdomen, viability of the stomach must be assessed directly and endoscopically. Small perforations and tears may be repaired primarily in two layers with an omental flap buttress if the stomach is viable. Any necrotic stomach must be resected. If the patient remains unstable from septic shock, requiring vasopressors, performing immediate anastomosis and reconstruction is not advisable. Rather, a damage control strategy should be adopted. The esophagus and stomach may be decompressed using a nasogastric or gastrostomy tube, and reconstruction with esophagojejunostomy or gastrojejunostomy may be performed 24–48 h later. It is nearly impossible to lengthen the esophagus transabdominally and often quite difficult to resect the hernia sac. In such instances, the surgeon needs to reduce the volvulus, ensure viability of the stomach, and try to prevent re-herniation in the short term. This can be done by placing a gastrostomy tube or performing an anterior gastropexy. Repairing the hiatus is not always possible and simply getting the patient out of imminent danger may be the appropriate

endpoint, realizing that a definitive hiatal hernia repair may ultimately be required in the future when the patient can tolerate it. Placement of nonabsorbable mesh to close the hiatus or reinforce hiatal closure in the acute setting should be avoided.

6.4.2 Transthoracic Versus Transabdominal Approach

Some surgeons advocate that all giant paraesophageal hernias with greater than 50% of stomach in the chest should be approached via left thoracotomy. Moreover, mobilization of the esophagus may be more extensive with good visualization of the hiatus, and a tension-free repair can be potentially more easily achieved. However, there are cases reported where the stomach could not be completely reduced from the chest, and the patient required subsequent laparotomy to untwist a gastric volvulus [19]. Thus, in the instance of emergency surgery for the strangulated or perforated stomach, laparotomy is likely to be superior to thoracotomy. Opening the pleura and allowing potential contamination of this space predisposes to serious respiratory complications, including pneumonia and empyema.

In general, however, given the high morbidity and pain associated with a transthoracic approach with a thoracostomy tube, most surgeons believe that the Belsey-Collis procedure has become obsolete, except in specific circumstances where prior transabdominal repairs have already failed or there is a history of other major abdominal surgery. Though technically challenging, advanced minimally invasive foregut surgeons have demonstrated that even the total intrathoracic stomach may be reduced laparoscopically with success and adequate esophageal length. Esophageal shortening due to chronic reflux, inflammation, and fibrosis is not common, but does need to be addressed with a Collis gastroplasty procedure, as this is a risk factor for

recurrence. In cases where there have been multiple previous attempts and recurrences, the authors favor a left thoracoabdominal incision for maximal exposure. Equivalent outcomes between transthoracic and transabdominal repair have been shown in terms of recurrence, though no randomized trial comparing the two has been published to date [20]. The authors are not aware of any minimally invasive thoracic approaches to PEH repair that are routinely practiced or well described in the literature at this time.

6.4.3 Hernia Sac Excision Versus Simple Reduction

The fundamental tenets of PEH repair to prevent recurrence, regardless of approach, include (1) tension-free reduction of the hernia with at least 2–3 cm of intra-abdominal esophageal length, (2) complete excision of the hernia sac, and (3) closure of the hiatus [21]. Dissection of the hernia sac off of the crura and mediastinum is a key component of successfully being able to reduce the stomach into its normal configuration in the abdomen when performing either a laparoscopic or open repair. In addition, the planes of dissection are often easier to visualize, especially laparoscopically, and injury to the wall of the esophagus, left gastric vessels, and vagus nerves may be more consistently avoided. In large, long-standing hernias where the sac may be completely fused with surrounding structures, at least partial excision of the sac is recommended to allow for more complete reduction of the hernia and possible performance of a wrap. Leaving a portion of the hernia sac attached to the lesser curve often reduces blood loss, but the hernia sac should be completely dissected from the greater curve, fundus and gastroesophageal junction in order to be certain that normal anatomy has been restored. Failure to excise any of the hernia sac is associated with a higher early recurrence rate. In 1998, Edey

et al. reported a 20% recurrence rate within 8 weeks without excision of the hernia sac [22].

6.4.4 Primary Repair Versus Mesh Repair

Recurrence rates following PEH repair with primary closure of the hiatus have been quoted to be as high as 42% in studies where patients have been followed over the long term [12]. Many of the recurrences are small, asymptomatic, and less than 5 cm in size, however. Hence, it is important to discriminate between the radiographic and clinically significant recurrence rates. Figure 6.3 demonstrates our preferred technique of primary hiatal closure using double-pledgeted sutures. In order to reduce the recurrence rate, many surgeons have used mesh reinforcement of the crural closure, especially if there is any degree of tension or the hiatal defect is large (greater than 5 cm). Of note, the normal hiatus is, on average, 2.4 cm in size [23]. Three techniques have been described that incorporate mesh into the crural closure: (1) reinforcement technique where the crura are approximated primarily and the mesh is placed in an onlay fashion to reinforce the repair using stitches to the crura to keep the mesh in place while fibrosis occurs (Fig. 6.4); (2) bridging technique where mesh is interposed between the crura and is sewn to each crus, so as to eliminate tension altogether; and (3) a keyhole technique where a hole is cut in the mesh so that it can be placed almost circumferentially around the esophagus [24]. In addition to the deciding which of these techniques is appropriate, the surgeon must also decide which type of mesh to use: absorbable/biologic, polypropylene, or polytetrafluoroethylene (PTFE).

There are concerns regarding long-term erosion of mesh into the wall of the esophagus or stomach, which can result in a rare but extremely challenging situation for the surgeon. Removing the mesh and performing a reconstruction is a major undertaking and represents a life-threatening scenario

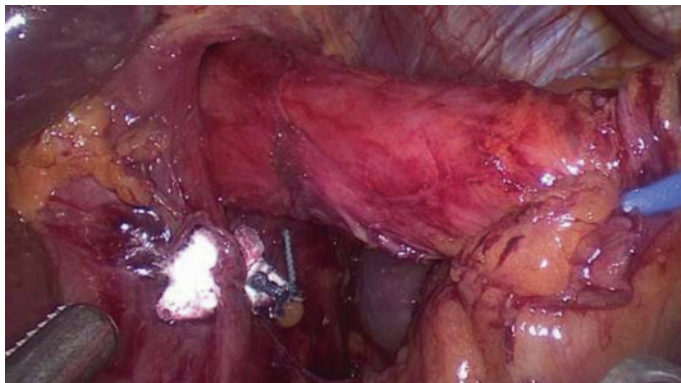


Fig. 6.3 Primary closure of hiatus, reinforced with a double-pledgeted suture

for the patient. An esophagectomy may be ultimately required after the mesh and affected tissues are removed to control sepsis. Due to this possibility we believe that synthetic mesh should be avoided in patients under age 50. Newer biologic mesh onlays made of porcine submucosa or acellular human dermis, on the other hand, are usually resistant to infection and become incorporated into native tissues over time without excessive scar formation. Multiple reports claim that mesh reinforcement of hiatal closure significantly reduces the recurrence rate. Furthermore, a randomized controlled trial conducted with biologic mesh initially supported these claims, with no reported complications related to mesh. However, a 5-year follow-up of these patients showed similar rates of recurrence whether mesh was used or not, and hence the value of biologic mesh as pertains to long-term recurrence rate is questionable [25]. At this time, while short-term data does support the use of mesh, longer-term data does not. Furthermore,

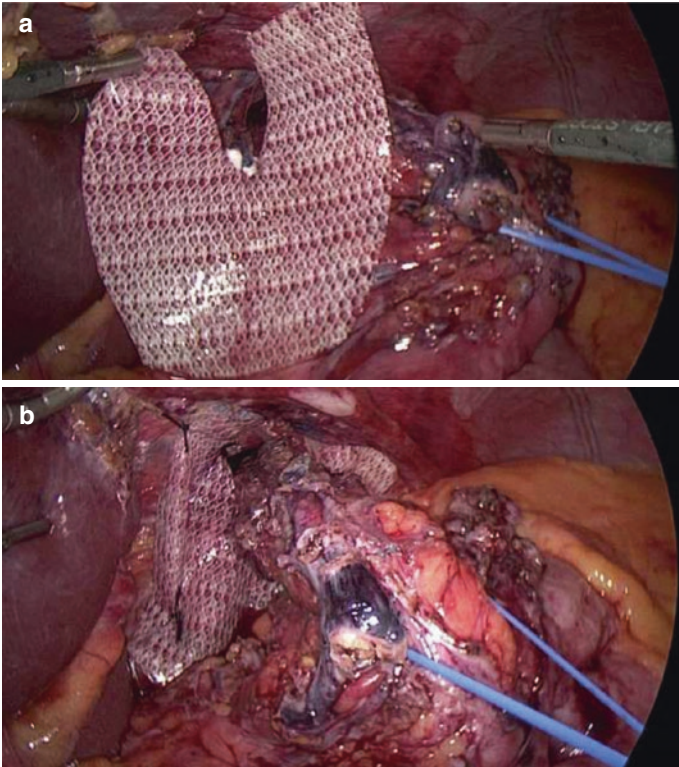


Fig. 6.4 The use of synthetic mesh to reinforce the hiatus. **(a)** U-shaped Mesh prior to fixation. **(b)** Mesh after fixation

in the setting of emergent repair of PEH for strangulation or perforation where there is any concern for contamination of the mediastinum or peritoneal cavity, mesh is generally to be avoided. The use of a biologic mesh may be acceptable, however, if necessary to help salvage a difficult hiatal closure.

A better alternative that we prefer to employ for difficult crural closure is the use of relaxing incisions. It is safe to make a longitudinal incision in the right crus to allow the medial portion to slide centrally and then be primarily sutured to the left crus. One can then patch the defect in the right crural muscle with a small piece of absorbable mesh or even leave the defect open, as it is almost always covered by the caudate lobe of the liver. Others have described making a relaxing incision in the left crus in a similar fashion.

6.4.5 *Gastropexy Versus Wrap*

Traditionally, an anti-reflux procedure is performed concomitantly with PEH repair as a method to anchor the stomach in the abdomen and also to reconstruct the lower esophageal sphincter mechanism as a barrier to reflux. The rationale is that following full dissection and mobilization of the gastroesophageal junction, lower esophagus, and hiatal attachments, the geometry of the lower esophageal sphincter has been significantly disrupted and is thus rendered incompetent. The rate of postoperative reflux has been reported to be as high as 65 % following PEH repair without fundoplication, though most argue that symptomatic reflux is far less common, and can be managed with medical therapy if it persists [26]. Because many of the patients who require PEH repair are elderly and have impaired esophageal motility a full 360° fundoplication may predispose to postoperative dysphagia. Hence many surgeons advocate for at least a partial 270° wrap, such a Toupet or Dor fundoplication, particularly if there is a significant history of GERD in order to minimize dysphagia. Although many surgeons believe fundoplication adds bulk and gastropexy to the hiatal hernia repair, the impact of re-herniation after fundoplication has not been examined in a robust fashion in any published data to the authors' knowledge.

In the case of the elderly patient or patient that requires a complex or emergent operation for acute presentation of PEH, a fundoplication is even more difficult to justify. It can prolong the operation significantly, which may be of critical importance in unstable patients with multiple or severe comorbidities. Two retrospective case-controlled studies demonstrate minimal benefit to performing fundoplication routinely as a part of paraesophageal hernia repair [27, 28]. Thus, gastropexy or gastrostomy is preferred under these circumstances. Although recurrence rates associated with gastropexy are high, it can be lifesaving. If gastropexy alone is performed without hiatal closure and sac excision, recurrence is reported to be 23% at 3 months [29]. Gastropexy involves fixation of the greater curve of the stomach to the diaphragm and abdominal wall in the left upper quadrant anterior to the spleen after all of the short gastric vessels are divided. The goal should be restoration of the normal anatomic position of the stomach without tension. In order to successfully fix the stomach in the abdomen, adequate intra-abdominal esophageal length of 2–3 cm is still required even in the absence of fundoplication (Fig. 6.5).

The major causes of re-herniation are related to increased intra-abdominal pressure postoperatively, lack of tension-free closure of the hiatus, incomplete dissection and removal of the hernia sac, and inadequate intra-abdominal esophageal length. The addition of a Collis gastroplasty to lengthen the esophagus is recommended when the esophagus appears foreshortened. Lower recurrence rates have been published when concomitant gastroplasty is performed. However, there is a non-trivial risk of leak from the gastroplasty staple-line, which is estimated at 3% [30]. One must also be cognizant of the risk of ischemic stricture when Collis gastroplasty is performed in an unstable patient.

In the scenario of emergent or urgent surgery in the elderly or debilitated patient, if there is concern for sufficient intra-abdominal esophageal length even after extensive mediastinal dissection of the esophagus, placement of a gastrostomy tube

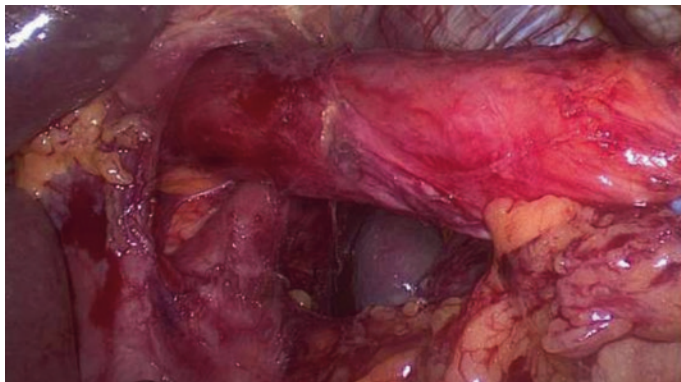


Fig. 6.5 Obtaining adequate intra-abdominal esophageal length

should be considered. The benefits of gastrostomy tube placement include enteral access and decompression of the stomach postoperatively, which can be useful especially if the vagus nerves are damaged or divided inadvertently and delayed gastric emptying becomes an issue.

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Chapter 7

Upper Gastrointestinal Haemorrhage

Michael D. Kelly

7.1 Introduction

Upper gastrointestinal haemorrhage (UGIH) is a frequent cause of acute hospital admission, and in the United Kingdom, it accounts for 70,000 admissions per year with the majority of cases being non-variceal [1] (Table 7.1). Gastroduodenal (peptic) ulcers are the most common cause and account for well over 50% of admissions. This is despite the ready availability of proton pump inhibitors and the recognition of the role of *Helicobacter pylori* and nonsteroidal anti-inflammatory drugs (NSAIDs) in their genesis.

Although there have been significant advances in endoscopy and interventional radiology (IR), UGIH remains a significant cause of morbidity and mortality. In fact, the 30-day mortality seems unchanged at a level of around 11% as the patients tend to be older with more serious co-morbidities [2, 3]. Management has evolved over the last few decades with fewer cases requiring surgery, but therapy will depend on the place of treatment as

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Table 7.1 Aetiology of upper GI haemorrhage

Peptic ulceration (gastric or duodenal)
Erosive gastritis
Ulcerative reflux oesophagitis,
Mallory-Weiss tear
Dieulafoy lesion
Oesophageal varices
Carcinoma (stomach, duodenal, oesophageal)
Ulcerated gastrointestinal stromal tumour (GIST) (gastric or duodenal)
(Fig. 7.1)
Gastric ectasias
Cameron lesions
Barrett's ulcer (Fig. 7.2)
Splenic artery haemorrhage (pancreatic pseudocyst, aneurysm)
Aortoenteric fistula (Fig. 7.3)

**Fig. 7.1** Endoscopic view of an ulcerated GIST. The patient presented with haematemesis and melaena

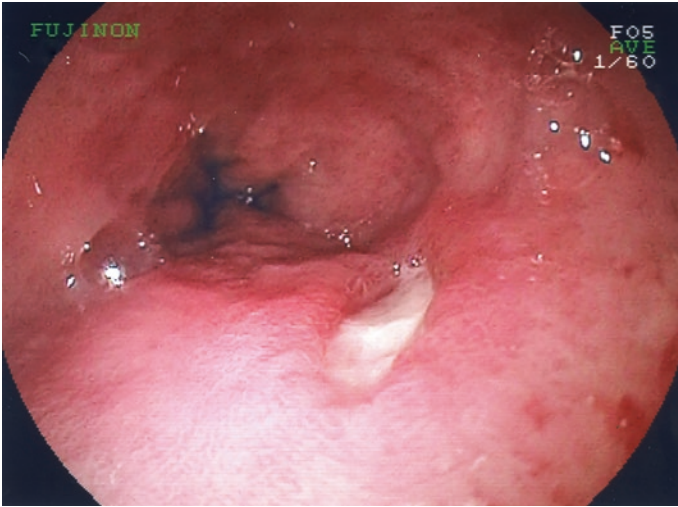


Fig. 7.2 Ulcer in a segment of columnar-lined oesophagus (Barrett's ulcer); a rare cause of an acute upper GI haemorrhage

smaller hospitals may not have the equipment or expertise to use the most modern techniques. In addition, in some health systems, gastroenterologists manage the patient, and surgical involvement is limited to severe cases requiring operation that have failed endoscopy and IR.

UGIH is defined as bleeding proximal to the ligament of Treitz and may present with melaena or haematemesis. Milder forms may present with anaemia and non-specific symptoms. The majority of cases are due to peptic ulceration, but the initial management and general response to non-surgical treatments are similar irrespective of the aetiology. This does not include variceal haemorrhage due to portal hypertension as this requires a different approach from the outset although patients with varices can also bleed from other lesions.

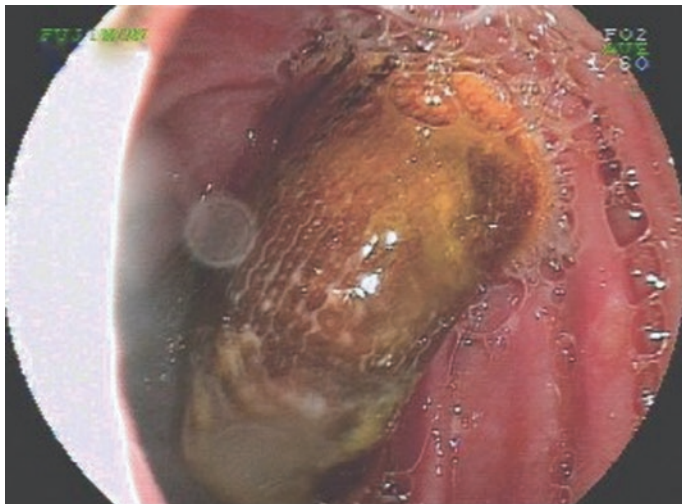


Fig. 7.3 Aortoenteric fistula in the distal duodenum due to knitted polyester (Dacron) graft which had ulcerated through the intestinal wall. The patient presented with melaena and the first endoscopy had not detected the problem as the surgeon only went to the second part of the duodenum. Once a diagnosis of aortoenteric fistula is entertained, discussion with a vascular surgeon is mandatory, and emergency surgical intervention will usually be required. In most instances, the aortic graft is removed with closure of the duodenum, followed by an extra-anatomic vascular bypass to revascularise the lower extremities. The perioperative mortality rate is high, and major complications are common. Lesser alternative procedures such as not removing the graft primarily or endovascular stenting as a bridge to more definitive treatment in a high-risk patient have been described [26]

There still remain a small number of patients who require emergency surgery, and for the emergency general surgeon, this can be quite a challenging problem due to a lack of experience with elective gastroduodenal surgery.

7.2 Initial Management

The classic clinical presentation of a significant UGIH is a patient with haematemesis and melaena. A full medical history should be taken including a detailed medication history. Ulcerogenic drugs (NSAIDs, aspirin, steroids), antiplatelet drugs and anticoagulants (warfarin, rivaroxaban) and B-blockers are especially important to note. Discussion with haematologist should be undertaken in those who are on the newer antiplatelet or anticoagulant medications or when massive transfusion is likely.

All standard resuscitation measures should be instituted including transfusion. A restrictive transfusion policy aiming for a haemoglobin of 70–80 g/L is suggested in haemodynamically stable patients [4]. Gastric acid suppression should be achieved by intravenous proton pump inhibitor (PPI) bolus followed by infusion (e.g. omeprazole 80 mg stat then 8 mg per hour for 72 h). There appears no place for routine use of nasogastric tubes or lavage, antifibrinolytics (e.g. tranexamic acid) or octreotide. There is some evidence that erythromycin acting as a prokinetic (motilin agonist) improves visualisation at emergency endoscopy [4]. CT (computed tomography) angiography has a role in the subgroup of patients who are suspected of having an unusual cause of UGIH, e.g. tumour or aortoenteric fistula.

7.3 Endoscopy

Endoscopy is the critical step in managing patients with UGIH. It allows diagnosis, gives prognostic information and can control bleeding. It has been shown in randomised studies of peptic ulcer to lead to a reduction in blood transfusion, shortened intensive care unit and hospital stay, decreased need for surgery and lower

mortality rate [5]. Endoscopic haemostasis is now accepted as the first-line treatment in patients with active bleeding. Several controlled trials have shown that endoscopic therapy using a variety of combined techniques significantly reduces the need for blood transfusion and emergency surgical intervention [6]. While most studies relate to peptic ulceration, endoscopic therapy has been shown to be effective in treatment of other causes, in particular, Dieulafoy or Mallory-Weiss lesions.

The Forrest classification of endoscopic appearance of peptic ulcers is used to give prognostic information: F1a spurting haemorrhage, F1b oozing haemorrhage, F11a non-bleeding visible vessel, F11b adherent clot, F11c flat pigmented spot and F111 clean base [7]. Endoscopic therapy should be used when there is active bleeding or a visible vessel. For F11b lesions, there is no consensus as studies have conflicting results, and the decision whether to remove the clot to allow therapy should be made on a case-by-case basis. The therapy will depend on the experience of the endoscopist and the equipment available and include injection of epinephrine, heater-probe coagulation, bipolar electrode coagulation, laser coagulation, argon plasma coagulation, endoscopic clips and banding devices [8, 9].

Patients presenting with an UGIH who stabilise quickly with minimal resuscitation should have endoscopy within 24 h [10]. Debate continues on the relative merits of emergency endoscopy in these patients as there is increased risk of the procedure and it may be suboptimal due to blood and clots making therapy difficult [11]. Patients who are well and are found to have low-risk ulcers probably do not need inpatient monitoring. The use of risk scoring systems may help to identify the low-risk patients suitable for outpatient management; however, a recent study from Denmark found that none of the popular risk scoring systems examined were suitable for predicting risk of rebleeding or 30-day mortality, possibly due to inter-country variation in patient characteristics [2].

Patients with severe, life-threatening haemorrhage not responsive to resuscitative efforts need to be transferred to the

operating room for emergency endoscopy. These patients should have endotracheal intubation and preparations made for laparotomy. Emergency operation should be carried out if the endoscopist cannot stop the bleeding and there is no option for IR with angioembolisation or infusion therapy [12].

Up to 15% of endoscopically treated patients experience recurrent bleeding [13]. Pre-endoscopic predictors of rebleeding are haemodynamic instability and co-morbid illness. Endoscopic predictors of rebleeding are active bleeding at endoscopy, large ulcer size, posterior duodenal ulcer and lesser gastric curvature ulcer [13].

Several studies have looked at patients at high risk of rebleeding and whether they should have early elective or planned operation. A randomised trial of patients with initial endoscopic control of ulcers with arterial bleeding or large visible vessel found that 4% of the surgery group rebled versus 50% in the repeat endoscopic treatment group although the mortality rate was the same [14]. Other prospective studies have shown that early planned surgery in high-risk groups may be beneficial [15]. It is likely that the endoscopic techniques in these studies are not current best practice and there would appear to be no place for routine operation or IR in patients who have had successful endoscopic haemostasis. Similarly most authorities do not recommend routine second-look endoscopy although it should be considered in high-risk patients [4].

For patients that do rebleed after initial successful endoscopic therapy, there is evidence from a randomised trial that repeat endoscopy is superior to surgery. In that study, endoscopy reduced the need for operation without increasing the risk of death, and there were fewer complications [16]. It is generally accepted that a second attempt at endoscopic haemostasis is indicated although there may be a subgroup where surgery may be a better option such as large posterior duodenal ulcer and shock at first presentation [15]. If available, IR has a role in this group and appears superior to surgery. A retrospective study of

118 patients for endoscopy refractory ulcer bleeding found it to have a lower mortality and lower rate of complications. Surgery was associated with a higher rate of primary haemostasis (100 % vs 91 %, $p = .007$) and a lower rate of rebleeding (15 % vs 40 %, $p = .004$) but also a higher rate of complications (60 % vs 38 %, $p = .02$) and an increased mortality when adjusting for confounding factors [17].

7.4 Surgery

The role of surgery in UGIH is now restricted to patients who fail primary endoscopic haemostasis or rebleed after having had two therapeutic endoscopies and where IR is not available or has itself failed. Endoscopic failure of haemostasis occurs in 5–10 % of patients due to reasons such as inability to get a view due to blood and clots, the ulcer position or that the vessel is too large to control with the available equipment and expertise [17]. In the rare case of a patient presenting with exsanguinating haemorrhage, urgent operation will be needed but usually an on table endoscopy is attempted to at least exclude varices.

While surgery plays a minor role in the overall management of the community of patients with UGIH, the surgery itself can be a significant challenge. Most emergency surgeons nowadays will not have had experience with elective surgery for peptic ulceration. This is then coupled with the fact that the cases that come to emergency surgery will tend to be difficult ulcers in poor-risk patients due to chronic co-morbidities who are acutely unwell after numerous endoscopic attempts at haemostasis over several days. Surgeons should intervene decisively and the operation should be tailored but, in principle, kept to the simplest procedure possible, i.e. conventional open surgery and underrun of a bleeding vessel.

Operations for UGIH today focus on safely arresting the haemorrhage and there is no place for acid-reducing operations sometimes called definitive surgery. Historically the surgical options for a bleeding duodenal ulcer included vagotomy (truncal or selective) and drainage procedures or gastric resection. Each of those operations was associated with an incidence of ulcer recurrence, postgastrectomy syndrome and mortality. With the availability of effective acid-suppressing drugs and knowledge of the role of *H. pylori*, logically there is no need for the surgeon to do a definitive acid-reducing procedure. Occasionally when operating for a chronic bleeding ulcer, a distal gastrectomy may be necessary, but even then the resection should be limited and undertaken for technical reasons and not for long-term acid control.

There are still surgeons that challenge this change in practice citing a lack of high-quality evidence and still feel there is a place for so-called definitive operations as a routine. In fact, there are published data to support their view, and Schroder and colleagues recently reported a retrospective study of emergency surgery for bleeding peptic ulcers. They found that vagotomy and drainage was superior to local oversew as it was associated with a significantly lower 30-day postoperative mortality rate (of note distal gastrectomy had a higher rate). They postulated that the decreased mortality observed among patients who underwent vagotomy/drainage was due to superior perioperative acid reduction (through vagotomy) and were therefore less likely than patients who underwent local oversew to have recurrent ulcer bleeding in the postoperative period. However, the difference in major postoperative bleeding rates between the two groups did not reach statistical significance and they could not confirm that the patients included in the analysis received appropriate medical therapy (proton pump inhibitors and/or *H. pylori* eradication) after their emergency ulcer oversew operation [18].

A recent much larger population-based cohort study from Sweden of 4163 patients compared definitive surgery with a

minimal approach for refractory peptic ulcer bleeding in relation to survival. This study found overall survival after minimal surgery was no worse and was associated with better long-term survival during the more recent study period and concluded that a minimal approach is sufficient in most cases [19].

While laparoscopic surgery for perforated peptic ulcer is well established, it appears not to have a role at present for UGIH and conventional open surgery is the standard of care when operation is required. A midline epigastric incision is made and if the source of bleeding had not been identified on gastroscopy, inspection and palpation may give a clue as to where the lesion lies. A longitudinal gastrotomy commencing from the prepyloric region through the pylorus to the first part of the duodenum is the standard and the incision can be easily extended proximally. If a gastric lesion is likely, then this initial incision does not need to cross the pylorus. Babcock-type forceps or stay sutures can be used to hold open the gastrotomy.

Clots and blood should be removed from the stomach and sometimes narrow Deaver-type retractors can be used to retract the edges of the gastrotomy to get a view of the mucosa especially proximally. A small lesion may be easier to feel than see, and this is classically the case for the Dieulafoy lesion palpable in the gastric fundus. When the lesion is found to be high in the stomach, for example, a Dieulafoy lesion or Mallory-Weiss tear at the OG junction, access can be difficult. A Goligher frame with a sternal hook to elevate the sternum can be used with a Deaver retractor to elevate the left lobe of the liver. It may be necessary to dissect around and mobilise the distal oesophagus, sometimes dividing some upper short gastric vessels taking care with the spleen. This is most easily done with a harmonic scalpel. A nylon tape can be slung around the distal oesophagus and with this the surgeon has control to assess and oversee any proximal lesion.

For bleeding gastric ulcers (GU), a gastrotomy with under-running of the bleeder with biopsy may be appropriate, while those in a favourable position, such as those away from the

lesser curve, local excision, may be a better option. A nasogastric tube is usually left in for 24 h.

The preferred operation today for a bleeding duodenal ulcer (DU) is to underrun the bleeder having exposed the lesion via a longitudinal duodenotomy which usually will have crossed the pylorus. This opening can be closed anatomically or with a pyloroplasty. I have always found that anatomical closure is easier and appears more secure and the theoretical risk of stenosis has not been borne out.

The gastroduodenal artery is usually the vessel visible in a large posterior DU and is ligated proximally and distally to the bleeding site. Numerous authors advocate a third suture as a horizontal mattress placed to control haemorrhage from the transverse pancreatic branch of the gastroduodenal artery. Some surgeons recommend using a non-absorbable suture such as polypropylene. There are some large penetrating posterior DUs or rarely the bleeding and perforated posterior DU that require a distal gastric resection due to the first part of the duodenum having been destroyed. This is a conservative resection done inside the gastroepiploic arcade to enable safe control of the acute pathological problem and not as a definitive antiulcer operation. Gastrointestinal continuity can then be re-established by either gastroduodenostomy (Billroth I) or gastrojejunostomy with closure of the duodenal stump (Billroth II). The former is preferred due to the better functional outcome; however, it can be a more technically challenging operation. Traditionally a drain has been left to the site of the duodenal closure in the Billroth II procedure and left for up to a week. Numerous methods have been described in the past for dealing with the difficult duodenal stump closure including forming a controlled fistula via a tube duodenostomy. These operations have the added advantage of excluding the bleeding vessel/ulcer base from the GI tract so rebleeding cannot occur. A feeding jejunostomy should be made in any high-risk case to enable early nutrition while protecting the suture line.

Millat and colleagues reported a randomised controlled trial comparing vagotomy and pyloroplasty with gastric resection for bleeding DU. They found that the rebleeding rate was higher (17% vs 3%) with vagotomy and pyloroplasty, but the overall mortality was not different [20]. Similarly a multicentre randomised prospective trial by Poxon and colleagues compared minimal surgery (underrunning the vessel or ulcer excision and adjuvant ranitidine) with conventional ulcer surgery (vagotomy and pyloroplasty or partial gastrectomy) for the treatment of bleeding peptic ulcer. Sixty-two received conservative surgery and 67 conventional operation. Twenty-nine patients died, 16 (26%) after conservative surgery and 13 (19%) after conventional operations. The only significant difference between the groups was the incidence of fatal rebleeding, which occurred in six patients (10%) after conservative surgery compared with none after conventional surgery ($P < 0.02$) [21]. These are historical results before the use of PPI and when operations were done to control acid, but they show that gastrectomy, if it can be done safely, is a very effective way to definitively stop bleeding from a difficult DU. Emergency partial gastrectomy in the elderly carries a higher morbidity and mortality rate, but a recent report showed an anastomotic leak requiring reoperation occurring in only 6% with no recurrence of DU disease [22, 23].

Rebleeding after operation can be a significant management problem. Historically, the rates have been relatively high when simple underrunning of the ulcer, leaving it in the GI tract, was coupled with inadequate acid suppression. Usually endoscopic treatment is not appropriate due to the immature suture line, and IR should be used if at all possible. When reoperation is needed, a more aggressive procedure would usually be taken with distal gastrectomy as described above, and a feeding jejunostomy should be formed.

Patients may require monitoring in an intensive care or high dependency unit depending on their co-morbidities and condition at the completion of the operation. Acid-reducing therapy

with PPI should continue intravenously. Consideration should be given to early feeding via a nasoenteric tube or jejunostomy that was placed at the time of surgery. For duodenal ulcers, empiric *H. pylori* eradication therapy can be used, and for gastric ulcers, a repeat endoscopy with biopsy should be done to exclude an underlying malignancy. Patients who require continuation of dual antiplatelet therapy (e.g. aspirin and clopidogrel) should remain on PPI although management should be individualised in consultation with their cardiologist [4]. Unusual or recurrent acid-peptic lesions require a serum gastrin level (off PPI) to exclude the Zollinger-Ellison syndrome (gastrinoma secreting abnormally high levels of gastrin). Patients who have had gastrectomy will require monitoring of their nutritional state and may require iron and vitamin B12 supplementation.

Uncommon causes of UGIH, i.e. those not due to peptic ulceration, that come to operation have to be dealt with on a case-by-case basis, but the general principles include adequate exposure and assistance, secure haemostasis and distal enteral nutrition.

7.5 Variceal Haemorrhage

Patients with acute variceal haemorrhage from portal hypertension require a different management strategy to those with non-variceal UGIH due to the underlying mechanism of the bleeding. Discussion with or transfer to a liver unit should be initiated. First-line treatment includes resuscitation, correction of coagulopathy or thrombocytopenia, vasoconstrictor drugs such as terlipressin and endoscopic band ligation. Failure to control bleeding or early rebleeding means second-line treatments should be instituted. These include balloon tamponade and insertion of a transjugular intrahepatic portosystemic shunt (TIPS), and recently there are promising

reports of insertion of removable covered self-expandable metallic oesophageal stents. Balloon tamponade achieves haemostasis by direct compression of the bleeding varices and is effective in 80 % of cases but requires close monitoring due to its high complication rate [24, 25]. In-depth discussion of the management of this complex problem is beyond the scope of this chapter.

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Chapter 8

Management of Perforated Peptic Ulcer

Kjetil Søreide and Kenneth Thorsen

8.1 Introduction

Perforated peptic ulcer (PPU) remains a formidable health burden worldwide and one of the most frequent emergency conditions requiring surgery [1]. The condition is associated with a rather high rate of fatal outcome although variation in mortality (from 6% up to 30%) is considerable among regions [2]. A steady decrease in overall gastroduodenal ulcer incidence was noted in the decades after the discovery of *Helicobacter pylori* as a causative bacteria and the introduction of acid-reducing drugs (first, the H₂-blockers and later proton pump inhibitors). Notably, the ulcer complications have not decreased to the same

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degree, and particularly the rate of perforations appears to be stable [3, 4]. While several factors may be involved in explaining this, including changing population demography and more widespread use of ulcerogenic drugs, the consequence for the surgeon is the continued need for emergency ulcer management [5], although with changes in frequencies compared to the past. Bleeding ulcers has seen a dramatic change in management, with the majority now managed by endoscopic techniques alone or, with the support of interventional radiologists using transarterial embolization techniques [6]. Contrary to the predominant nonsurgical approach to bleeding ulcers, the management of perforations is still done by surgical repair although new methods are being developed. Here we will present the current management of PPU based on best available evidence.

8.2 Clinical Presentation and Diagnosis

Sudden onset of acute, severe pain in the upper abdomen is a classical presentation of PPU in many patients. However, clinical presentation and lack of frank peritonitis should be noted in special populations, including extremes of age, particularly the elderly patient with several comorbidities, who may have a cognitive impairment and who takes ulcerogenic drugs that also may mask inflammatory response and peritonitis (such as steroids). It should be noted that up to one-third of patients may lack clear signs of peritonitis despite having a PPU [7]. In the past, most patients had an erect abdominal X-ray to look for free air, but the superiority and current availability of CT scanners have led to a shift in diagnostic work-up in many centers [8]. The very high sensitivity of CT scans (>95%) compared to abdominal X-ray (about 75%) allows for a more precise and rapid diagnosis, as well as detection of potential differential diagnoses. We thus routinely perform a CT scan in all patients, unless the patient presents with frank peritonitis and in septic

shock or pending shock that requires and emergent laparotomy. While we do not use the CT scans for determining indication for laparoscopy or laparotomy, others have suggested that CT findings may be utilized to avoid laparoscopy in perforations with difficult-to-access locations as well [9].

8.3 Preoperative Evaluation and Care

A thorough clinical exam should be accompanied with appropriate preoperative measures to optimize outcomes. Vital signs need to be monitored and sepsis recognized and treated. Adherence to a bundled care protocol for resuscitation, early start of broad-spectrum antibiotics, and pre- and postoperative optimization of care have reduced mortality in PPU from 26 to 17% in one study [10]. Numerous scores for prognostication have been suggested, but reliable and robust universal predictors are not available [11, 12]. However, the combination of high age, presence of one or more comorbidities, and delay to surgery remains negative predictors [13]. Futile surgery in the very elderly patients with reduced cognitive impairment who presents with ominous signs of organ failure or shock should be avoided [14].

8.4 Surgical Management

Surgery is generally performed by closing the defect with simple, interrupted sutures by abdominal access through either laparotomy or a laparoscopic approach. Evidence in favor of either method is lacking, and mortality is not influenced by the choice of repair [15]. Surgery should, independent from choice of access to the abdominal cavity, focus on localization of the defect, placement of appropriate and safe sutures, and addition of an omental patch for coverage (Figs. 8.1 and 8.2) [16].

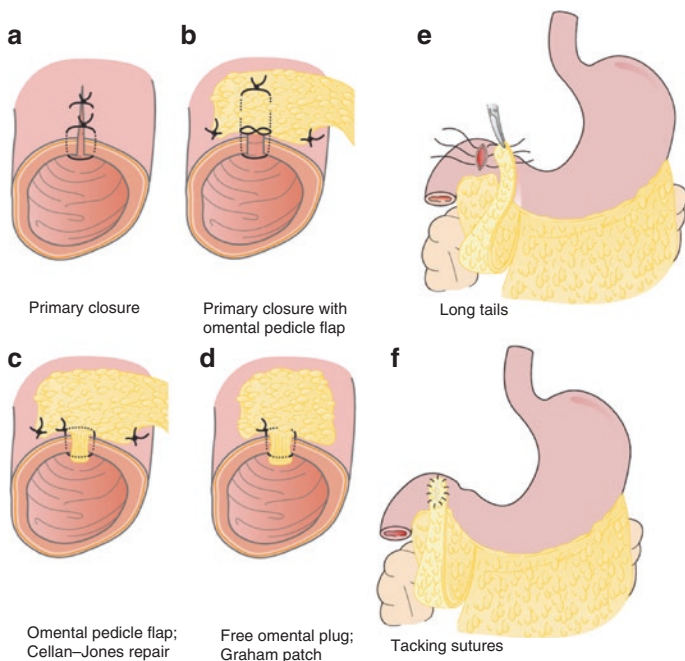


Fig. 8.1 Suture repair of PPU (Reproduced with permission from Søreide et al. [16] ©John Wiley & Sons)

8.5 Postoperative Management

Broad-spectrum antibiotics should be continued after surgery. Acid-reducing therapy with PPI should be given intravenously and a nasogastric tube kept in place as long as the patient is unwell. The nasogastric tube should be removed as soon as the patient tolerates fluids. The level of care should be considered according to the preoperative and postoperative state, but a considerable number of patients may require one or more days in a

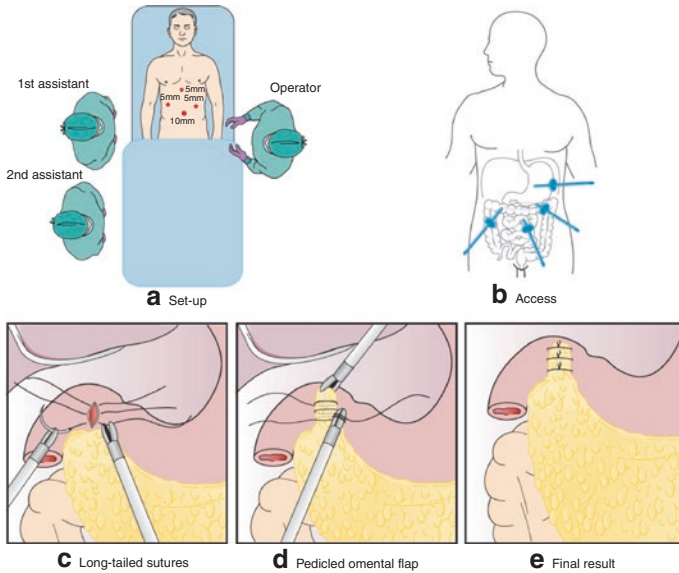


Fig. 8.2 Suggested laparoscopic surgery set up (Reproduced with permission from Søreide et al. [16] ©John Wiley & Sons)

high-dependency unit if not in the intensive care if on a ventilator. Obviously, the younger, fitter patients with a minor perforation and a rapid diagnosis and short delay to surgery may recover remarkably fast and be dismissed within 2–4 days. However, increasing age and added comorbid conditions make the length of stay likely more prolonged, often due to infectious complications and need for subsequent drainage of intra-abdominal abscesses in a contaminated abdomen. An active approach to mobilization and lung exercise should be implemented in the elderly, preferably guided by a physiotherapist, to facilitate recovery and prevent respiratory tract infections. Antifungal therapy is commenced in some patients with a

complicated course and with no improvement on broad-spectrum antibiotics alone, as opportunistic fungal infection is common. However, this has no effect on mortality based on available evidence [17].

For duodenal ulcers, empiric *H. pylori* eradication therapy is recommended [18]. For gastric ulcers, a subsequent endoscopy with biopsy should be done if biopsies were not obtained during primary repair, in order to rule out an underlying malignancy as cause of the perforation.

8.6 Nonoperative Management and Alternative Managements

One randomized trial has demonstrated the feasibility of nonoperative management (intra-abdominal drains, antibiotics, nasogastric tube, antisecretory drugs, nil per mouth) for PPU [19]. Notably, elderly patients fared worst with this approach. While several reports exist on the conservative approach to patients with minimal symptoms, small amounts of free air, and little involvement of clinical signs and inflammatory parameters, there are currently no good measures for appropriate patient selection. A number of endoscopic approaches are available to repair perforations in a more minimal-invasive manner, but most of these are either experimental, anecdotal, or based on very limited evidence [2, 20, 21].

8.7 Complex Situations and Alternatives

For very large defects or for failed repairs in a hostile inflammatory environment, there may be alternative options to the suture repair. One, a drainage strategy by means of a T-tube and

creation of a controlled fistula may be necessary in some cases, in particular in patients who will not tolerate a resection procedure [22]. Also, some would prefer a primary resection, such as distal gastrectomy, if needed. Of note, emergency gastrectomy in the elderly carries a higher morbidity and mortality rate [23]. In areas with a higher incidence of gastric cancers, such as in Japan, primary resection appears to be a more frequent approach to PPU. In the critically ill patient, a damage control strategy may be adopted and primary repair or complex surgery delayed until the patient recovers, but evidence supporting this is weak. [24].

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Chapter 9

Small Bowel Bleeding

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Abbreviations

CTE	Computed tomography enterography
DBE	Double-balloon endoscopy
DE	Deep endoscopy
GI	Gastrointestinal
GIB	Gastrointestinal bleeding
IOE	Intraoperative endoscopy

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MDCTA	Multidetector CT angiography
OGIB	Obscure gastrointestinal bleeding
PE	Push enteroscopy
sbB	Small bowel bleeding
SBE	Single-balloon endoscopy
SE	Spiral enteroscopy
VCE	Video capsule endoscopy

9.1 Introduction

Gastrointestinal bleeding (GIB) is a common cause of admission in the emergency departments.

Annual hospital admission for GIB in the United States and the United Kingdom is estimated at up to 150 patients/100,000 population, with a mortality rate of 5–10% [1]. Upper GIB has an annual incidence that ranges from 40 to 150 episodes/100,000 persons and a mortality rate of 6–10%; lower GIB has an annual incidence ranging from 20 to 27 episodes/100,000 persons and a mortality rate of 4–10% [1]. Acute GIB is more common in men than in women and its prevalence increases with age [1, 2].

It is estimated that upper, lower, and obscure GI bleeding account, respectively, for 50%, 40%, and 10% of total GI bleedings [1]; 5% of GIB occurs from the small bowel, between the ligament of Treitz and the ileocecal valve. The small bowel is the commonest cause of obscure GI bleed.

Small bowel bleeding (sbB) may present as occult or overt, persistent or recurrent bleeding; it can be massive, leading the patient to shock. The etiology is varied and it is highly determined by age, being tumors, as lymphoma; carcinoids and GIST, more likely in patients of less than 40 years of age; and vascular lesions, as angiodysplasia, more usual in elder patients, comprising 40% of all cases [3] (Tables 9.1 and 9.2).

Table 9.1 GIB's etiology, according to age

Age of the patients	Common causes of Mid GI bleeding
Pts <40 years of age	Meckel diverticulum, Dieulafoy's lesion, tumors (such as GIST, lymphoma, carcinoids, etc.), inflammatory bowel disease, celiac disease
Pts aged between 40 and 60 years	Small bowel tumors, angiodysplasia, celiac disease, NSAID-related lesions
Patients aged >60 years	Angiodysplasia, small bowel tumors, NSAID-related lesions

Table 9.2 Uncommon causes of small bowel bleeding

Aortoenteric fistula
Small bowel varices and/or portal hypertensive enteropathy
Inherited polyposis syndromes (FAP, Peutz–Jeghers)
Blue rubber bleb nevus syndrome
Henoch–Schoenlein purpura
Osler–Weber–Rendu syndrome
Kaposi's sarcoma with AIDS
Plummer–Vinson syndrome
Hemosuccus entericus
Pseudoxanthoma elasticum
Malignant atrophic papulosis
Hematemesis
Amyloidosis
Ehlers–Danlos syndrome

The commonest lesions responsible for sbB are vascular anomalies, such as angiodysplasia, telangiectasia, phlebectasia, arteriovenous malformations, Dieulafoy's lesion, and varices; tumors, inflammatory lesions, and medications represent other causes of bleeding (NSAID, aspirin, and anticoagulants).

In elder patients, under oral anticoagulants therapy, the risk of severe bleeding episode increased up to 4–23 %, being higher when INR was above 4. Risk factors associated with higher bleeding risk in this group of patients are:

- Age.
- A previous episode of GIB or peptic ulcer increases the risk up to 2.1–6.5 %.
- Comorbidities such as chronic kidney failure, diabetes, cardiac disease, and alcohol abuse.
- Association with antiplatelet drugs.

Some new anticoagulants have been developed with lower rates of intracranial bleedings but with a likely increase in GIB [3].

Small bowel tumors have been reported to be the second most common cause of sbB, accounting for 5–10 % of causes. Adenocarcinoma is the most common primary malignancy of the sb, accounting for 20–40 %, lymphomas 14 % and sarcomas 11–13 %. Adenocarcinomas are more common in the duodenum and proximal jejunum, whereas lymphomas and carcinoid tumors are most frequently located in the distal sb; the sarcomas are evenly distributed throughout the sb.

Sb ulcers or intestinal mucosa's erosions are another important cause of GIB. These lesions can be found in the Crohn's disease, in the intestinal tuberculosis, and in the NSAID enteropathy. The prevalence of sb ulcers increases with age [4].

There are various other less frequent causes of sbB such as radiation enteritis, mesenteric ischemia, endometriosis, and intestinal infestations by worms, especially in tropical countries [5].

Rare causes of mid-GIB (<1 %) are hemobilia, aortoenteric fistula (it has to be suspected for patients known to have an abdominal aortic aneurism or an aortic graft, until proven otherwise), and hemosuccus pancreaticus.

Hematochezia can be the clinical manifestation of both a lower GI bleeding and fast upper GI bleeding; melena can indicate bleeding occurring anywhere from the nose to the large bowel. Frank blood hematemesis suggests more active and severe bleeding in comparison to coffee-ground emesis. The

patient can present with abdominal pain and/or symptoms of anemia such as lethargy, syncope, and angina.

Generally anatomic and vascular causes of bleeding present with painless, large volume blood loss, whereas inflammatory causes of bleeding are associated with diarrhea and abdominal pain. The classical triad (Sandblom triad) of hematemesis, upper abdominal pain, and jaundice may point to hemobilia. Patients affected by hemosuccus pancreaticus typically present with intermittent epigastric pain in the abdomen, GIB, and hyperamylasemia. Painless bleeding may suggest vascular lesions, whereas painful bleeding may be due to small bowel tumors or NSAID-related GI injury.

9.2 Diagnosis and Management

9.2.1 *Primary Evaluation*

Rapid assessment and resuscitation should precede diagnostic evaluation in unstable patients (tachycardia, hypotension) with acute severe bleeding.

Early resuscitation provides immediate assessment of hemodynamic status of the patient with prompt intravascular volume replacement, initially using crystalloid fluids if hemodynamic instability exists, and restrictive red blood cell transfusion strategy, that aims for a target hemoglobin between 7 and 9 g/dL. A higher target hemoglobin should be considered in patients with significant comorbidity (e.g., ischemic cardiovascular disease) [6].

Initiating high-dose intravenous proton pump inhibitors (PPI), intravenous bolus followed by continuous infusion (80 mg then 8 mg/h), in patients presenting with acute GIB awaiting endoscopic evaluation are recommended [6].

When hemodynamic stability is assured, the standard of care for the initial diagnostic evaluation of GIB is urgent upper endoscopy and colonoscopy, if they are available, and the patient should be evaluated for the immediate risk of rebleeding and complications, if the source of the bleed is detected.

In literature, there are many score systems used to evaluate the risk of rebleeding and prognosis in patients presenting with acute upper gastrointestinal hemorrhage such as the Rockall score, based on endoscopic findings, and the Glasgow-Blatchford score for pre-endoscopy risk stratification based upon the patient's clinical presentation, but there are no studies available that evaluated their use in the patients with sbB [7].

Clinical and medication history (use of aspirin and NSAIDs, anticoagulants), physical examination and initial laboratory findings are important to localize the source of bleeding and the cause.

In case of patient affected by abdominal aortic aneurism or in patient with known aortic graft with clinical signs of GIB, aortoenteric fistula most commonly at the duodenum should be strongly suspected, and computed tomography of the abdomen or CT angiogram is mandatory to look for loss of tissue plane between the aorta and duodenum, contrast extravasation, and the presence of gas indicating graft infection [1].

9.2.2 Secondary Evaluation

The diagnosis and management of sbB is particularly challenging due to the length and complex loops of the intestine.

Early endoscopy (esophagogastroduodenoscopy and colonoscopy) are recommended to eliminate gastric and colonic sources of bleeding, when they are available [6–8].

If they are negative for sources of bleeding, following the diagnostic procedure selected depends on patient's symptoms, bleeding severity, local expertise, and availability [1–3].

No large randomized trials have demonstrated superiority of a particular strategy.

In hemodynamically stable patient, with risk of rebleeding, computed tomography (CT) should be done, when bleeding ileal tumor is suspected.

CT enterography (CTE) is a new noninvasive imaging technique that offers superior small bowel visualization compared with standard abdominopelvic CT, because it combines small bowel distension with a neutral or low-density oral contrast mixture and abdominopelvic CT examination, during the enteric phase following administration of intravenous contrast.

CTE shows a good visualization and better delineation of mural details, but its sensibility is operator dependent. The compliance of the patient is necessary to perform the exam: he has to be able to drink approximately 1.5–2 l of oral contrast over 45–60 min [4–9].

The limitation of CTE is that it cannot diagnose flat lesions such as ulcers, superficial erosions, and vascular lesions (angiodysplasias or AVM), but CTE detects small bowel tumors, especially those tumors with a predominantly exophytic component [4].

CTE can also help in localization of active bleeding as the presence of active GI bleeding would be seen as a focal area of hyperdense attenuation in the bowel lumen on plain scan or as focal area of contrast enhancement or extravasation into the lumen on a contrast-enhanced study [4]. In patients where active–intermittent gastrointestinal bleeding is suspected, a multiphase scan protocol can be used to identify sites of occult gastrointestinal bleeding. This protocol would frequently include pre-contrast, arterial, and delayed-phase CT examinations of the abdomen and pelvis, but CTE does not allow prolonged imaging times, which are necessary for detection of intermittent bleeding, and pre-existing high-attenuation material within the bowel may limit the detection of active bleeding.

Multidetector CT angiography (MDCTA) does not require bowel loop distension and is performed using intravenous contrast agent. It demonstrated a sensitivity of 86% and specificity of 95% in the evaluation of the patient with acute GIB. MDCTA shows the site of active bleeding as a focal area of hyperattenuation or contrast extravasation in the bowel lumen and has a higher sensitivity in detecting active hemorrhage, with bleeding rates as low as 0.3 mL/min being detected in animal models. This is better than the detection threshold of 0.5 mL/min of mesenteric angiography and is close to the detection threshold of 0.2 mL/min of RBC scintigraphy [1–4]. However, inability to perform therapeutic procedures is a major limitation of CT angiography (CTA). By demonstrating the precise site of bleeding and the etiology, the patient has to be direct to surgery or to catheter angiography.

Catheter angiography is both a diagnostic and therapeutic tool allowing the infusion of vasoconstrictive drugs and embolization and not requiring small bowel preparation. The sensitivity for a diagnosis of acute GIB is 42–86% with the specificity close to 100% [1, 2]. Sensitivity can decrease in case of intermittent bleeding, atherosclerotic anatomy, and venous or small vessel bleeding [1].

Complications, as access site hematoma or pseudoaneurysm, arterial dissection or spasm, and bowel ischemia, occur in 0–10% of patients with the incidence of serious complications occurring in <2% of patients [1–4].

All these diagnostic modalities require the administration of a contrast; consequently, they are contraindicated in patients with contrast allergy and renal impairment. The alternative is magnetic resonance enterography, with similar sensitivity and specificity rate.

9.2.3 Endoscopic Visualization of the Small Intestine

In 2015, the American College of Gastroenterology stated that video capsule endoscopy (VCE) should be considered as

first-line procedure for small bowel investigation, when it is available and not contraindicated [8].

VCE enables the complete small bowel visualization noninvasively.

Apostolopoulos carried out a prospective study on 37 patients admitted for GIB, to evaluate the role of capsule endoscopy in the diagnosis of active mild-to-moderate GIB, immediately after a negative upper endoscopy and ileocolonoscopy; he concluded that VCE appeared to have a high diagnostic yield in patients with acute, mild-to-moderate, active hemorrhage of obscure origin when performed in the hospital after a negative standard endoscopic evaluation and has important clinical value in guiding medical management [10]. Carey et al. evaluated 260 patients with OGIB and found the diagnostic yield of CE to be 53%, and this was higher in patients with obscure–overt (60%) GIB as compared with patients with obscure–occult GIB (46%) [11].

VCE is indicated in the investigation of suspected sbB, iron deficiency anemia, inflammatory bowel disease, celiac disease, nonocclusive small bowel tumors, and hereditary polyposis syndrome; it is contraindicated in patients with cardiac pacemakers and implantable cardioverter defibrillators because telemetry can interfere with VCE, in patients with organic or functional swallowing disorders, in patients with suspected small bowel obstruction for the increased risk of retention of the video capsule or with previous abdominal surgery, and in pregnant female patients because the potential teratogenic effects of transmitted microwaves [12, 13].

VCE findings were categorized into three types: lesions considered to have high potential for OGIB (P2), lesions with uncertain bleeding potential (P1), and lesions with no bleeding potential (P0). VCE evaluation is considered positive if one or more P2 lesions are found, and it is negative if only P1 or no abnormalities are found.

The primary limitation is that the technology is purely diagnostic and offers no therapeutic benefit such as obtaining

biopsies or administering therapy, besides directing further therapeutic measures.

Deep endoscopy techniques, such as push enteroscopy (PE), double-balloon endoscopy (DBE), single-balloon endoscopy (SBE), and spiral enteroscopy, allow the endoscopic observation of the small bowel beyond the angle of Treitz by a dedicated enteroscope.

PE consists in the passage of an enteroscope by mouth which makes possible the exploration of a variable length of the small bowel ranging from 30 to 160 cm beyond the angle of Treitz; it permits only a partial vision of the small bowel, but its main indication is OGIB, with a global diagnostic yield of 12–80% and better results in overt OGIB [3]. Complications are rare and include pancreatitis and mucosal injuries.

DBE enables all of the diagnostic and therapeutic interventions used in standard endoscopy – such as biopsy, polypectomy, and dilation – to be carried out anywhere in the entire small bowel. In literature, the diagnostic yield and treatment success of DBE for OGIB ranges from 60 to 81% and 43 to 84% [14]. DBE is restricted by its limited availability, prolonged times, and sedation requirements. The complication rate is 0.8% for diagnostic procedures and up to 4% for therapeutics such as polypectomy, electrocautery, or dilatation. Complications include bleeding, ileum, intestinal perforation, pancreatitis, or those related to sedation.

Comparative data for the SBE and the DBE, with regard to diagnostic yield and therapeutic impact, are equivalent, while DBE appears to be more favorable in relation to the complete enteroscopy rate.

SE utilizes a spiral-shaped overtube with a raised helix at the distal end. The main advantage of this technique is that it allows rapid advancement of the endoscope to the maximum distance [13–15].

Any method of deep enteroscopy can be used when endoscopic evaluation and therapy are required. VCE should be performed before deep enteroscopy if there is no contraindication and it is available [8].

We have to consider high costs of these investigation tools; limited availability; prolonged times and sedation requirements and the risk of rebleeding, which is high in elderly patients, often affected by multiple, vascular lesions; and the experience of the endoscopist.

Limitations of VCE in evaluation of small bowel pathology are limited to the visual field of the bowel lumen, poor bowel preparation, inadequate luminal distension, rapid passage around the proximal small bowel, and incomplete study of the cecum. For these reasons, VCE can miss significant lesions, with poor prognosis in patients with OGIB in the long term [16–19].

Although there have been many reports determining the clinical impact of negative VCE, the long-term risk of recurrent bleeding in patients with OGIB after negative VCE remains controversial.

Tan and Al retrospectively analyzed data of consecutive patients who underwent VCE for OGIB, with the aim to identify the risk factors associated with rebleeding and long-term outcomes after VCE; the overall rebleeding rate after VCE in patients with OGIB was 28.6% (97/339) during a median follow-up of 48 months; multivariate analysis of data showed that age ≥ 60 years, positive CE findings, hemoglobin ≤ 70 g/L before CE, nonspecific treatments, and the use of anticoagulants, antiplatelet, or nonsteroidal anti-inflammatory drugs after VCE were independent risk factors associated with rebleeding. Finally he concluded that VCE has a significant impact on the long-term outcome of patients with OGIB, but further investigations and close follow-up are necessary in patients with negative VCE findings [20].

Kim and Al retrospectively analyzed data from 125 patients who had received VCE for OGIB. Substantial rebleeding events were observed with similar frequency both after negative VCE without subsequent treatment (26.7%) and after positive VCE without specific treatment (21.2%) ($P=0.496$); he affirmed that rebleeding episodes were observed after negative VCE; consequently, further complementary diagnostic work-ups and close

follow-up are needed to be considered for patients with OGIB and negative VCE results [21].

Koh and Al carried out a prospective study on data from 95 patients followed up after being evaluated for OGIB by VCE. The overall rebleeding rate was 28.4%. The rebleeding rate was higher in patients with positive VCE (36.8%) than in those with negative CE (22.8%). However, there was no significant difference in cumulative rebleeding rates between the two groups. He showed that patients with OGIB and negative VCE have a potential risk of rebleeding, and he concluded that close observation is needed even in patients with negative CE, and alternative modalities should be considered in clinically suspicious cases [22].

There are no clear guidelines for evaluating patients with initially negative CE results. The management of these patients with OGIB remains controversial. However, patients with evidence of ongoing or recurrent OGIB need further investigation. The options include repeat upper and lower endoscopy, CE, DBE, radiology or nuclear medicine, and intraoperative enteroscopy [23].

9.3 Treatment of Small Bowel Bleeding and Outcomes

The clinicians have many therapeutic options in the management of sbB, and the decision depends on the age of the patient, the type of lesion responsible of the bleeding, the numbers of lesions, the risk of rebleeding, the degree of bleeding and anemia, comorbidities, and the severity of patient's clinical presentation on admission to the emergency department.

When the endoscopist detects the source of bleeding, and it is a vascular lesion, he can use:

- The argon plasma coagulation
- The endoscopic band ligation

All the patients endoscopically treated need multiple endoscopic exploration to assess the success of the therapeutic technique and eventually treat other lesions potentially at risk of bleeding.

The long-term rebleeding rates reported in literature ranges widely from 17 to 40%. Sakay and Al conducted a retrospective study in a cohort of 68 patients affected by bleeding from small bowel angioectasia, with the aim to evaluate the significance of endoscopic treatment and determine the long-term outcomes in patients with small bowel angioectasia. The results of the study indicated that initial endoscopic treatment was not sufficient to control the risk of rebleeding from small bowel angioectasia; this is probably because, in some patients, even if the lesions thought to be responsible for the bleeding are treated appropriately, other tiny, multiple, lesions can bleed later or because definitive lesions could be overlooked by the first CE examination in patients with OGIB. In fact small bowel angioectasia is reported to occur more frequently in the proximal small bowel than in the distal small bowel where the rapid capsule transit or reduced bowel visibility due to the presence of bile and bubble artifacts, small bowel lesions, can decrease VCE sensibility [24].

Pharmacologic therapy [5] can be used in case of multiple vascular lesions and in patients judged unfit for surgery or repeated endoscopy (high ASA score).

Hormonal therapy is based on the administration of estrogen and progesterone for the treatment of vascular malformations in the GI tract, originated from the treatment of hereditary hemorrhagic telangiectasia (HHT).

There is no definitive evidence for the efficacy of hormonal therapy in GIB that is unrelated to HHT.

Anti-angiogenics as thalidomide and bevacizumab are studied in the treatment of GIB, and although results are promising, there are significant adverse events associated with their use such as leukopenia, deep vein thrombosis, and peripheral neuropathy.

Octreotide, a somatostatin analogue, has the ability to inhibit the production of intestinal enzymes, decrease splanchnic blood flow, decrease platelet aggregation, and decrease angiogenesis. In literature it shows good outcomes, but more studies are needed to evaluate its success rate in the treatment of sbB and its side effects (diarrhea, abdominal discomfort).

9.4 The Role of Surgery

Surgery is indicated in patients who have failed medical and endoscopic therapy, in patients treated by angiographic embolization complicated by intestinal ischemia, and in patients who present with massive hemorrhage and persistent hemodynamic instability after resuscitation.

Preoperative localization of the source of bleeding allows surgeons to make a curative resection. Intraoperative localization of the source of bleeding is possible by enteroscopy.

Intraoperative endoscopy (IOE) has been considered the gold standard for small bowel examination for long time; today it is an important tool in emergency setting.

It can be accessed either by open laparotomy or by laparoscopic-assisted technique [4]. IOE consists in the insertion of the endoscope through an enterotomy, after small bowel lavage by cold water, exploring the mucosa while the surgeon facilitates the advance of the endoscope and observes the serosal surface. Palpation and transillumination play an important role in this procedure, which allows the whole bowel examination in more than 90 % of patients. The endoscopes which are preferred for this purpose are the gastroscope or pediatric colonoscope. The intraoperative detection of the bleeding lesion decreases the length of bowel to resect. If no lesion is found by IOE, surgeon can make a temporary diverting ileostomy to understand if the origin of the bleeding is in the upper or in the lower GI tract. In

this case, a second-look upper GI endoscopy and colonoscopy are indicated. IOE has a diagnostic yield of 50–100% with therapeutic possibilities, but it is invasive, with 12–33% of complications and a mortality rate of 8% [3].

9.5 What to Do in Emergency Setting? Proposal of a Diagnostic Algorithm

At the admission, the patient with suspected small bowel bleeding has to be evaluated for hemodynamic instability: the patient instable needs to be resuscitated; when his hemodynamic stability is assessed, the initial evaluation starts with upper endoscopy and colonoscopy. If they are negative for sources of bleeding, in the patient presented with massive hemorrhage at the admission, temporarily stable, urgent CTA can be performed to detect bleeding sources and eventually guide surgical treatment; surgery associated with IOE is indicated when there is a high risk of rebleeding.

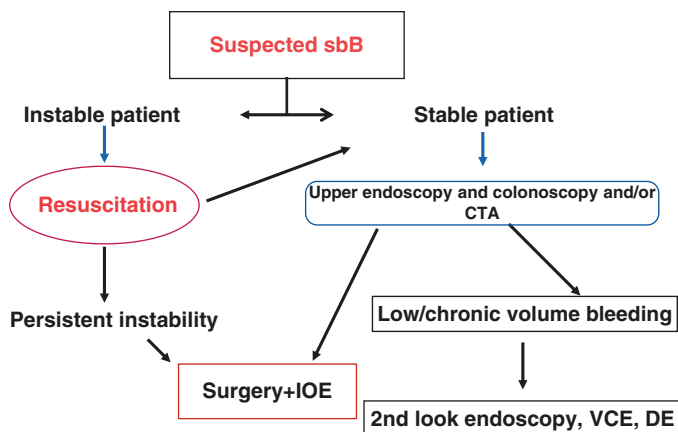
In the patient with intermittent bleeding or low volume bleeding, we can consider evaluation by VCE, DE, and endoscopic therapeutic strategies.

The hemodynamic unstable patient, nonresponsive to resuscitation, has to undergo surgery associated with IOE (Table 9.3).

9.6 Appendix: Reminder Keywords: Definitions

The GI bleeding is classified into:

- *Upper GI bleeding* includes hemorrhage originating from the esophagus to duodenojejunal flexure.
- *Lower GI bleeding* originates from a site distal to the ligament of Treitz.

Table 9.3 Proposal of a diagnostic – therapeutic algorithm

sbB small bowel bleeding, *CTA* computed tomography angiography, *IOE* intra-operative endoscopy, *VCE* video capsule endoscopy, *DE* deep endoscopy

- *Mid-GI bleeding* indicates the bleeding that occurs between the papilla and the ileocecal valve.

GIB can be:

- *Overt or acute*: signs of overt GI bleeding are hematemesis, coffee-ground emesis, melena, or hematochezia. The bleeding can be active and acute or inactive and chronic.
- *Occult or chronic*: defined by the American Gastroenterological Association as “the initial presentation of a positive fecal occult blood test (FOBT) result associated or not with iron deficiency anemia, when there is no evidence of visible blood loss to the patient or clinician”.
- *Obscure*: it is defined as “persistent or recurrent bleeding associated with negative findings on upper endoscopy and colonoscopy”; it can be classified into obscure–overt and

obscure–occult bleeding, depending on the presence or the absence of clinically evident bleeding.

Hematochezia: passing of red blood from the rectum.

Melena: black tarry stools.

Hematemesis: vomiting of fresh blood.

Small bowel bleeding (sbB)=*obscure GI bleeding (OGIB)*

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Chapter 10

Colorectal Bleeding Emergencies

Leslie Kobayashi and Raul Coimbra

10.1 Introduction

Approximately 60–80 % of lower GIBs are due to colorectal causes [1]. CRB primarily presents in an insidious fashion with hemoccult-positive stools or anemia, but can present acutely with hematochezia or bloody diarrhea. The most common cause of CRB is diverticulosis accounting for 20–65 % of lower GIBs and becomes increasingly common as age increases [1–4]. Other common causes include tumors/polyps, colitis (infectious, inflammatory, and ischemic), and anorectal disorders such as hemorrhoids, solitary rectal ulcer, and anal fissures. Solitary rectal ulcer is particularly common among patients in the intensive care unit presenting with new-onset hematochezia, accounting for 32 % of these cases [2]. Polypectomy and other colonoscopic procedures are also frequent causes of in-hospital CRBs. CRB is less common than upper GIB and is more com-

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mon among men and the elderly [1]. Nearly 80% of patients presenting with CRB will resolve without intervention; however, the recurrence rate can be as high as 25–46% [5–7]. Severe bleeding is less frequent in CRB compared to upper GIB, and mortality is expectedly lower, ranging from 2 to 4%, but can be increased in high-risk groups including the elderly, those on anticoagulants, CRBs due to malignancy, and in patients with significant comorbidities [1, 6, 8].

10.2 Resuscitation

The first step in managing patients with an acute CRB is assessment of the severity of hemorrhage and timely initiation of aggressive resuscitation in those with severe bleeding (Fig. 10.1). Patients who are hemodynamically unstable, those with severe anemia or significant transfusion requirements, and those with severe comorbidities, on anticoagulants, or are coagulopathic due to cirrhosis or other diseases should be considered to have severe hemorrhage [1]. These patients should be treated immediately with placement of two large-bore peripheral intravenous lines or placement of a central line. Early transfusion of blood and blood products should be initiated to rapidly restore hemodynamics. Patients who are coagulopathic due to pharmacologic treatment or renal or hepatic disease should be aggressively reversed with plasma, vitamin K, and platelets. Consideration should also be given to the use of adjunctive agents such as desmopressin, prothrombin complex, and tranexamic acid [2, 9, 10]. A Foley catheter should be placed to monitor ongoing resuscitation, and strong consideration should be given to placement of an arterial line for hemodynamic monitoring. In patients who are altered or may not tolerate sedation required for endoscopy, early intubation should be considered. Lastly, labs should be sent immediately including a type and cross, complete blood count, arterial blood gas, PT/INR, aPTT, and thromboelastography.

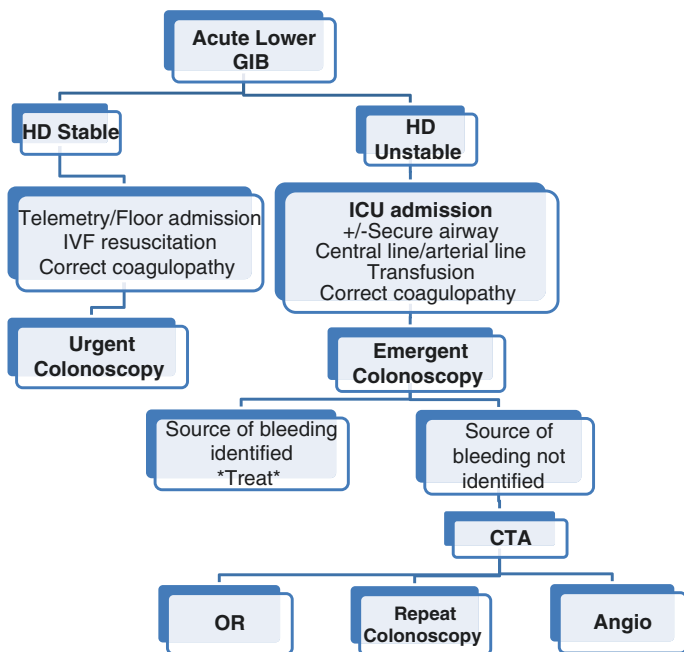


Fig. 10.1 Treatment algorithm for acute LGIB (Adapted from Kobayashi et al. [36])

After stabilization of the patient and airway protection if required, a careful history should be taken from the patient or family members. Attention should be focused on the use of anticoagulants and nonsteroidal anti-inflammatory drugs (NSAIDs) as these are associated with higher risks of rebleeding, complications, and death. Patients should also be asked about personal/family history of malignancy and inflammatory bowel disease (IBD) and changes in weight or bowel habits. Physical exam should include an abdominal and digital rectal exam with anoscopy looking specifically for hemorrhoids, fissures, ulcers, and masses.

Key Point

Initial management of acute CRB should begin with the establishment of adequate intravenous access, fluid resuscitation, and judicious transfusion of blood and blood products to ensure hemodynamic stability and reverse coagulopathy.

10.3 Diagnosis and Localization

Upper endoscopy or, if unfeasible or unavailable, nasogastric lavage should be performed to rule out upper GI sources of bleeding which may occur in up to 9–11 % of cases of hematochezia [1, 11]. Once upper GIB has been ruled out, colonoscopy should be performed as soon as possible, ideally within 12–24 h of admission, as early colonoscopy is associated with a significantly higher diagnostic yield. Early colonoscopy is also associated with decreased length of stay, transfusions, and cost [2–4, 12–16]. Diagnostic accuracy of colonoscopy ranges from 74 to 100 % depending on timing, rate of hemorrhage, and bowel preparation [1, 16]. Bowel preparation significantly improves diagnostic yield, appears to be safe even in the acute setting, and may reduce risk of endoscopic perforation. Because of this bowel preparation prior to urgent/emergent colonoscopy has been recommended by the American Society for Gastrointestinal Endoscopy (ASGE) [1, 2, 7]. A polyethylene glycol (PEG) solution at a rate of 1 l every 30–45 min should be administered via nasogastric tube until all fecal material is cleared. Bowel preparation appears to be well tolerated in the majority of patients [4]. Colonoscopy should be performed within 1–2 h of completion of the bowel preparation [2, 17]. Overall complications associated with colonoscopy are low, even in the emergent setting with a rate of perforation of 0.6 % and total complications of 3 % [1, 7, 14, 16].

If a bleeding source cannot be identified or colonoscopy is not possible due to lack of availability or patient factors, computed tomography (CT) scan and angiography are both good options in the acute setting. Recent improvements in imaging technology have improved the sensitivity and specificity of CT angiography (CTA). CTA has a sensitivity ranging from 91 to 92% in acute bleeding and may be able to detect bleeding as slow as 0.3 to 0.5 mL/min [1]. CTA also has the additional ability to identify extraluminal sources of hemorrhage [18]. Angiography requires rapid blood loss (0.5–1 mL/min) to be detectable, making it ideal for acute CRB, and unlike CTA it has the potential to be both diagnostic and therapeutic [7]. Diagnostic yield is variable from 40 to 86%, with yields increasing in acutely bleeding and hemodynamically unstable patients [1, 7, 14]. Direct comparisons between endoscopy and radiographic studies are few, but suggest that endoscopy has a higher diagnostic yield than angiography and a similar yield to CTA [13, 14, 19].

Key Point

Rapid bowel preparation with PEG solution and colonoscopy should be performed within 12–24 h of presentation.

Key Point

Patients unable to tolerate colonoscopy and those with nondiagnostic endoscopy should undergo rapid CTA to guide further endoscopic, angiographic, or surgical treatment.

10.4 Treatment

Endoscopy

Endoscopic therapies for LGIB include injection of epinephrine, thermal coagulation, and placement of endoscopic clips.

Epinephrine achieves hemostasis by causing local vasoconstriction and creating a tamponade effect due to the volume of fluid injected. Coagulation hemostasis can be achieved with heater probes, bipolar cautery, or argon plasma beam. Argon plasma coagulation is particularly useful for diffuse or multifocal sources of hemorrhage. Endoscopic clips physically tamponade bleeding to achieve hemostasis. They are most effective with diverticular and hemorrhoidal causes of CRB. Choice of endoscopic therapy is dependent on available resources, patient anatomy, and operator experience. However the ASGE recommends injection or coagulation for diverticular bleeding, clips for recurrent diverticular bleeding, and argon coagulation for bleeding angioectasias [2]. Endoscopic therapies are generally successful with effective hemorrhage control in 80–96 % of patients [14, 20]. Rebleeding rates range from 7 to 46 % depending on the endoscopic technique used, etiology of the CRB, and length of follow-up [1, 4, 6, 13, 14, 21]. Complication rates are generally low, ranging from 0 to 2 %, and include rebleeding, ischemia, perforation, and cardiopulmonary complications [1, 11, 14]. New innovations in endoscopic technology include spray on topical hemostatic agents (Ankaferd BloodStopper, EndoClot Polysaccharide Hemostatic System, and Hemospray) and larger over-the-scope clips (OTSC, Ovesco Endoscopy AG). Data on these newer technologies is limited, but preliminary case series are promising with hemostasis rates of 76–96 % [22].

Angioembolization

Angiographic therapies include embolization with microcoils, polyvinyl alcohol particles, or gelfoam. In the past continuous infusion of vasopressin was also used; however, associated cardiac and ischemic complications in addition to high rates of recurrence have made this a less desirable treatment option. Embolization can be used to control hemorrhage successfully in 63–100 % of patients [1, 7, 14, 23, 24]. Angioembolization

appears to be most effective in cecal lesions and in CRB of diverticular origin. Rebleeding rates following angioembolization range from 8 to 26% [14, 23–27]. Risk factors for recurrent bleeding include post-procedural bleeding, non-diverticular source, multisystem organ failure, presence of comorbidities, transfusion requirements ≥ 6 units, shock, and coagulopathy/anticoagulants. Complications following coil embolization are generally low ranging from 0 to 25% and include ischemia, contrast-induced nephropathy and access site complications (pseudoaneurysm, dissection, or hematoma) [1, 7, 14, 23, 24, 27]. The most concerning of the possible complications is bowel ischemia, which is quite rare, particularly if using super-selective catheterization. Minor ischemic complications occur in 4.5–9% of cases, and major ischemic complications occur in 0–1.4% of cases [1, 24, 27]. When endoscopic treatment is unavailable or fails to identify or control bleeding, angioembolization appears to be similar to surgery in efficacy and may be the preferred option in poor surgical candidates.

There are few trials directly comparing endoscopic and angiographic control of CRB; however, there is some suggestion that colonoscopy results in a better diagnostic yield and percentage of patients amenable to intervention with reduced complications and length of stay [13, 14]. As such the ASGE recommends endoscopic treatment as the first-line treatment for CRB and recommends surgical and radiologic consultation only for patients who fail endoscopic treatment or cannot be stabilized enough to undergo endoscopy [2].

Surgery

Surgery for control of CRB is decreasing in frequency as endoscopic and angiographic intervention improves in efficacy and availability dropping to 4–20% in recent studies [8, 20, 23, 27–29]. Surgery is primarily utilized after bleeding is localized

with CTA, endoscopy, or angiography but cannot be controlled with less invasive techniques and in cases of bleeding malignancies. Indications for surgery include transfusion of ≥ 6 units of blood, inability to achieve hemodynamic stability with appropriate resuscitation, and failure to control bleeding by other means [1, 7]. The rate of rebleeding is significantly lower with preoperative radiographic or endoscopic localization 14% vs. 42% following blind resection. Preoperative localization also increases hemostasis and decreases postoperative morbidity and mortality [1, 28, 30]. Blind segmental resection and intraoperative endoscopy for anything but previously identified small bowel lesions are no longer recommended due to low diagnostic/therapeutic yield and high rates of morbidity and mortality [1, 7]. Total abdominal colectomy is a reasonable salvage surgical option in patients where repeated endoscopic and radiographic exams have failed to identify a source, bleeding is ongoing, and diverticular source is highly suspected. Morbidity associated with surgical treatment of CRB is high, occurring in up to 23% of patients [31]. Overall surgical mortality ranges from 2.9 to 20% [1, 28, 29] but can be significantly increased following blind resection reaching 25–57% [30, 32].

Key Point

The majority of lower GIB stops spontaneously. In patients who require therapeutic intervention, endoscopy is the first-line treatment, because it is effective and associated with a low rate of complications.

Key Point

In patients who fail endoscopic therapy or are not candidates for colonoscopy, angioembolization is the preferred treatment. Surgery is reserved for recurrent active bleeding, unresponsive hemodynamic instability, and malignant causes of CRB.

10.5 Outcomes

Morbidity and mortality associated with CRB are particularly high among elderly patients who may have additional comorbidities, have poor physiologic reserve, and are frequently on anticoagulants, antiplatelet agents, and NSAIDs. They are also higher among patients with in-hospital bleeding compared to those coming from home [8].

Overall morbidity ranges from 6.4 to 21 %, with minor complications occurring in 26 % and major complications in 17 % of patients [14, 27, 33]. These include cardiopulmonary complications, rebleeding, bowel ischemia, access site complications, contrast-induced nephropathy, and allergy. Risk factors for morbidity include the presence of comorbidities, malignancy, age, anticoagulant use, and need for surgery [8, 33].

CRB has historically been associated with a lower mortality than upper GIB, but results in longer hospitalizations and more resource utilization [1, 34]. Mortality ranges from 3.6 to 25 % with the majority of studies reporting rates between 3 and 5 % [7, 8, 28, 31, 33–35]. Mortality is highest among patients with active bleeding, hemodynamic instability, and those requiring surgical treatment. Other risk factors for mortality include in-hospital bleeding, advanced age, intestinal ischemia, anticoagulant use, multiple transfusions, malignancy, severe comorbidities, and male gender [8, 29, 33].

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Chapter 11

Acute Pancreatitis

Ari Leppäniemi

11.1 Etiology and Pathogenesis

Alcohol and gallstone disease are the two commonest etiological factors for acute pancreatitis comprising about 70–80% of the patients. The other causes are much rarer and include hypercalcemia, hypertriglyceridemia, trauma, a variety of drugs, infections, postoperative conditions (e.g., cardiac surgery), endoscopic retrograde cholangiopancreatography (ERCP), developmental anomalies (such as pancreas divisum), tumors, and hereditary and autoimmune diseases. In about 10% of the cases, the etiology remains unknown.

The main pathogenic determinant in acute pancreatitis is the excessive activation of a systemic inflammatory response cascade leading to multiple organ dysfunction. At first a triggering factor is needed to initiate the pancreatic acinar cell injury. After several intracellular events, pancreatic proenzymes (zymogens) become activated intracellularly, resulting in acinar cell injury. This is fol-

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lowed by local inflammation of the pancreas resulting in activation of several inflammatory cells and release of inflammatory mediators. If this inflammation cannot be controlled locally, excessive uncontrolled activation of inflammatory cells and mediators leads to a systemic inflammatory response syndrome (SIRS) that is similar to other SIRS-associated conditions, such as sepsis or severe trauma, for example. Leaking microvessels cause a loss of intravascular fluid and in conjunction with vasodilatation lead to hypotension and shock. Accumulation of inflammatory cells in tissues, increased interstitial fluid, and activation of coagulation with microvascular thrombosis further impair oxygen supply of tissues. Clinical manifestation of all this is a multiple organ dysfunction syndrome (MODS), characterized by dysfunction or failure of the respiratory, cardiovascular, renal, hepatic, hematological, gastrointestinal, and central nervous system functions. MODS usually develops early during the course of the disease, and over half of the patients with severe acute pancreatitis have signs of organ dysfunction on hospital admission.

Recently, increased intra-abdominal pressure (IAP) and the development of abdominal compartment syndrome (ACS) have been recognized as significant contributors to the development of early MODS in severe acute pancreatitis. If the patient survives the initial inflammatory insult, a second critical phase usually follows 2–4 weeks later with the appearance of septic, local, and other complications. Infection of the pancreatic and peripancreatic necrosis occurs in about 20–40% of patients with severe acute pancreatitis and is associated with worsening MODS.

According to the updated Atlanta classification 2012, the peripancreatic collections associated with necrosis are acute necrotic collection (ANC) and walled-off necrosis (WON). In the early phase, poorly demarcated “acute peripancreatic fluid collections” are commonly seen on CT scan. They are homogenous, are confined to normal fascial planes, can be multiple, usually remain sterile, and resolve spontaneously without intervention. A “pancreatic pseudocyst” refers to a well-defined fluid

collection containing no solid material. The development of pancreatic pseudocyst is extremely rare in acute pancreatitis and is often confused with ANC. However, it may form many weeks after operative necrosectomy due to localized leakage of a disconnected duct in the necrosectomy cavity.

ANC is a collection seen during the first 4 weeks and containing variable amount of fluid and necrotic tissue involving the pancreatic parenchyma and/or peripancreatic tissues (Fig. 11.1). WON is a mature, encapsulated collection of pancreatic and/or peripancreatic necrosis with a well-defined, enhancing inflammatory wall (Fig. 11.2). The maturation takes usually 4 weeks or more after the onset of acute pancreatitis.

11.2 Diagnosis and Estimation of Severity

Previous medical history can consist of previous episodes of acute pancreatitis; previously known gallstone disease or symptoms typical for biliary colic; chronic pancreatitis; metabolic disorders, such as hyperparathyroidism or hyperlipemia; history of a recent abdominal trauma; surgical or endoscopic procedures; new drugs; infections; and family history of acute pancreatitis. Sudden pain in the epigastrium, often radiating into the back and feeling like a belt around the upper abdomen, is the most common symptom and is usually constant rather than colicky. Nausea and vomiting are frequent. Fever is common in patients with accompanying cholangitis.

In severe form with unstable vital signs, securing airways and adequate ventilation and starting fluid resuscitation in hypovolemic shock should precede any diagnostic work-up. In addition to the assessment of hemodynamic, pulmonary, and renal functions, abdominal examination is crucial and should consist of inspection noting abdominal distension (caused by ileus, ascites, visceral edema) and possible discolorations around the



Fig. 11.1 Acute necrotic collection (ANC)



Fig. 11.2 Walled-off necrosis (WON)

umbilicus (Cullen's sign) or in the flanks (Grey Turner's sign). Palpation shows epigastric or generalized tenderness, percussion can reveal significant amount of ascites, and auscultation detect the absence of bowel sounds if the patient has paralytic ileus. Furthermore, general findings indicative of alcohol abuse, hyperlipemia, and other general disorders can help in determining the etiology.

Laboratory examinations usually show elevated plasma amylase (or lipase) levels, but the amylase levels may have returned to normal, if several days have passed from the onset of symptoms. C-reactive protein level (CRP) is a useful clinical marker of the severity, but it lags 24–48 h behind and can be completely normal in the initial phase of even a severe form of the disease. Blood count, liver function tests, electrolyte and glucose levels, as well as creatinine should be taken routinely, and in severe cases, arterial blood gas analysis and serum lactate measurements show the extent of cellular hypoperfusion. Triglyceride levels should be measured if known or suspected to be the cause.

The most reliable diagnostic method for acute pancreatitis is the CT scan. Except for differential diagnosis (free intra-abdominal air) when CT is not available or is too time consuming, plain abdominal radiographs are not needed and chest radiographs may be obtained to evaluate pulmonary status. In performing the CT scan, oral contrast can be administered (but is not necessary), whereas intravenous contrast material should be used with caution and only after confirming adequate circulating volume and urine output. CT scan without intravenous contrast is sensitive in detecting acute pancreatitis. Later on in patients with necrotizing pancreatitis, the contrast enhancement and patency of the pancreas itself can be evaluated using intravenous contrast CT scan.

Ultrasound is useful in identifying gallstones in the gallbladder and a dilated common bile duct when duct stones or cholangitis is suspected. In some cases, magnetic resonance cholangiopancreatogram (MRCP) can be used for suspected

bile duct stones and is sometimes helpful to confirm that a common bile duct stone has passed through to the duodenum, thus saving an unnecessary ERCP examination. However, ERCP is needed when ultrasonography reveals dilated common bile duct and there is a suspicion of a persistent stone or the patient has signs of cholangitis. Endoscopic sphincterotomy with clearance of the common duct from stones and/or drainage of pus (in cholangitis) is justified, even if it does not change the natural course of the pancreatitis itself.

The amount or progression of amylase levels do not correlate with severity; CRP >150 mg/L is better but manifests only 24–48 h later, and other markers such as procalcitonin are not in everyday clinical use. Clinical scoring systems such as those described by the late Ranson or Imrie are inaccurate and not used anymore. APACHE II score >8 demonstrates fairly accurately the acuity of the disease indicating significant physiological derangement, but probably the best way to monitor and quantify the organ dysfunction is by using the Sequential Organ Failure Assessment (SOFA) score and especially its cardiovascular, pulmonary, and renal components to determine if the patient should go to the ICU directly from the emergency room.

Although there is no reliable single marker to differentiate between edematous and necrotizing acute pancreatitis, the combination of clinical evaluation, CRP, CT scan, and the presence or absence of organ dysfunctions are usually sufficient. If severe form of acute pancreatitis is suspected or anticipated and especially if the patient already has signs of organ dysfunction, early admission to an intensive care or high dependency unit is mandatory in order to be able to monitor and support vital organ functions.

The most common differential diagnoses include diseases presenting with acute epigastric or mid-abdominal pain and include perforated peptic ulcer, biliary colic, acute cholecystitis, ruptured abdominal aortic aneurysm, reflux esophagitis, acute mesenteric ischemia, intestinal obstruction, acute hepatitis, inferior myocardial infarction, and basal pneumonia. It is particularly important to differentiate between secondary peritonitis

caused by hollow organ perforation usually requiring urgent surgery and acute pancreatitis where early surgery is usually harmful. Therefore, when in doubt, a CT scan is important provided that it does not delay the initiation of treatment in critically ill patients, whether having pancreatitis or peritonitis.

11.3 Treatment

11.3.1 Mild Acute Pancreatitis

The treatment of mild or edematous pancreatitis is mainly supportive consisting of fluid resuscitation and therapy, pain medication, and sometimes the management of accompanying delirium tremens in patients with alcohol-induced pancreatitis. Urine output should be monitored, usually with the placement of a Foley catheter (goal 0.5–1.0 ml/kg/h), and adequate volume restoration secured. Nasogastric tube is not routinely indicated, but is helpful in patients with dilated stomach or paralytic ileus. Oral feeding should be started as soon as it is tolerated. Any signs of severe pancreatitis should be noted early (clinical condition, CRP, organ dysfunctions) and evaluated for the need to admit the patient to the ICU.

In patients with mild biliary pancreatitis, laparoscopic cholecystectomy can be performed before discharging the patient.

11.3.2 Severe Acute Pancreatitis

11.3.2.1 Fluid Resuscitation

Aggressive fluid therapy during the early phase of acute pancreatitis used to be one of the cornerstones in the early treatment phase of severe pancreatitis, but gradually the negative effects of

excessive fluid resuscitation have been recognized, and a more measured and moderate policy of fluid resuscitation has become the standard. No doubt, the rationale behind aggressive fluid resuscitation was sound, that is, to correct hypovolemia caused by third-space fluid loss. However, excess volume loading may increase intra-abdominal pressure (IAP) and cause intra-abdominal hypertension (IAH) or even abdominal compartment syndrome (ACS). Unfortunately, there are no good resuscitation end points for specific severe acute pancreatitis, and one has to rely on the more common end points similar to other diseases causing severe physiological derangement, such as severe sepsis or septic shock. The principles of early goal-directed resuscitation including monitoring of central venous pressure (CVP), mean arterial pressure (MAP), and either central venous oxygen saturation or mixed venous oxygen saturation can be used. In addition, IAP should be monitored and the abdominal perfusion pressure ($APP = MAP - IAP$) calculated. The APP could also serve as a good resuscitation end point, at least in patients with IAH. Maintaining APP above 50–60 mmHg is needed in order to provide sufficient perfusion to the abdominal organs.

Base deficit and blood lactate levels should be monitored, and resuscitation should be targeted to normalize the lactate level. As soon as the set resuscitation end points are reached, the infusion rate should be slowed down in order to avoid fluid overloading.

11.3.2.2 Enteral Nutrition

Fasting does not help, and it does not alleviate the inflammatory response. Enteral feeding is superior to parenteral feeding, and the only contraindication is poor motility of the gastrointestinal tract. Enteral nutrition prevents bacterial overgrowth in the intestine and reduces bacterial translocation and reduces the risk of systemic infections, organ dysfunction, and mortality.

Besides, all critically ill patients are at risk of malnutrition, and therefore enteral nutrition of patients with severe acute pancreatitis should be started as soon as possible.

The route of enteral feeding can be either gastric or postpyloric. Most patients tolerate gastric feeding via a nasogastric tube, but the residuals should be monitored every 6 h. If gastric feeding is not possible because of impaired gastric emptying and not relieved with the use of erythromycin or other prokinetics, a nasojejunal feeding tube should be inserted either with the help of endoscopy or using self-advancing tubes.

Tube feeding should be started slowly, 10 ml/h, for example, and increased by 10 ml/h every 6 h providing that gastric residual volume is below 250 ml. This should be continued until the target volume of enteral nutrition is achieved. Volumes should not exceed 60 ml/h to avoid the rare but catastrophic complication of bowel necrosis. If the patient does not tolerate enteral nutrition in sufficient volumes, parenteral nutrition can be combined with enteral nutrition to fulfill the nutritional requirements.

11.3.2.3 Antibiotics

About 25% of the patients with acute pancreatitis suffer from an infectious complication, and they are more common in patients with severe acute pancreatitis. The majority of infections in patients with severe acute pancreatitis are extrapancreatic, such as bacteremia or pneumonia, and half of them develop during the first week after admission. Infection of the pancreatic or peripancreatic necrosis comes usually later and peaks at about week 3–4. The risk factors for infected necrosis include early bacteremia, organ failure, and extent of necrosis.

The diagnosis of infected necrosis is controversial, and the earlier reliance on fine needle aspiration (FNA) of the necrosis, usually performed with ultrasound guidance, has been ques-

tioned, as it has been shown to have a false-negative rate of 20–25 %.

Clinical signs of sepsis are too unspecific for definitive diagnosis, although a new increase in the CRP value without any other good explanation might alert you to look for the infected necrosis. Gas bubbles in the CT scan are reliable signs of infection, but they are present only in less than 10% of patients with infected necrosis.

There are many randomized controlled trials showing that prophylactic antibiotics do not benefit patients with acute pancreatitis. However, when looking at the studies more carefully, there has been a nonsignificant trend for lower mortality and reduced number of infections, especially extrapancreatic infections in patients treated with prophylactic antibiotics. The randomized trials have been conducted with small sample sizes, and some studies included a substantial number of patients with mild pancreatitis with minimal risk of mortality and low risk of infectious complications. Acknowledging the limitations of the trials and that patients with organ failure are susceptible to infections, some surgeons use prophylactic antibiotics in patients with severe pancreatitis at least when they have organ dysfunctions and are admitted to the ICU. Clinical judgment taking into account the presence of SIRS, the presence of IAH, hyperglycemia, low plasma calcium, high creatinine, or other signs of organ dysfunction can be used to guide the decision-making.

If prophylactic antibiotics are not given, empiric use of antibiotics is appropriate in patients who develop organ dysfunctions, because of the high risk of bacteremia during the first week. After the end of the second week, empiric antibiotics may be needed for treatment of infected pancreatic necrosis if sepsis continues or the patient does not recover. The antibiotics should cover gram-negative rods and gram-positive cocci. The role of empiric antifungals is not clear. FNA for

microbiological samples should be taken if infected necrosis is suspected, although negative samples do not rule out infection. Positive samples help in the selection of antimicrobials and initiation of possible antifungal therapy. Whatever the reason for starting antibiotics, they should be discontinued when the patient recovers from organ dysfunctions, and there is no evidence of infection.

The principles of early management of acute pancreatitis are summarized in Table 11.1.

11.3.2.4 Surgical Management

In addition to surgical or endoscopic interventions required for gallstone-associated pancreatitis, there are a few reasons to operate on patients with severe acute pancreatitis, and the majority of patients never develop these complications.

Table 11.1 Early management principles in severe acute pancreatitis

Early and timely admission to an intensive care or high dependency unit

Fluid resuscitation goals:

MAP > 65 mmHg

SvO₂ > 65 % (requires pulmonary artery catheter)

Normal lactate level

Urine output > 0.5–1.0 ml/kg/h

IAP measurement every 4–6 h of IAP

Vasoactive support (norepinephrine and dobutamine if cardiovascular failure)

Goal: APP (MAP-IAP) > 60 mmHg

Analgesia, sedation, lung-protective ventilation

Normoglycemia

Thrombosis prophylaxis

Early enteral feeding

Prophylactic antibiotics

Early biliary decompression, if obstruction (especially, if cholangitis)

11.3.3 Abdominal Compartment Syndrome

The combination of excessive fluid resuscitation and capillary leakage lead to tissue edema of the abdominal and retroperitoneal organs, and ascites formation. Intestinal paralysis usually adds to the increase of the intra-abdominal volume. The extra need for space can partly be compensated by the increase in the abdominal domain, but at some stage, the reserve capacity is used, and the intra-abdominal pressure (IAP) starts to increase leading to intra-abdominal hypertension (IAH). The incidence of IAH in patients with acute pancreatitis admitted to ICU is about 60%, and the incidence of the clinical syndrome of abdominal compartment syndrome (ACS) comprising of IAP >20 mmHg and a new-onset organ dysfunction can be as high as 27% as reported in the largest published series. All patients treated for severe acute pancreatitis should undergo repeated and routine measurement of the IAP, usually via a urinary bladder catheter. Already IAP levels of 12 mmHg impair renal function. In patients with IAH, the abdominal perfusion pressure (APP=MAP-IAP) should be calculated because patients in shock can easily have inappropriately low APP (<50–60 mmHg) even with moderate IAH. Poor perfusion increases bowel mucosal injury which is associated with infectious complications and organ failure. In addition, IAH may play significant role in ischemic bowel complications, especially colonic necrosis or even small bowel ischemia.

Although adequate fluid resuscitation is important in the early phase of severe acute pancreatitis, excessive volumes should be avoided. Prevention and management of gastric dilatation with a nasogastric tube and percutaneous drainage of excessive pancreatic ascites are useful adjuncts to nonoperative management. Short-term use of neuromuscular blockers may also be considered. Removal of fluid by extracorporeal techniques is effective in rapidly removing excess fluid.

When nonsurgical interventions fail to change the progressive deterioration of organ dysfunctions in the presence of fulminate ACS, surgical decompression should be considered. The most commonly used method is midline laparostomy, where all abdominal wall layers are divided through a vertical midline incision extending from the xiphoid to the pubis with a few centimeters of fascia left intact at both ends to facilitate subsequent closure or late reconstruction. An alternative method utilizes a bilateral subcostal incision few centimeters below the costal margins. A less invasive technique is the subcutaneous linea alba fasciotomy (SLAF) where the fascia alone is divided through three short horizontal skin incisions leaving the peritoneum intact. The aim of surgical decompression, whatever method is used, is to achieve adequate APP of >60 mmHg. Opening the abdomen to reduce IAP is associated with severe morbidity most commonly associated with the management and complications of the open abdomen, such as enteric fistulas and giant ventral hernias. The best temporary abdominal closure technique seems to be the vacuum-assisted wound closure combined with mesh-mediated fascial traction. It has the highest fascial closure rate (80–90%) and lowest enteric fistula rate when compared with the other currently available techniques.

11.3.4 Infected Pancreatic Necrosis

According to the updated Atlanta classification 2012, the peripancreatic collections associated with necrosis are acute necrotic collection (ANC) and walled-off necrosis (WON). In the early phase, poorly demarcated “acute peripancreatic fluid collections” are commonly seen on CT scan. They are homogenous, are confined to normal fascial planes, can be multiple, usually remain sterile, and resolve spontaneously without intervention.

A “pancreatic pseudocyst” refers to a well-defined fluid collection containing no solid material. The development of

pancreatic pseudocyst is extremely rare in acute pancreatitis and is often confused with ANC. However, it may form many weeks after operative necrosectomy due to localized leakage of a disconnected duct in the necrosectomy cavity.

ANC is a collection seen during the first 4 weeks and containing variable amount of fluid and necrotic tissue involving the pancreatic parenchyma and/or peripancreatic tissues. WON is a mature, encapsulated collection of pancreatic and/or peripancreatic necrosis with a well-defined, enhancing inflammatory wall. The maturation takes usually 4 weeks or more after the onset of acute pancreatitis.

Infected necrosis is a significant source of sepsis, and removal of devitalized tissue is believed to be necessary for control of sepsis. However, infection usually continues after necrosectomy, especially if necrotic tissue is left in place. Before demarcation of necrosis develops, usually after 4 weeks from disease onset, it is impossible to remove all necrotic tissue without causing bleeding, and too early surgical debridement is associated with high risk of hemorrhage leading to increased organ dysfunction and death. Because high mortality is associated with early surgery and multiple organ dysfunction, surgery for infected necrosis should be postponed as late as possible, preferable later than 4 weeks from the onset of the disease.

Percutaneous drainage of the liquid component of the infected acute necrotic collection may serve as a bridge to surgery and sometimes suffices alone. Sterile collections do not need drainage, because placement of a drain into a sterile necrotic collection can result in secondary infection, especially after prolonged drainage. There are no randomized studies comparing operative treatment and catheter drainage in patients with worsening multiple organ failure within the first few weeks from disease onset. The only randomized trial comparing open necrosectomy and minimally invasive step-up approach included only 28 (32%) patients with multiple organ failure, and the median time of interventions was 30 days from disease onset. In

this study, the mortality rate was the same between the groups; no data of subgroup analysis of patients with multiple organ failure was shown.

Although the use of mini-invasive techniques are increasingly used for infected pancreatic necrosis, the lowest published mortality rate in patients operated on for infected necrosis is with open debridement and closed packing with 15 % mortality. In patients without preoperative organ failure, minimally invasive necrosectomy is associated with fewer new-onset organ failure than open surgery. However, a considerable number of patients are not suitable for mini-invasive surgery because of the localization of the necrotic collection.

According to the IAP/APA evidence-based guidelines for the management of acute pancreatitis, the indications for intervention (surgical, radiological, or endoscopic) in necrotizing pancreatitis are listed in Table 11.2. The timing of intervention is usually postponed until at least 4 weeks after the initial presentation to allow the WON to be formed, and for some of the other indications, it is more than 8 weeks.

The preferred technique for open necrosectomy used at our institution is as follows: transverse bilateral subcostal incision (often extending more to the left), dividing the gastrocolic ligament (we prefer not to go through the transverse mesocolon), opening the right tissue planes with blunt dissection, and utilizing harmonic scalpel or old-fashioned ligatures for good exposure. Usually the necrosis is mostly found around the pancreas, while the pancreas itself is firm and protrudes like a transverse ridge. In these cases it should be left alone. If on the other hand (and as might be suggested in a preoperative CT) the necrotizing process has destroyed the middle part of the pancreas, the distal part can usually be removed easily by squeezing it out distally with gentle finger dissection being careful not to damage the splenic vessels. The spleen is left intact if possible. Once the dead distal pancreas has been removed (sometimes only a small proximal remnant is left), one can try to find the

Table 11.2 Indications for surgical radiological or endoscopic indications in severe acute pancreatitis

Clinically suspected or documented infected necrosis with clinical deterioration or ongoing organ failure for several weeks
Ongoing gastric outlet, intestinal, or biliary obstruction due to mass effect of WON
Patient not getting better with WON but no infection (after 8 weeks)
Disconnected duct syndrome (full transection of the pancreatic duct) with persisting symptomatic collection with necrosis without signs of infection (>8 weeks)

divided pancreatic duct and ligate it. Usually it cannot be seen and a pancreatic fistula may occur, but that can be managed with an endoscopically placed stent later on. After removing the necrotic tissue, the area is packed for a few minutes and the hemostasis is secured with amply placed sutures. Minor oozing usually stops by itself. Draining the peripancreatic area with a couple of well-placed (one coming behind the left hemicolon into the pancreatic area if the necrosis is mainly on the left side) completes the procedure. Unless there is a risk of increased IAP, the wound is usually closed.

Endoscopic variations for the management of peripancreatic necrotic collections have been introduced and include endoscopic transgastric or retroperitoneal drainage or necrosectomy. The value of these techniques is still under assessment, and only small randomized series with well-selected patients have been published.

If the disease process has eroded the pancreas leaving a considerable portion of the distal pancreas intact (disconnected duct syndrome) and the patients develop symptomatic collections, the distal pancreatic remnant can be resected or connected to a Roux-en-Y loop with pancreaticojejunostomy. Although saving viable pancreatic tissue might be beneficial, the long-term benefits of internal drainage over resection have not been established.

11.3.5 Surgery for Extrapancreatic Complications

Bleeding is a rare complication in severe acute pancreatitis, but when occurring requires prompt management either by surgical intervention or angiographic embolization. Sometimes the bleeding has to be packed in a reoperation leaving the abdomen open and doing a reoperation two days later removing the packs.

Necrosis of a part of the colon in acute pancreatitis is associated with high mortality and is difficult to diagnose until perforation occurs. Gas bubbles in the colonic wall can be a useful hint. Colon necrosis is probably caused by retroperitoneal spread of the necrotizing process to colon with fat necrosis and pericolicitis. Usually, the inner layers of colon remain viable longer. The most common places of colon necrosis are in the cecum where it is aggravated by dilatation or in the transverse colon where it can be related to the thrombosis of the middle colic artery branches associated with the peripancreatic necrosis. There should be a low threshold for colonic resection due to unreliable detection of ischemia or imminent perforation just by seeing the outside of the colon during surgical exploration. Obviously, in patients with clear perforation, removal of the affected segment is mandatory. Primary colonic anastomosis under these circumstances is risky, and a temporary colostomy is a safer option.

11.3.6 Biliary Surgery

The 2002 evidence-based guidelines of the International Association of Pancreatology recommended early cholecystectomy in mild gallstone-associated acute pancreatitis and delayed cholecystectomy in severe pancreatitis. Cholecystectomy should be delayed in patients with moderate to severe pancreatitis and

demonstrated peripancreatic fluid collections or pseudocysts until the pseudocysts either resolve or beyond 6 weeks, at which time the pseudocyst drainage can safely be combined with cholecystectomy. Therefore, in patients with severe gallstone-induced acute pancreatitis, cholecystectomy should be delayed until the inflammatory response resolves and clinical recovery occurs.

In patients with mild gallstone pancreatitis, laparoscopic cholecystectomy performed within 48 h of admission, regardless of the resolution of abdominal pain or laboratory abnormalities, is safe and results in a shorter hospital length of stay with no apparent impact on the technical difficulty of the procedure or perioperative complication rate. It has become more common for patients with mild biliary pancreatitis to undergo laparoscopic cholecystectomy at the same hospitalization period once the clinical signs of pancreatitis have resolved.

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Chapter 12

Acute Cholecystitis and Cholangitis

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12.1 Introduction

Although stones in the biliary tree (and especially in the gallbladder) are an extremely common (and many times asymptomatic) disorder, they may even be the cause of insidious infections. Acute cholecystitis (AC) is a bacterial infection (most likely preceded by an inflammation of the gallbladder wall) produced by an obstruction of the cystic duct by gallstones. The obstruction results in gallbladder distension, wall edema, inflammation, ischemia, and ultimately bacterial infection, causing necrosis, gangrene, and eventually perforation of the gallbladder wall, with the development of a local abscess or generalized peritonitis. The obstruction is usually caused by gallstones (>90%), thereby identifying the acute calculous cholecystitis (ACC), but AC may

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infrequently be acalculous (acute acalculous cholecystitis, AAC) [1]. Instead acute cholangitis is a bacterial infection caused by an obstruction of the biliary tree most commonly from gallstones, independent of the gallbladder and cystic duct (termed choledocholithiasis, CL), resulting in elevated intraluminal pressure and bile infection. CL in many cases can be even associated to AC.

12.2 Acute Calculous Cholecystitis

Acute calculous cholecystitis (ACC) is an inflammatory condition of the gallbladder resulting from a spectrum of pathophysiologic processes. While the diagnosis of ACC is frequently straightforward, in some setting, it can be quite complex. ACC is most commonly the result of acute obstruction of the cystic duct by biliary stones or cholelithiasis, termed calculous cholecystitis. ACC may progress to gangrenous cholecystitis with an elevated risk of free perforation and perioperative complications. The diagnosis of most cases of typical ACC can usually be achieved with a high degree of accuracy with the combination of clinical presentation and diagnostic imaging. The signs and symptoms that suggest a diagnosis of ACC are due to one of the two pathophysiologic processes: (1) contraction of the gallbladder against obstruction to biliary outflow causing biliary colic and (2) inflammation of the gallbladder that occurs secondary to the obstruction. To address the variable physical exam findings, lack of a specific laboratory test, and the emergence of imaging technology, objective diagnostic criteria for the diagnosis of AC were established by an international consensus conference in 2007 and subsequently validated (termed the Tokyo Guidelines), see Table 12.1 [2].

12.2.1 Presentation of ACC

Clinical presentation of patients presenting with AC most commonly includes right upper quadrant and/or epigastric pain, occurring in 72–93% of cases [3–5]. Often this pain is intermittent or may be described as coming in waves. This intermittent, crampy RUQ pain can be also referred to as biliary colic. Nausea and vomiting are also very common, occurring in 62–83% of cases [3, 6, 7]. Symptoms can frequently occur in the postprandial period, particularly after meals with a high fat content. Fevers are less common with only 10–30% of patients manifesting temperatures over 38 °C [6, 8, 9]. The physical exam finding most connected to the diagnosis of AC is the

Table 12.1 Diagnostic criteria for acute cholecystitis, according to Tokyo Guidelines

<i>Local symptoms and signs of inflammation</i>
Murphy's sign
Pain or tenderness in the right upper quadrant
Mass in the right upper quadrant
<i>Systemic signs of inflammation</i>
Fever
Leukocytosis
Elevated C-reactive protein level
<i>Imaging findings</i>
A confirmatory finding of acute cholecystitis on imaging (US or CT)
<i>Suspected diagnosis</i>
The presence of one local sign of inflammation and one systemic sign of inflammation
<i>Definite diagnosis</i>
The presence of one local sign or symptom, one systemic sign, and a confirmatory finding on an imaging test
Must rule out acute hepatitis, chronic cholecystitis, and other acute abdominal diseases

Murphy's sign. However the sensitivity of Murphy's sign remains fairly low, reported as low as 20.5 % to as high as 65 % [10, 11]. Thus, its use as a diagnostic test can result in a high rate of false-negative findings.

12.2.2 Laboratory Test

Although there is not a biomarker that specifically correlates with AC diagnosis, markers of generalized inflammation in combination with other clinical and imaging findings can increase the reliability of this diagnosis. Leukocytosis and elevated C-reactive protein (CRP) are most commonly employed. Mild leukocytosis (over 10,000 cells/ μ L) is suggestive of systemic inflammation. Higher WBCs are more likely to be associated with more severe disease, like gangrenous cholecystitis. However, studies have not clearly delineated where the transition from noncomplicated AC to gangrenous cholecystitis occurs. In different studies, WBC counts over 13,000, 15,000, and 17,000 cell/ μ L have all been associated with increased risks of gangrenous cholecystitis [12–14]. CRP is also present in conditions of systemic inflammation. Values over 3 mg/dL are consistent with inflammatory conditions. When elevated CRP is combined with positive ultrasound findings for AC, sensitivity is 97 %, with 76 % specificity [15]. Similar to leukocytosis, higher elevations of CRP correlate with greater likelihood of the presence of gangrenous cholecystitis [15, 16]. Transmural inflammation of the gallbladder during AC may involve adjacent liver parenchyma, producing a mild elevation in gamma-glutamyl transpeptidase (GGT), aspartate aminotransferase (AST), alanine aminotransferase (ALT), and bilirubin, but such abnormalities do not significantly aid in establishing the diagnosis.

12.2.3 Imaging

12.2.3.1 Ultrasound

Ultrasound (US) is probably the most frequently used diagnostic imaging modality for ACC. It should be considered the first imaging option for all suspected cases of ACC. US can visualize gallstones, which can be difficult to identify using CT or HIDA scan, is quick and noninvasive, and does not expose the patient to ionizing radiation. There are a few clear limitations for US; it is well known to be operator dependent, and gallbladder visualization can be limited by patient body habitus and by bowel gas between the US probe and the gallbladder. While ACC on US can have a variable appearance, there are a few findings that are considered indicative of ACC. These include the concurrent presence of thickened gallbladder wall (≥ 5 mm), pericholecystic fluid, and a sonographic Murphy's sign. Other findings which may also indicate ACC include gallbladder distention/enlargement, gallstones, debris echo or sludge, and gas within the gallbladder wall. US has demonstrated good sensitivity in multiple studies. A meta-analysis by Kiewiet showed sensitivities ranging from 50 to 100 % with an overall sensitivity of 81 %. Specificities were shown to be a bit better with an overall specificity of 83 %, despite a range of 30–100 % [17]. US by emergency department (ED) physicians has also proven to be reliable in the detection on AC. ED physician-performed US was shown in a study of 116 patients to have a sensitivity of 92 %, a specificity of 78 %, and an accuracy of 86 % when compared with radiologist-performed US [18]. More recently, 96 % sensitivity and 79 % specificity were noted on ED physician-performed US when compared to surgical pathology. Additionally, this study noted an 85.5 % rate of agreement when compared with blinded radiologist reading [19].

US is most effective when utilized, not in isolation but in combination with other clinical and laboratory findings suggestive of inflammation. For patients with suspected AC, US plus elevated CRP showed a sensitivity of 97% [15] for ACC. The Tokyo Guidelines themselves are based on the idea of combining imaging findings of ACC with clinical findings of inflammation.

12.2.3.2 Computed Tomography (CT)

CT scanning is a common imaging modality in patients with abdominal pain. It can differentiate other causes of RUQ pain. CT scanning is available in almost every hospital and has significantly decreased operator dependence compared to US. Findings of AC on CT scan are similar to those seen on US. Positive findings of the disease include gallbladder wall thickening >3 mm, pericholecystic fat stranding, and gallbladder distention [20]. Pericholecystic fluid, subserosal gallbladder edema, and high-attenuation gallbladder can also be visualized but less commonly [20]. Gallstones may also be visualized depending on the composition and size of the gallstones, but the presence of gallstones may often present in the absence of AC. CT may not be an effective screening modality for AC. There is a paucity of data regarding the sensitivity of CT for ACC diagnosis. In a comparative study with US in 117 patients, CT was shown to have 39% sensitivity and 93% specificity and was significantly worse than US, which had a sensitivity and specificity of 83% and 95%, respectively [21]. Although negative predictive value was good for CT (89%), it was still lower than US (97%). The authors concluded that US is a better initial imaging study and that CT should be reserved for patients with a wider differential diagnosis and/or nonstandard symptomatology.

12.2.3.3 Magnetic Resonance Imaging (MRI)

Previously, MRI was not a popular imaging modality for suspected ACC. MRI was a long study that was expensive for patients and not readily available after hours at most institutions. Additionally, many patients can develop discomfort or outright claustrophobia in the MRI scanner. Due to the danger of the magnet of the MRI machine, critically ill patients or those needing frequent access are not candidates for MRI scanning. MRI has become the imaging modality of choice for hepatobiliary, pancreatic, and pelvic pathology. Scanning protocols have been developed that can now complete an abdominal study in 15–30 min [22]. For the ACC diagnosis, sensitivity (85%) and specificity (81%) fall in between CT and US [17]. As with CT imaging, MRI findings of gallbladder wall thickening, pericholecystic fat stranding, and gallbladder distention are characteristic of AC. Currently, MRI is mostly used for the detection of AC for those with ambiguity or a contraindication to one of the other modalities or those where additional information is required on hepatobiliary pathology.

12.2.3.4 Hepato-iminodiacetic Acid (HIDA) Scintigraphy

Hepato-iminodiacetic acid (HIDA) imaging is an attractive option for the diagnosis of AC as it is highly sensitive with good specificity. The modality is not operator dependent, and it can often differentiate between acute and chronic cholecystitis, a feature that ultrasonography can often fail to do. Normal findings relate to the rapid filling of the gallbladder with radio-tracer and passage into the duodenum which should occur within 30 min. Failure of the gallbladder to fill within 60 min is abnormal but not diagnostic of AC. The absence of any filling

after 3 or 4 h of delayed images qualifies as a diagnostic study (Fig. 3.3). Also considered a positive study is no filling after 90 min when morphine was administered at 60 min. These delayed images confirm no delayed filling of the gallbladder. This indicates cystic duct obstruction and is highly sensitive for AC. Chronic cholecystitis can also cause cystic duct obstruction but much less commonly. However, HIDA imaging also possesses some disadvantages. This study generally requires a period of no oral intake for 3–4 h before the study. Then, the study itself can take up to 3–4 h to complete depending on how rapidly the radiotracer transits into the gallbladder. Sensitivity and specificity of scintigraphy in acute cholecystitis were both shown to be significantly higher ($p < .001$) than that of ultrasound (94 % vs. 80 % and 89 % vs. 75 %, respectively) [17].

12.2.4 Treatment

The status of patient symptoms remains the most important factor in determining the appropriate management for cholelithiasis. Due to the increased risk for recurrent biliary colic or complicated gallstone disease, cholecystectomy is indicated in patients with symptomatic cholelithiasis. Additionally, cholecystectomy is indicated after an episode of complicated biliary disease (AC or gallstone pancreatitis) because there may be a 30% chance of having a recurrence of complicated disease within 3 months. Antibiotic therapy is an important key component in the management of patients with AC [23, 24]. Antibiotics are always recommended in complicated AC and in delayed management of uncomplicated AC. Although there are no clinical or experimental data to support the use of antibiotics with biliary penetration for these patients, the efficacy of antibiotics in the treatment of biliary infections may depend on effective biliary antibiotic concentrations too. However, in patients with obstructed bile ducts, the biliary penetration of antibiotics may

be poor, and effective biliary concentrations are reached only in a minority of patients. Antibiotics are usually used to treat biliary tract infections, and their biliary penetration ability are shown in Table 12.2. In patients with severe comorbidities or those who present with sepsis or cholangitis, preoperative stabilization is required. A percutaneous cholecystostomy tube may be a useful adjunct when patients are critically ill. In patients who can tolerate the operation, an early laparoscopic cholecystectomy (ELC) is preferable to delayed laparoscopic cholecystectomy (DLC) as long as it is completed within 10 days of onset of symptoms [25, 26]. ELC should not be offered for patients beyond 10 days from the onset of symptoms unless the symptoms suggestive of worsening peritonitis or sepsis warrant an emergency surgical intervention. In people with more than 10 days of symptoms, delaying surgery for 45 days is better than immediate surgery [27]. It should be noted that earlier surgery is associated with shorter hospital stay and fewer complications [28, 29].

Laparoscopic cholecystectomy (LC) is the gold standard for the treatment of AC and should always be attempted at first except in case of absolute anesthesiological contraindications

Table 12.2 Antibiotics biliary penetration ability (indicated as the ratio of bile to serum concentrations)

Good penetration efficiency (ABSCR \geq 1)	Low penetration efficiency (ABSCR $<$ 1)
Piperacillin/tazobactam (4.8)	Ceftriaxone (0.75)
Tigecycline ($>$ 10)	Cefotaxime (0.23)
Amoxicillin/clavulanate (1.1)	Meropenem (0.38)
Ciprofloxacin ($>$ 5)	Ceftazidime (0.18)
Ampicillin/sulbactam (2.4)	Vancomycin (0.41)
Cefepime (2.04)	Amikacin (0.54)
Levofloxacin (1.6)	Gentamicin (0.30)
Penicillin "G" ($>$ 5)	
Imipenem (1.01)	

ABSCR antibiotics bile/serum concentration ratio

and septic shock [30]. LC for AC is safe, feasible, with a low complication rate, and associated with shortened hospital stay [28, 29]. In certain cases, however, it still remains necessary to convert from laparoscopic to open cholecystectomy. Indications include intolerance of pneumoperitoneum, severe inflammation or otherwise limited view, anatomy difficult to be recognized, uncontrollable bleeding, malignancy, or suspected or confirmed biliary injury [31, 32]. Subtotal cholecystectomy is an important tool and achieves morbidity rates comparable to those reported for total cholecystectomy in simple cases. Laparoscopic or open subtotal cholecystectomy is a valid option for advanced inflammation, gangrenous gallbladder, and more in general in “difficult gallbladder” where anatomy is difficult to be recognized and main bile duct injuries are highly probable [31].

Complications of cholecystectomy include bile duct injury, bile leak, bleeding, and retained stones. The associated clinical syndromes vary widely in timing and severity of presentation.

Percutaneous tube cholecystostomy (PC) (followed or not by surgery) is reported in the literature as an alternative for the emergency treatment in septic high-risk patients. The panel of the Tokyo Guidelines states that it is known to be an effective option in critically ill patients, especially in elderly patients and patients with complications. Prompt PC improves survival in high-risk surgical patients [33].

12.2.5 Acute Calculous Cholecystitis and Choledocholithiasis

Choledocholithiasis (CL) is defined as the presence of gallstones in the biliary tree, independent of the gallbladder and cystic duct. The incidence of CL among patients undergoing cholecystectomy is between 3 and 40% [34, 35], with a reported lower incidence during acute cholecystitis ranging from 5 to

15% of the patients. Investigations for CBD stones require time and could delay the surgical intervention for ACC. Due to the relatively low incidence of CBD stones during acute cholecystitis, the great issue is to select patients with a high likelihood to have common bile duct stones who could benefit from further diagnostic tests and eventually the removal of the stones. Elevation of liver biochemical enzymes and/or bilirubin levels is not sufficient to identify patients with CL, and further diagnostic test is needed. At transabdominal US, the visualization of CBD stones is a very strong predictor of CL. Indirect signs of stone presence as increased diameter of CBD are not sufficient to identify patients with CL and need to be combined with other parameters. The American Society of Gastrointestinal Endoscopy combined the various published validated clinical scores and proposed a risk stratification of CBD stones. The score is showed in Table 12.3.

Fundamentally in cases of suspected or documented CL associated to ACC, the goals of treatment are to clear the CBD of stones if present and to remove the gallbladder. The first step in the aforementioned process involves determining the likelihood of CL. Fundamentally, three strategies may be identified to approach the patients with CL. The first option involves inpatient admission of patients at high risk of CL. In these patients, ERCP is performed first then an ELC can be done. The second option involves inpatient admission of patients at moderate risk of CL. These patients should have noninvasive preoperative investigation such as intraoperative cholangiography/laparoscopic ultrasound (followed by intraoperative CBD exploration or ERCP after ELC if CL is present) or endoscopic ultrasound/magnetic resonance cholangiopancreatography (followed by preoperative ERCP if CL is present or ELC if CL is not present). The third option involves inpatient admission of patients at low risk of CL. These patients should have ELC in case an ACC is present. The algorithm is shown in Fig. 12.1.

Table 12.3 A proposed strategy (American Society of Gastrointestinal Endoscopy) to assign risk of choledocholithiasis in patients with symptomatic cholelithiasis based on clinical predictors

Predictors of choledocholithiasis
<i>Very strong</i>
CBD stone on transabdominal US
Clinical ascending cholangitis
Bilirubin > 4 mg/dL
<i>Strong</i>
Dilated CBD on US (>6 mm with gallbladder in situ)
Bilirubin level 1.8–4 mg/dL
<i>Moderate</i>
Abnormal liver biochemical test other than bilirubin
Age older than 55 years
Clinical gallstone pancreatitis
<i>Assigning a likelihood of choledocholithiasis based on clinical predictors</i>

12.3 Acute Acalculous Cholecystitis

Acute acalculous cholecystitis (AAC) is now a well-recognized complication of serious medical and surgical illnesses [36, 37] and is being diagnosed more frequently in critically ill patients [38]. The mortality rate of AAC remains at least 30% because of the potential obscurity of the diagnosis, because of the underlying illnesses of the affected patients, and because of the potential rapid progression of the disease to gangrenous cholecystitis and gallbladder perforation (~10%) [39].

12.3.1 Diagnosis

AAC poses major diagnostic challenges. Most afflicted patients are critically ill and unable to communicate their symptoms. AC is but one of many potential causes in the differential

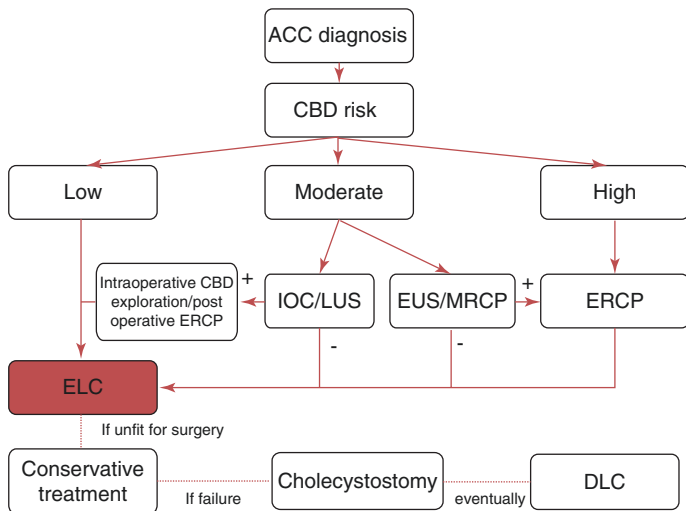


Fig. 12.1 Proposed algorithm. *ACC* acute calculous cholecystitis, *CBD* common bile duct, *DLC* delayed laparoscopic cholecystectomy, *ELC* early laparoscopic cholecystectomy, *ERCP* endoscopic retrograde cholangiopancreatography, *EUS* endoscopic ultrasound, *IOC* intraoperative cholangiography, *LUS* laparoscopic ultrasound, *MRCP* magnetic resonance cholangiopancreatography

diagnosis of systemic inflammatory response syndrome or sepsis in such patients. Rapid and accurate diagnosis is essential, as gallbladder ischemia can progress rapidly to gangrene and perforation. AAC is sufficiently common that the diagnosis should be considered in every critically ill or injured patient with a clinical picture of sepsis or jaundice and no other obvious source. Physical examination and laboratory evaluation are unreliable. The diagnosis of AAC thus often rests on radiologic studies. US of the gallbladder is the most accurate modality to diagnose AAC in the critically ill patient. Although US is accurate for detecting gallstones and measuring biliary duct diameter, neither is particularly relevant to the diagnosis of

AAC. Thickening of the gallbladder wall is the single most reliable criterion [17, 40], with reported specificity of 90% at 3.0 mm and 98.5% at 3.5 mm wall thickness and sensitivity of 100% at 3.0 mm and 80% at 3.5 mm. Accordingly, gallbladder wall thickness ≥ 3.5 mm is generally accepted to be diagnostic of AAC. Other helpful ultrasonographic findings for AAC include pericholecystic fluid or the presence of intramural gas or a sonolucent intramural layer, or “halo,” that represents intramural edema [41, 42]. Distension of the gallbladder of more than 5 cm in transverse diameter has also been reported [41]. CT appears to be as accurate as US in the diagnosis of AAC [43]. Diagnostic criteria for AAC by CT are similar to those described for US [44].

12.3.2 Treatment

In the past, the treatment for AAC was cholecystectomy, due to the ostensible need to inspect the gallbladder and perform a resection if gangrene or perforation was present. Other pathologies that could AC (e.g., perforated ulcer, cholangitis, pancreatitis) could also be identified at this time during open or laparoscopic operation if the diagnosis of AAC was incorrect. However, percutaneous cholecystostomy is now established as a lifesaving, minimally invasive alternative [45, 46]. Percutaneous cholecystostomy controls the AAC in 85–90% of patients [47, 48]. Rapid improvement should be expected when percutaneous cholecystostomy is successful. If rapid improvement does not ensue, suspicion should arise that the tube may be malpositioned and not draining properly, or the diagnosis of AAC may be incorrect. Rarely, in genuine AAC, the patient will fail to improve due to gangrenous cholecystitis, and an open procedure may be required [49]. Antibiotic therapy does not substitute for drainage of AAC but is an important adjunct.

12.4 Acute Cholangitis

Acute cholangitis is an infectious disease of the biliary tract with a wide spectrum of presentations, ranging in severity from a mild form, characterized mainly by fever and jaundice, to a severe form with septic shock. Reported mortality rates vary from 13 to 88% [50]. Cholangitis occurs in 6–9% of patients admitted with gallstone disease [51]. Although bile duct stones are the most frequent cause of acute cholangitis in the Western world, other causes of cholangitis should be kept in mind if stones are absent (Table 12.4). Experimental and clinical models strongly suggest that cholangitis is produced by biliary stasis in association with biliary infection. In fact, patients with partial bile duct obstruction are at increased risk compared to those with complete obstruction. The infecting organism is usually a gram-negative *Bacillus* (most commonly coliforms), but *Staphylococcus* and *Streptococcus* are also reported. The diagnostic criteria for acute cholangitis adopted by the Tokyo Consensus Meeting are reported in Table 12.5.

12.4.1 Presentation

Fever, right upper quadrant abdominal pain, and jaundice constitute the triad described by Charcot in 1877 that is still a generally

Table 12.4 Causes of acute bacterial cholangitis

Gallstones within the biliary tree, sludge
Benign or malignant biliary strictures
Choledochal cyst, choledochoceles, Caroli's disease
Stenosis papilla of Vater
Parasitic infections
Iatrogenic
Post-endoscopic retrograde cholangiopancreatography
Postoperative: "sump" syndrome post-choledochoduodenostomy
Choledochojunostomy

Table 12.5 Diagnostic criteria for acute cholangitis

A. Clinical context and clinical manifestations

1. History of biliary disease
2. Fever and/or chills
3. Jaundice
4. Abdominal pain

B. Laboratory data

5. Evidence of inflammatory response (a)
6. Abnormal liver function tests (b)

C. Imaging findings

7. Biliary dilatation or evidence of an etiology (stricture, stone, stent, etc.)

Suspected diagnosis

Two or more items in A

Definite diagnosis

1. Charcot's triad (2+3+4)
2. Two or more items in A+both items in B and item C

accepted clinical finding of acute cholangitis. About 50–70 % of patients with acute cholangitis develop all three symptoms; milder disease may not exhibit all three features, and, particularly in the elderly, abdominal pain may be absent; this might lead to misdiagnosis or delayed diagnosis. In severe disease, which Reynolds and Dargan [52] in 1959 defined as acute obstructive cholangitis, shock and a decreased level of consciousness are also observed in up to 30 % of patients. Authors have described this pentad as a well-characterized syndrome indicating the need for urgent decompression of the biliary tree.

12.4.2 Laboratory Test

Laboratory investigation for the diagnosis of acute cholangitis is characterized by (1) specific indicators of infection and flogosis such as leukocytosis, increased CRP, and high erythrocyte sedimentation rates; (2) specific indicators of cholestasis such as

increased bilirubin, alkaline phosphatase, and gamma-glutamyl transferase (GGT); and (3) indicators of hepatocellular disease with a pronounced and disproportionate increase in aspartate aminotransferase (AST) or alanine aminotransferase (ALT) that may cause confusion with viral hepatitis.

12.4.3 Imaging

It is usually impossible to identify evidence of bile infection itself by imaging modalities. The role of diagnostic imaging in acute cholangitis is to determine the presence/absence of biliary obstruction, the level of the obstruction, and the cause of the obstruction, such as gallstones and/or biliary strictures. Transabdominal US is the first step in detecting bile duct (as well as associated gallbladder) stones. However, the distal bile duct in particular may be difficult to visualize because of air-containing intestinal loops in front of it. Therefore, the sensitivity of US in detecting bile duct stones is rather low (depending on the experience of investigators and stone size: 27–49%) [53]. In contrast, the specificity of US bile duct stone detection is extremely high (99–100%) [53]. Normal bile duct diameter is considered to be less than 6 mm. One should also realize that normal-sized bile ducts do not exclude the presence of bile duct stones. Endoscopic US is another method to get a diagnosis. This technique permits the visualization of the bile duct, including the distal part, which may be too difficult to examine with US. In expert hands, this technique has a high sensitivity for the detection of bile duct stones [54]. Magnetic resonance cholangiopancreatography (MRCP) has recently been introduced as a one-step noninvasive investigation providing additional important information. MRCP allows the visualization of bile ducts as well as pancreatic ducts. The advantage of this technique compared to US is its increased sensitivity (overall >90%) in detecting bile duct stones. Sensitivity may be lower in the case of

small stones in strongly dilated bile ducts. Endoscopic retrograde cholangiopancreatography (ERCP) is commonly considered the gold standard for diagnosing bile duct stones. Although widely regarded as a safe procedure, ERCP carries a small but significant number of serious complications [55]. Recent studies have shown a morbidity of 3 % for diagnostic ERCP, particularly post-ERCP pancreatitis, and a mortality of 0.2 % [56]. In expert hands, success rates to cannulate the bile ducts are at least 90–95 %. A considerable advantage of the procedure is the possibility to proceed to therapeutic ERCP with papillotomy, stone extraction, and/or nasobiliary drainage or stenting.

12.4.4 Treatment

There are a number of factors to be considered when planning treatment strategies for acute gallstone cholangitis. These include the severity of cholangitis, the presence of concurrent medical illnesses, the underlying cause, and the available treatment options at each institution. Patients with cholangitis are best treated in hospitals where a management protocol exists, including resuscitation, antibiotic therapy, and the possibility of performing an effective, also endoscopic, biliary drainage. Biliary drainage is essential in the treatment of acute cholangitis, and its importance has been recognized for nearly the last 100 years. Urgent biliary decompression is required by 10–20 % of patients, based on their clinical condition at presentation. Nonoperative biliary drainage (endoscopic or transhepatic) has revolutionized the treatment of acute cholangitis and is preferred to emergency surgical decompression. Endoscopic drainage (ERCP) is now considered the treatment of choice in the case of acute gallstone cholangitis. The aim of ERCP with endoscopic sphincterotomy (ES) in patients affected by acute cholangitis due to CL is to quickly decompress the biliary tree

to resolve the biliary stasis, which is responsible for sepsis. This choice allows for the extraction of stones via the duodenum or, if this is not possible, for the placement of an internal stent or nasobiliary tube to drain the biliary tree obstructed by stones. An aggressive management with early endoscopic biliary drainage has been shown to be associated with a very low mortality rate [57]. On the other hand, delay in ERCP for patients with severe cholangitis has resulted in increased mortality and morbidity [58]. Percutaneous transhepatic drainage (PTD) is reserved for patients in whom endoscopic drainage fails for either technical or anatomical reasons. Percutaneous transhepatic cholangiography can be used for biliary drainage by passing a catheter into a dilated intrahepatic duct, and this route may also be used to retrieve stones via a Dormia basket [59]. Surgery represents the last option if instrumental techniques have failed. The surgical options are choledocolithotomy, bilioenteric anastomosis, and papillosphincterotomy. In patients with gallstone cholangitis, the definitive treatment should include cholecystectomy. If the stones are successfully cleared endoscopically, the patient simply proceeds to laparoscopic cholecystectomy (LC).

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Chapter 13

Endoscopic Techniques for Biliary and Pancreatic Acute Diseases

Stefania Gherzi, Marco Bassi, Carlo Fabbri, Anna Larocca, and Vincenzo Cennamo

13.1 Introduction

Digestive endoscopy has a central role in the diagnostic and therapeutic management of most gastrointestinal diseases, but in the years has had a truly revolutionary impact, especially in reference to the treatment of many diseases, allowing less invasive management of conditions otherwise only surgically treated. Moreover, if we consider the increase in life expectancy in Western countries, resulting in greater demand for health services by the octogenarian population, the possibility of offering a therapeutic, less invasive alternative may be even more important. More specifically, over the last 40 years, the primary role of endoscopic retrograde cholangiopancreatography (ERCP) in the diagnosis and therapy of biliopancreatic diseases

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has changed drastically with the introduction of new diagnostic procedures such as ultrasound (US), computerized tomography (CT), endoscopic ultrasonography (EUS), and magnetic resonance (MR), but leaving ERCP the role of main therapeutic tool for obstructive biliopancreatic acute diseases.

13.2 Biliopancreatic Acute Diseases

13.2.1 Acute Cholangitis

Acute cholangitis (AC) is a pathological condition, potentially lethal, that can manifest itself with a wide spectrum of severity. The main factor for the onset of cholangitis is an obstacle to the biliary outflow, with consequent increase of pressure and presence of bacteria in the biliary tree. The endoscopy has therefore assumed a prominent role in patients with acute cholangitis. The ability to perform a biliary decompression with nonsurgical methods is an important therapeutic option, easily available, and, in a high percentage of cases, effective. A biliary obstruction can occur in various pathological conditions summarized in Table 13.1.

The two main complications of the common bile duct stones are cholangitis and acute pancreatitis (AP). Both of these complications can lead to clinical pictures of different severities up to a possible rapid, unpredictable, and fatal evolution. Therefore, early detection and proper therapeutic management are essential to reduce mortality.

Table 13.1 Main causes of biliary obstruction

Common bile duct stones (40–75 %)
Malignant strictures (15–40 %)
Benign strictures
Postoperative iatrogenic strictures
Bile duct cysts

Table 13.2 TG13 diagnostic criteria for acute cholangitis

A: Systemic inflammation

A-1 Fever (>38 C) and/or shaking

A-2 Laboratory data: evidence of inflammatory response (WBC <4 or >10109/L, CRP 10 mg/l, and other changes indicating inflammation)

B: Cholestasis

B-1 Jaundice (serum bilirubin 34.2 mmol/l)

B-2 Laboratory data: abnormal liver function tests (ALP, GGT, ALT, AST >1.5xULN)

C: Imaging

C-1 Biliary dilatation

C-2 Evidence of the etiology on imaging (stricture, stone, stent, etc.)

Almost 80 % of cholangitis are due to choledocholithiasis. The typical symptoms of cholangitis are characterized by jaundice, pain, and fever (Charcot's triad); but the three symptoms are not always all present: septic fever with shivering occurs in up to 90 % of cases, jaundice in 60 %, and pain in about 50 %. In severe forms (about 5 %), the patient shows clinical signs of shock associated with an altered mental status [1]. The diagnosis of cholangitis based on clinical features is usually easy; however, it is important to keep in mind that sometimes even subjects in relatively good general condition can develop a suppurative cholangitis, characterized by the presence of pus in the biliary tree. So, it's important to never underestimate the occurrence of infectious signs.

Table 13.2 shows the diagnostic criteria for acute cholangitis as defined by the TG13 Tokyo Guidelines [2].

Ultrasound is certainly the first diagnostic procedure to be performed, but its accuracy is unsatisfactory for a secure diagnosis of common bile duct stones. On the other hand, US can also be helpful in differentiating acute cholangitis from an acute cholecystitis or showing the presence of liver abscesses. In order to perform a biliary drainage, in cases of overt acute cholangitis,

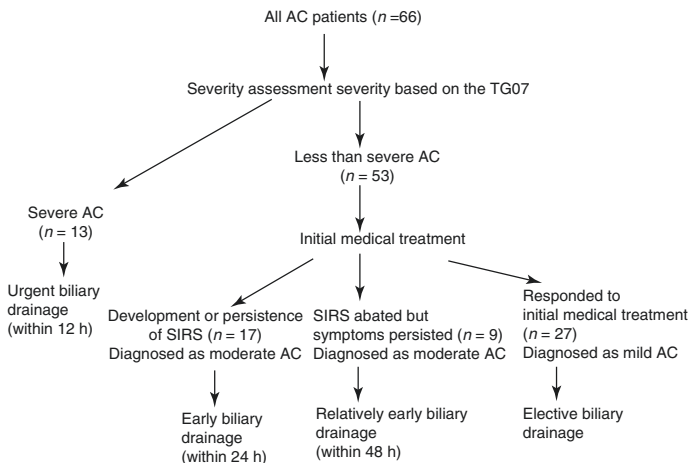


Fig. 13.1 Flowchart of the severity assessments and treatment of acute cholangitis according to the TG07

ERCP can also provide a diagnostic contribution, as well as the therapeutic one. The importance of early management for the outcome of severe sepsis and septic shock is now well established, especially in the control and restoration of hemodynamic conditions [3]. General support measures have a great prognostic value, but equally important is a timely biliary obstruction resolution. The need to obtain an effective biliary drainage, especially in cases that do not respond to antibiotic treatment, recommends the execution of an urgent ERCP [4]. In 2007, the Tokyo Guidelines for the management of acute cholangitis and cholecystitis were first published [5], subsequently revisited in 2013 [2]. Since then, they provided useful recommendations for the diagnosis, severity assessment, and management of AC. Figure 13.1 reported the flowchart of the severity assessments and treatment of acute cholangitis according to the TG07 with particular reference to selection criteria for urgent or early biliary drainage [6].

13.2.2 Acute Pancreatitis

A pancreatic involvement in choledocholithiasis results in most cases with a modest and transient hyperamylasemia. Generally, this clinical situation does not require diagnostic and therapeutic choices and is not associated with different outcomes from that of the uncomplicated choledocholithiasis. More rarely, however, the patient develops an acute pancreatitis which, in 20% of cases, evolves toward a clinically severe form, with a mortality rate that can reach 95% of cases.

According to the recent IAP/APA Guidelines, the definition of acute pancreatitis is based on the fulfillment of “two out of three” of the following criteria: clinical (upper abdominal pain), laboratory (serum amylase or lipase $>3\times$ upper limit of normal), and/or imaging (CT, MRI, ultrasonography) criteria (grade 1B, strong agreement) [7].

The unpredictable clinical evolution, the complexity of the disease, and the possible involvement of multiple organs require organizational, as well as diagnostic and therapeutic, choices. It also requires a multidisciplinary approach but, above all, a ready, effective, and safe management. In acute pancreatitis there is absolute consensus in the literature that ERCP can be lifesaving if the etiology is definitely biliary pancreatitis. Therefore, urgent ERCP is strongly recommended in cases with severe clinical picture, in the presence of jaundice, and in case of cholangitis. In these situations the endoscopic treatment is effective in reducing the complications, especially infective ones, and mortality. Less evidence exists on what should be the therapeutic management of the patient in which the pancreatitis is clinically mild/moderate, as there are no signs of obstruction (jaundice, ultrasound demonstration of choledocholithiasis, and/or dilatation of the bile ducts) or biliary infection. In these subjects the execution of ERCP is more controversial. In the absence of a diagnosis of biliary obstruction, less invasive diagnostic

Table 13.3 Biliary pancreatitis if one of the following definitions is present

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1. Gallstone and/or sludge on imaging (transabdominal or
 2. Endoscopic ultrasound or computed tomography)
 3. If no gallstones or sludge, a dilated common bile duct on ultrasound (>8 mm in patients ≤ 75 years old or >10 mm in patients >75 years old)
 4. The following laboratory abnormality: alanine aminotransferase (ALAT) level >2 times higher than normal values, with ALAT > aspartate aminotransferase
-

alternatives such as EUS and MR are available, despite the poor accuracy in detecting very small stones. The Dutch Pancreatitis Study Group provided the following criteria to define a biliary origin of acute pancreatitis as reported in Table 13.3 [8].

Over the last 30 years, the role of early ERCP in acute biliary pancreatitis remains disputed with a number of clinical trials and meta-analyses producing conflicting results [9]. In addition, the use of different diagnostic and therapeutic techniques is often closely related to the skills available.

13.2.3 Recurrent Acute Pancreatitis (RAP)

Recurrent acute pancreatitis is defined as more than two attacks of acute pancreatitis (AP) without any evidence of underlying chronic pancreatitis (CP). RAP usually occurs in the idiopathic group, which forms 20–25 % of cases of AP. The causes of idiopathic RAP (IRAP) can be mechanical, toxic–metabolic, anatomical, or miscellaneous. Microlithiasis is commonly reported from the Western countries; pancreas divisum (PD) is now believed as a cofactor, the main factor being associated with genetic mutations. The role of sphincter of Oddi dysfunction (SOD) as a cause of IRAP remains controversial. Malignancy should be ruled out in any patient with IRAP >50 years of age.

Early CP can present initially as RAP. Idiopathic recurrent acute pancreatitis represents a great diagnostic and therapeutic challenge. If the etiology remains undetermined, repeated attacks are associated with significant morbidity with the inevitable result of chronic pancreatitis [10, 11].

13.3 Endoscopic Management of Acute Biliopancreatic Diseases

In the past, surgery was the only effective form of treating severe septic cholangitis, but showed a high incidence of morbidity and mortality (13–88%) [12]. In recent years nonsurgical methods have established and allow effective and rapid biliary decompression: the transhepatic and endoscopic drainage. Endoscopic therapy represents a fundamental progress to a more rational and beneficial approach to the patient. Indeed, it allows to make a precise diagnosis of the cause of the obstruction and, very often, to perform definitive treatment in the same session, with lower incidence of complications and mortality compared to other methods.

13.3.1 ERCP

The most frequent causes of acute cholangitis are choledocholithiasis and benign strictures. Rarely malignant obstructions occur with a spontaneous cholangitis. The endoscopic therapy involves the sphincterotomy, the extraction of stones, and the application of nasobiliary drainage or of a stent, according to the different pathological situations. The success rate of endoscopic drainage is up to 90% with the resolution of severe cholangitis in 95% of cases [13].

The first step of endoscopic treatment is the sphincterotomy, performed after contrast medium injection, in order to define the location and nature of the obstruction. The

sphincterotomy is performed according to well-defined techniques, with variable amplitude depending on the detected pathology. In the case of biliary stones, the incision must always be extended so as to affect the whole course of the intramural choledochus, to guarantee an efficient drainage, and to allow the extraction of the stones. In case of neoplastic or benign stricture, an incision of lesser entity for the introduction of stents may be sufficient.

So, the biliary cannulation can be considered the prerequisite to ensure a safe and effective procedure, but the failure rate of standard technique may range from 15 to 35 % of cases, even when performed by experienced endoscopists [14]. In some cases you must resort to alternative techniques to cannulate the biliary duct, such as the needle-knife sphincterotomy. The term “precut” has also been used to describe this technique because an incision is made on the papilla prior to free cannulation and/or guidewire cannulation. Precut sphincterotomy is widely considered to be a risky procedure that should be used only by experts and only when all reasonable efforts at gaining access to the biliary tree by conventional methods have failed [15]. On the other hand, precut sphincterotomy is reasonable if attempts at conventional cannulation have failed and if there is a compelling need to have biliary access (suspected malignant jaundice, common duct stones, cholangitis, etc.). The decision must be made after an honest appraisal of one’s own endoscopic skill, the skill of others immediately available, and the availability of percutaneous or surgical methods. In experienced hands and in highly selected cases, precut sphincterotomy can be safe and effective, and sometimes you can be lucky as shown in Video 13.1.

In most cases stone removal can be obtained by using the standard extraction maneuvers with Dormia basket or balloon catheter. In patients with a so-called difficult lithiasis, elimination of the stones may require the use of associated techniques: intracorporeal lithotripsy and mechanical, hydraulic, or extracorporeal shock wave lithotripsy (ESWL). If it is not possible to

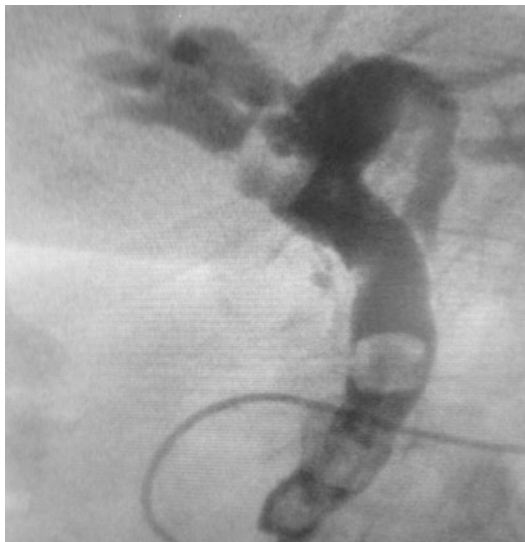


Fig. 13.2 Massive stones stacked in the common bile duct

remove the stones, in the same session, for technical reasons (size, number, concomitant stenosis), or to reduce the time and endoscopic maneuvers due to particularly poor general condition of the patient, a nasobiliary drainage tube should be introduced, and refer the removal of stones at a later session (Fig. 13.2). The success rate of endoscopic treatment, in obtaining the biliary drainage, is currently very high (about 95%), in skilled hands, while some limitations may be encountered to perform the complete removal of the stones. If acute cholangitis is supported by neoplastic biliary obstruction, an emergency drainage is generally necessary, or at least as early as possible, because in these conditions, the clinical picture is quite severe and still has a tendency to evolve negatively. Biliary drainage by endoscopic stents can be considered a therapeutic measure in all patients with jaundice due to neoplastic stenosis. It can be the

definitive treatment in cases not suitable for surgery for spread of the disease or severe general conditions. Stent insertion can be also a useful preoperative treatment in cases of reversible inoperability helping to bring the patient in surgical conditions and to reduce the operative mortality. In benign strictures, complicated by cholangitis, endoscopy is applied in the same way to that described for malignant stricture. Endoscopy is therefore the crucial point of reference for the therapeutic approach to acute cholangitis. In case of impossibility of using endoscopic technique, or its failure, other ways to perform the biliary decompression should be considered.

As regards the treatment of acute pancreatitis, endoscopic pancreatic sphincterotomy (EPS) is considered the cornerstone of endoscopic therapy, and once access is obtained, EPS may be used as a single therapeutic maneuver (e.g., to treat pancreatic-type sphincter of Oddi dysfunction) or in series with other endoscopic therapeutic techniques such as stone extraction or stent placement. Treatment of patients with IRAP is aimed at the specific etiology. Endoscopic sphincterotomy is advised if there is strong suspicion of SOD. Minor papilla sphincterotomy should be carried out in those with PD but with limited expectations. Regular follow-up of patients with IRAP is necessary because most patients are likely to develop CP in due course.

13.3.2 EUS-Guided Biliary Drainage

Over the years the diagnostic tools used in the gastroenterological field have undergone substantial progress, thanks to the introduction of new methods which broadened the diagnostic horizons and offer new therapeutic possibilities. Endoscopic ultrasonography is a new imaging technique that has been introduced in clinical practice about 30 years ago and, since then, still gives an important contribution in endoscopic diagnostics. In recent years this technique also obtained a therapeutic role.

Endoscopic ultrasound-guided biliary drainage (EUS-BD) has recently emerged as an effective alternative biliary drainage method after unsuccessful ERCP in malignant biliary disease.

ERCP occasionally fails for anatomical or technical problems such as upper intestinal obstruction, surgically altered anatomy, periampullary diverticulum, or periampullary tumor infiltration, despite a success rate higher than 90% in most reports. Percutaneous transhepatic biliary drainage (PTBD) or surgical interventions are conventionally performed as alternative biliary drainage methods after unsuccessful ERCP. However, both PTBD and surgical interventions are associated with considerable morbidity and mortality [16]. Even if guidelines have not yet been established, EUS-BD should be considered any time that conventional endoscopic retrograde cholangiopancreatography for biliary drainage is not possible or unsuccessful, even in expert hands [17].

Wiersema and colleagues in 1996 published the first series of 11 patients undergoing EUS-guided cholangiopancreatography after unsuccessful ERCP, demonstrating its feasibility [18]. Giovannini and colleagues subsequently described the first case of choledochoduodenal fistula with stent placement for biliary decompression in 2003 [19]. Since then, numerous approach techniques have been described that can be essentially divided into three different techniques: (1) EUS-guided transluminal biliary drainage including choledochoduodenostomy and hepaticogastrostomy, (2) EUS-*rendezvous* (EUS-RV) technique, and (3) EUS-antegrade approach [20]. In this last one, using linear EUS, the bile duct or the pancreatic duct can be punctured with a needle in locations where these ducts are in close proximity to the gastrointestinal lumen. The access to the biliary tract can be effected via trans-gastric (intrahepatic) approach or by transduodenal (extrahepatic) approach. Contrast injection through the fine-needle aspiration (FNA) needle allows for EUS-guided cholangiography (ESC). Once the cholangiogram has been obtained, ERCP accessories are then used through the working

channel of the echoendoscope to complete the procedure. ESC therefore represents a hybrid technique that combines EUS-guided FNA and ERCP (Video 13.2). In EUS-RV, the biliary duct is accessed under EUS and fluoroscopic guidance with the creation of a temporary fistula followed by guidewire placement via the biliary duct and ampulla into the duodenum. So, ERCP is reattempted using the EUS-placed guidewire. The guidewire is removed once biliary cannulation is obtained. Therefore, EUS-RV should be attempted for patients with an endoscopically accessible ampulla after failed biliary cannulation in conventional ERCP (Video 13.3).

In conclusion we can say that many of biliopancreatic acute diseases can be considered endoscopic diseases. At present, EUS-guided drainage is not yet able to replace ERCP drainage, despite huge progress in recent years. Technical and skillness improvements are still necessary, and it is becoming increasingly clear that, in reference centers, endoscopic skills should have the technical expertise to perform such complex procedures.

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Chapter 14

Laparoscopy in Surgical Emergencies

David Costa-Navarro and Manuel Romero-Simó

14.1 Introduction

The minimally invasive approach, as defined by Di Saverio, refers to a surgical procedure performed through smaller than one centimeter abdominal incisions, as opposed to the traditionally larger and more painful laparotomy ones, therefore captivating the patient's preference [1]. Laparoscopy is a currently spread technique for most procedures and, in some of them, has become the gold standard for optimal results in terms of recovery, costs, hospital stay, and so on. Nevertheless, only a few years ago, laparoscopy was presented as a developing technique with better cosmetic results and better comfort for the patient, but marked in some cases by higher rate of some complications and higher costs. In the recent years, the technique itself has improved for all procedures and also the instruments; thus the open approach is not acceptable for the most except in the case of contraindications for a laparoscopic approach, even in emergency cases.

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14.2 Laparoscopy: The Beginning

The word “laparoscopy” is a medical neologism coming from the German word *Laparoskopie* which, at the same time, comes from two Greek words: *laparos* (=flank) and *scopia* (=instrument to see through). Therefore, the meaning of the word would be “device to look through the flanks” [2]. The original idea of performing laparoscopy was reported more than one century ago, but the clinical application of the concept is much more recent. The first of these technical advances was reported in 1805 when the Frankfurt-born physician Philipp Bozzini successfully visualized the human urethra. His technique for this exploration involved the construction of an instrument consisting of a light chamber illuminated by a wax candle, from which a tube could be introduced into body cavities. A mirror reflection was used to illuminate shallow orifices for examination and manipulation [1]. Because the urethra was the easiest cavity to explore in this manner, it was the first to be examined. Later on, Bozzini was also able to view the urinary bladder and to visualize stones and neoplasms, but despite these accomplishments, Bozzini was reprimanded for his invention, which was discounted as a “toy” [3, 4].

Laparoscopy or endoscopically examining the peritoneal cavity was first attempted in 1901 by George Kelling who called this examining procedure “celioscopy,” but he failed in publishing his work [1, 2]. The investigator generally considered to be the man responsible for popularizing the technique in humans was the physician from Stockholm called Hans Christian Jacobaeus, whose technique involved the use of a trocar to establish pneumoperitoneum. He published an article in 1912, in which he described 109 laparoscopies performed on 69 patients [3, 5].

One of the most important recommendations made was the idea proposed by Zollikofer in 1924, who wrote about the benefit of obtaining pneumoperitoneum by using carbon

dioxide because it avoided some thermal complications and resulted in less pain [5]. Thus, techniques of laparoscopic surgery truly became integrated into the discipline of general surgery until after 1986, by developing a video chip that allowed the projection of the images obtained by the endoscope to the television screen. The history of laparoscopic surgery continues to unfold with every refinement in technique and instrumentation that is developed, and nowadays, laparoscopy is the gold standard for many procedures not only in digestive surgery but also in other surgical specialties such as gynecology, thoracic surgery, urology, and so on. Particularly, recent evolution of technology has dramatically changed the range of available instruments and, subsequently, the therapeutic options that can be offered to patients needing surgical interventions and eventually even emergency surgery.

14.3 The Introduction of Laparoscopy to Emergency Procedures

In the past decades, few pioneering experiences have highlighted the potential advantages of diagnostic and therapeutic laparoscopy for the management of acute abdomen but have also advocated a better definition of the exact role of emergency laparoscopic surgery [4, 5]. Laparoscopy is extensively used for elective surgery, but, classically, there are some arguments for not using laparoscopy in emergency cases:

- Laparoscopy in emergency is still considered too challenging; technical difficulties are encountered for most surgeons that are used to elective procedures but lacking experience in emergency cases [6].
- Difficulties to plan a laparoscopic approach for after-hours emergencies or during a night shift or lack of accessibility to

the instruments in those cases have been discussed as reasons for making laparoscopic approach in emergencies [6].

These and many more issues contribute to make a laparoscopic approach challenging and risky in an emergency setting and have prevented the development of “laparoscopic emergency surgery.” Nevertheless, with adequate experience and appropriate laparoscopic skills associated with laparoscopic techniques that have been conveniently modified and adjusted for acute care surgery, recent publications have reported good results and have made laparoscopy as a recommendable approach in certain cases. This approach can be safely used in cases of appendicitis complicated with diffuse peritonitis or large purulent abscesses and in cases of acute cholecystitis, even gangrenous or perforated, or associated with common bile duct lithiasis and/or acute pancreatitis, among others. Edematous or fibrotic cholecystitis can harbor technical challenges, but, indeed, appropriate skills may allow the safe completion of a laparoscopic procedure [1], as well as cases of bowel obstruction, certain cases of acute diverticulitis, incarcerated hernias, and even polytrauma patients.

The only real contraindication to the use of laparoscopy in an emergency setting as an acute care surgery procedure is in patients exhibiting hemodynamic instability and severe hemorrhagic or septic shock. The induction of pneumoperitoneum and venous flow return compromise may be easily fatal in such cases. A further relative contraindication to be considered remains a severe respiratory failure with severe hypercapnia, owing to the possible reabsorption of CO₂ and development of malignant hypercapnia and toxic shock syndrome [7]. However, a wise ventilatory strategy, increasing the minute volume of ventilation, and further measures by decreasing the intra-abdominal pressure and the angle of Trendelenburg position might be helpful in mitigating these challenges.

As previously mentioned, laparoscopy has clear benefits for the patient in terms of less pain, less wound infections, and other types of complications such as cardiovascular ones, shorter length of stay, and others. These benefits are relevant not only in young patients but, contrary to commonly held beliefs, even more significant in the elderly patients [1].

14.4 Laparoscopy in Acute Appendicitis

Open acute appendicitis (AA) is the most frequent cause of acute abdominal pain in Western countries, marked with an incidence of 100/100.000 cases per year [1], and the risk of having AA is around 8% in a lifetime [8–10]. Open appendectomy (OA) has been the standard surgical procedure for the treatment of AA for over a century, since it was described by McBurney in 1894 [11], and still remains the procedure of choice in many centers [8–10]. Subsequently, due to the development of endoscopic surgery, Semm introduced the laparoscopic appendectomy (LA) in 1981 [12], rendering a minimally invasive procedure for the skin and abdomen; although many studies published in the very early years of the twenty-first century, comparing OA and LA, didn't really determine a superiority of the laparoscopic approach [13–16], some more recent papers, however, substantiate that LA is the technique of choice in the treatment of AA in terms of clinical advantage and cost-effectiveness [8, 10, 12–18]. The relatively recent reluctance to accept laparoscopic appendectomy as the gold standard is due to many studies at the end of the twentieth century and the beginning of the twenty-first century that failed to prove the superiority of LA over OA for several reasons [18], but on the contrary, many recent papers have been published with substantially different results supporting LA as the technique of choice for all cases of AA instead of OA [18]. It was proven to

have better cosmetic effects and lower costs explained by shorter length of stay, shorter operating time (not described by all papers), and lower morbidity rate, even for complicated cases of acute appendicitis for expert surgeons.

Over the last decade, an innovative technique, single-incision laparoscopic surgery (SILS), has been developed with the aim to improve cosmetic effects, postoperative pain, and return to normal activity [19, 20]. Transumbilical SILS has recently attracted the attention of surgeons worldwide with the innovative possibility of performing virtually scarless surgery [21]. Several studies and randomized trials have tested and compared single-incision laparoscopic appendectomy (SILA) with LA showing similar postoperative results [22, 23]. Notwithstanding, the increased costs for SILA compared with LA are still a major disadvantage that limits this technique [24, 25]. Moreover, the use of angled instruments and the loss of triangulation between them, due to coaxiality, make SILS a difficult procedure requiring advanced laparoscopic skills. These factors may be associated with an increased rate of postoperative complications and longer surgical times [25]. In addition to the cosmetic results related to the reduced number of incisions (and trocars), this has also been thought to decrease postoperative pain and to accelerate postoperative recovery, but in some studies, SILA has been found to cause even more pain in the trocar site than laparoscopic appendectomy due to its size. As for the length of hospital stay and return to normal activity, SILA recovery time is nearly equal to LA [22, 23] and is, therefore, not a singularly decisive factor in choosing one procedure over the other. Although the low rate of surgical site infection has been one of the primary advantages of LA over OA, the use of a single laparoscopic access point surprisingly emerged early on to potentially increase the risk of postoperative wound infections [26, 27]. Interestingly, a large retrospective study found that the wound infection rate became smaller over time, suggesting that the

infection rate may depend more on the surgeon's experience than with the technique [6].

Hence, we can conclude that the classic three-port laparoscopy is the recommended approach for all cases of acute appendicitis unless formal contraindication.

14.5 Laparoscopy in Perforated Peptic Ulcers

Every year, peptic ulcer disease (PUD) affects 4 million people around the world. Complications are encountered in 10–20% of these patients and 2–14% of the ulcers will perforate. Perforated peptic ulcer (PPU) is relatively rare, but life-threatening with the mortality varying from 10 to 40%. When PPU are diagnosed expeditiously and promptly treated, outcomes are excellent. Mortality ranges from 6 to 14% in recent studies. Poor outcomes have been associated with increasing age, major medical illness, per-operative hypotension, and delay in diagnosis and management (greater than 24 h) [28].

Although some authors have published and described nonoperative management of peptic ulcers in certain cases, nowadays there is no recommendation for conservative treatment; therefore, surgery must be indicated in patients presenting with pneumoperitoneum and signs of peritonism.

Laparoscopic surgery has brought new possibilities in the way these cases are managed: laparoscopy allows the confirmation of the diagnosis and furthermore allows the identification of the position, site, and size of the ulcer [28]. The procedure also allows stitching the defect and adequate peritoneal cleanup without the need for a large laparotomy that, otherwise, in an open access would be required. In the rare occurrence of large perforation with a profound contamination, conversion may be required for complete peritoneal lavage.

It remains very clear that the surgical steps of the treatment must be treatment of the perforation, peritoneal aspiration, and gentle lavage and omental patch, if possible, along with drainage placement. It is arguable if there is any standard laparoscopic technique to treat the perforation. Considering that one of the problems with emergency cases is the technical difficulty, some authors have advocated sutureless ulcer repair, which would simplify the laparoscopic technique. Nevertheless, it was considered as safe as suture repair, but it carried extra costs such as the use of fibrin glue, and, although the rationale of this sutureless technique was to simplify the procedure and shorten operative time, it did not gain wide acceptance owing to its high leakage rate as compared to suture repair (16–6%) [28].

In the case of large perforations, its closure may not be performed or, at least, safely enough; for those cases with perforations larger than 2 cm, suture plus diversion or resection should then be performed, but in these cases, conversion to open surgery should be done.

Di Saverio et al. proposed the following recommendations in the laparoscopic approach of perforated peptic ulcers in his WSES position paper published in 2013 [28]:

- Laparoscopic sutureless repair may be a viable option in the presence of limited laparoscopic experience, only in the presence of small-sized perforations (i.e., microscopic or <2 mm perforations) without significant peritoneal contamination and for low-risk patients.
- Primary repair in case of perforated peptic ulcer larger than 5 mm and smaller than 2 cm.
- We suggest routine use omental patch to further protect the suture line.
- We recommend avoiding use of glue as only method of closure of PPU.
- We suggest use of glue only as an adjunctive measure to protect suture line or the omental patch.

14.6 Bowel Obstruction

Acute intestinal obstruction is a frequent presentation in emergency. The small bowel obstruction (SBO) is the most frequent site of obstruction (76%), and adhesions following open surgery are the most common etiology (65%) (Markogiannakis 2007). Managing SBO by means of surgery may cause new adhesions, whereas conservative treatment does not remove the cause of the obstruction [19]. Conservative treatment involves nasogastric intubation, intravenous fluid administration, and clinical observation. Strangulation of the bowel requires immediate surgery, but intestinal ischemia can be difficult to determine clinically [29]. Therefore, the indication of the correct management can be sometimes controversial; nevertheless, some recommendations for the management of the SBO have been determined based on evidence:

- Patients without the signs of strangulation or peritonitis or history of persistent vomiting or combination of CT scan signs (free fluid, mesenteric edema, lack of feces signs, devascularized bowel) and partial SBO can safely undergo nonoperative management (level of evidence 1a, grade of recommendation A) [29].
- Patients who had surgery within the six weeks before the episode of small bowel obstruction, patients with signs of strangulation or peritonitis (fever, tachycardia, leukocytosis, metabolic acidosis, and continuous pain), patients with irreducible hernia, and patients who started to have signs of resolution at the time of admission are *not* candidate for conservative treatment with or without water-soluble contrast administration (level of evidence 1a, grade of recommendation A) [29].
- Complete SBO (no evidence of air within the large bowel) and increased serum creatine phosphokinase predict NOM failure (level of evidence 2b, grade of recommendation C).

- Free intraperitoneal fluid, mesenteric edema, lack of the “small bowel feces sign” at CT, and history of vomiting, severe abdominal pain (VAS>4), abdominal guarding, raised WCC, and devascularized bowel at CT predict the need for emergent laparotomy at the time of admission (level of evidence 2c, grade of recommendation C) [29].
- The appearance of water-soluble contrast in the colon on abdominal X-ray within 24 h of its administration predicts resolution of ASBO (level of evidence 1a, GoA). Among patients with ASBO initially managed with a conservative strategy, predicting risk of operation is difficult [29].
- Non-responding patients to long tube and conservative treatment (level of evidence 2b, grade of recommendation) [29].

The first laparoscopic adhesiolysis for small bowel obstruction was performed by Clotteau in 1990. Following this first case, laparoscopy has been delivered for treating SBO by several surgeons, because of its perceived advantages in selected cases. Open surgery is the preferred access to treat SBO, once indication for surgery has been set and laparoscopy in SBO has no clear role yet; it may have a therapeutic and diagnostic role as well. In several series laparoscopic or laparoscopy-assisted surgery is considered feasible and convenient more than conventional surgery for SBO; there is also reason to suspect both difficulties and risks. In this way, it has been indicated that intraoperative selection of patients after exploratory laparoscopy is a good practice, because this approach allows as many patients as possible to benefit from this mini-invasive procedure. The only absolute exclusion criteria for laparoscopic adhesiolysis in SBO are the presence of pneumoperitoneum, hemodynamic instability, or cardiopulmonary impairment and the lack of laparoscopic skills. The influence of dense adhesions and the number of previous operations on the success of laparoscopic adhesiolysis are controversial. Some authors have stated that documented history of severe or extensive dense adhesions is a

contraindication to laparoscopy. When other etiologies are found, such as internal hernia, inguinal hernia, neoplasm, inflammatory bowel disease, intussusception, and gallstone ileus, conversion to a minilaparotomy or a formal laparotomy is often required. However, no randomized controlled trial (RCT) comparing open to laparoscopic adhesiolysis exists up to date, and both the precise indications and specific outcomes of laparoscopic adhesiolysis for adhesive SBO remain poorly understood. The only RCT on laparoscopic adhesiolysis assessed the incidence of chronic abdominal pain after randomization to laparoscopic adhesiolysis or no treatment during diagnostic laparoscopy, and it failed to demonstrate any significant differences in terms of pain or discomfort [29].

14.7 Acute Diverticulitis

Diverticulosis is nowadays a common disease of the large bowel; its prevalence increases with age, being 5% in 30–39-year-old population but rising up to 60% above in those aged over 80 years [30]. Acute diverticulosis may be non-symptomatic, but progressing to bleeding, diverticulitis, or perforation. It was reported that up to 25% of patients sustaining diverticuli in the large bowel may develop acute diverticulosis², but more recent data show that there was an overestimation and only 4% of these patients will develop an episode of inflammation [31]. Diverticulitis admissions vary from 70 to 160 per 100,000 population in Western countries. Meanwhile, perforated diverticulitis has an estimated adult incidence of only 3·5 per 100,000 population. Nonetheless, acute diverticulitis represents an increasing surgical problem worldwide [32].

Based on the presence of complications such as abscess, fistula, obstruction, or perforation, diverticulitis can be classified

as either complicated or uncomplicated. Complicated diverticulitis, demonstrated by CT imaging and graded according to the Hinchey classification, often requires operative or percutaneous intervention [32].

Hinchey classification is the most used classification to distinguish different degrees of acute inflammation and complication. In the past, emergency surgery for acute diverticulitis was performed in 15 % of cases to manage acute diverticulitis complicated by intra-abdominal or pelvic abscess. Today, it is commonly assumed that a small abscess of less than 4 cm without peritonitis (Hinchey stage 1) can be managed successfully with broad-spectrum antibiotics, bowel rest, and observation only, whereas, in cases of peridiverticular abscess larger than 4 cm (Hinchey stage 2), CT-guided percutaneous drainage is recommended [33]. Hinchey stage 3 and 4 diverticulitis, the presence of a large inaccessible abscess, as well as the lack of improvement or deterioration within three days of conservative management are all well-accepted indications for emergency operative treatment [32].

In this way, the World Society of Emergency Surgery has more recently proposed a newer classification based on CT scan findings and clinical presentation too. Based on this classification, decision-making in nonoperative and operative management of acute diverticulitis can be accurately done [34] (Table 14.1).

14.8 Management Based on Grade and Clinical Status

1. *Stage 1*: patients may be managed as outpatients without antimicrobial antibiotics unless they have risk factors (immunosuppression) or signs of sepsis.
2. *Stage 2A*: clinically stable patients may be treated initially by oral treatment. Any patients require intravenous antibiotics.

Table 14.1 Classification based on CT findings

Grade	CT findings
1	Diverticula, thickening of the wall, increased density of the pericolic fat
2A	Pericolic air in the form of air bubbles or little pericolic fluid without abscess
2B	Abscess <4 cm (without distant free air)
3	Abscess >4 cm (without distant free air)
4A	Diffuse fluid without distant free air
4B	Localized fluid associated to distant free air
4C	Diffuse fluid with distant free air

In these patients intravenously antimicrobial therapy should be shift to oral therapy as soon as possible.

3. *Stage 2B*: patients may be treated by antibiotics alone. If antimicrobial treatment fails, percutaneous drainage is suggested.
4. *Stage 3*: diverticular abscesses greater than 4 cm (stage 3) are best treated by percutaneous drainage as long as the patients do not have severe sepsis or septic shock. When percutaneous treatment is not feasible, laparoscopic peritoneal lavage appears to be a useful option.
5. *Stage 4A*: laparoscopic peritoneal lavage may be useful for management of generalized peritonitis without signs of colonic perforation in clinically stable patients without comorbidities, because it can avoid a probable stoma. After laparoscopic peritoneal lavage, no improvement of clinical conditions or rapid deterioration of clinical conditions suggests prompt colonic resection. Surgical resection and anastomosis with or without stoma may be suggested for stable patients with no multiple comorbidities. Hartmann resection should be carried out either in unstable patients (severe sepsis/septic shock) or in patients with multiple comorbidities.
6. *Stage 4B*: abscess or localized fluid associated to distant air may have a higher risk of failure and recurrence and may

necessitate surgical resection. Surgical resection and anastomosis with or without diverting ileostomy are suggested. Hartmann resection is suggested in high-risk patients.

7. *Stage 4C*: Hartmann resection is still useful in managing diffuse peritonitis with signs of diverticular diffuse perforations; however, in clinically stable patients with no comorbidities, primary resection with anastomosis and diversion stoma may be performed. Damage control surgery may be a useful strategy in clinically unstable patients with perforated diverticulitis (severe sepsis/septic shock).

As recommended by the WSES, laparoscopy has applications in emergency setting in cases of acute diverticulitis. In perforated diverticulitis, it can not only confirm the diagnosis but can also be therapeutic by allowing the lavage of the peritoneal cavity. Laparoscopic lavage requires careful patient selection and assessment for occult perforations. In 1991 the first laparoscopic sigmoid colectomy for diverticular disease was reported by Jacobs [35]. To date, many studies and a few randomized trials have been published on this subject [36]. Elective laparoscopic resection emerged to be feasible and safe as well as associated with increased operative time with fewer postoperative complications and shorter hospital stay as compared to standard open colectomy [36]. Indications for laparoscopic colectomy remain uncertain and not yet widely accepted [37]. Laparoscopic colectomy in emergency setting is likely to become adopted as the standard surgical procedure for complicated diverticulitis, but this requires surgeons in general to become more confident with the technique.

14.9 Acute Cholecystitis

Acute cholecystitis is an extremely prevalent disease on practice, and 36% of cholecystectomies are performed for acute

cholecystitis [38] on average, which are calculous in 60% of the cases. Laparoscopic cholecystectomy has already showed its superiority to open surgery in terms of recovery, complications, costs, and cosmetic effects; therefore, around 90% of cholecystectomies are nowadays performed laparoscopically [33, 39–48] out of 1.5 million laparoscopies performed per year [39]. It has been clearly stated, according to the recent recommendations, that cholecystectomy should be done in the first 24 h, having been proved to be superior to other approaches [41], but even up to 72 h after symptom onset is a good policy; otherwise, antimicrobial and symptomatic treatment should be given to the patient. This is due to the reason of conversion to and bile duct injury probability in those cases in which inflammation adhesions have already been established [42–44].

In the past, there were concerns with LC due to the higher morbidity rates in emergency procedures and the higher conversion rate to an open procedure [45]. Severe inflammation and fibrotic adhesions are the main reasons for conversion in early and delayed laparoscopic cholecystectomy, respectively [42, 43], and are the most important causes associated with bile duct injury [44]. Although retrospective studies reported a larger number of bile duct injuries associated with early laparoscopic surgery [46, 47], no significant differences have ever been found in randomized trials [40]. The optimal timing of surgery in cases of acute cholecystitis has always been a topic of debate. Not long ago, patients were managed conservatively for the purpose of cooling down the inflammatory process so that operations could be performed weeks later. Current data suggest that early laparoscopic cholecystectomy for acute cholecystitis is superior to late or delayed laparoscopic cholecystectomy in terms of outcome and costs [39]. During the first 72 h, surgical dissection may be easiest because of the lack of organized adhesions, reducing the risk of bile duct injuries and decreasing the rate of complications.

The concept of early cholecystectomy has now been discussed by some authors in terms of what should be considered the “golden hours.” In fact, although guidelines recommend laparoscopic cholecystectomy within the first 48–72 h after symptom onset, a recent randomized trial has showed laparoscopic cholecystectomy performed within 24 h of admission to be superior when compared to delayed laparoscopic cholecystectomy [6].

Transumbilical single-incision laparoscopic cholecystectomy (SILC) has recently been introduced with the intent to improve cosmetic results by leaving no visible exterior abdominal scars and theoretically reduce postoperative pain and enhance recovery compared to multitrocar laparoscopy. Interestingly however, randomized trials comparing SILC vs. conventional laparoscopy showed no differences in the visual analog scale pain score or postoperative analgesic administration [33, 48]. It is likely that total wound tension may rise nonlinearly with increasing incision length, and so tension across multiple incisions may be less than the total tension for a single incision of the same total length [49]. Accordingly, using two small trocars should be better than using a single large trocar [6]. In addition, despite the smaller skin incision in SILS, the total size of fascial defects may be equal to the size required for classic laparoscopy.

At the beginning, SILC has been associated with slightly longer surgical times [33, 48], mostly due to the advanced laparoscopic skills required to perform it. Recent papers showed that some surgical skills and maneuvers introduced to the technique have reduced surgical times and difficulty to the classical laparoscopy although these results have not been reproduced by all groups; nevertheless, SILC is still marked with higher complication rates (such as incisional hernias and wound complications). Also, these positive improvements have been described mainly for elective cases, but not for acute cholecystitis, marked with higher surgical difficulties [50]. Also, SILS is marked with higher costs mainly due to the trocar and the special instruments that are required, and although some studies have intended to show similar costs, this has not been proven.

As summary, classic laparoscopic cholecystectomy is the gold standard for the treatment of acute cholecystitis. SILC may be considered as a safe alternative to LC for the treatment of gallstone-related disease in selected uncomplicated patients, for a better cosmetic result but marked with higher costs and trocar site complications. However, further study will be required before widespread use of this technique can be advocated.

14.10 Laparoscopy in Trauma

Laparoscopy in trauma has very concrete but clear indications in the current management of trauma patients. The most important criterion that the patient must comply is to be hemodynamically stable. In such cases, laparoscopy can be a very helpful to rule out a hollow viscous injury as a complement to the diagnostic peritoneal lavage as well as to find out a diaphragmatic injury when the imaging techniques' findings are in doubt [51–53]. In these cases, laparoscopy will permit confirmation of the lesion and treatment, considering that the surgeon has to be skilled enough. In this sense, early series have demonstrated that laparoscopy can reduce negative laparotomies [54], with a decrease in morbidity and mortality rates related to the additional surgical trauma [55].

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Chapter 15

Prevention of Biliary Injuries in Acute Cholecystitis

Jose Schiappa

Already in 1944, the famous British surgeon Grey Turner wrote in the “Lancet”:

CBD (Common Bile Duct) lesions are, almost always, a result of an accident during surgery and, therefore, it can only be attributed to the surgical profession. These lesions cannot be seen as a normal operative risk...

This remark was rather important by recognising, already by then, the fact that errors are an unfortunate part of surgical practice. The problem was that it was not recognised as such then, and it is only nowadays that it is being accepted differently and demanding a different attitude.

1985 was the year Eric Muhe, with a special device of his own, executed the first laparoscopic cholecystectomy, having some personal and professional problems because of this; two years after, in 1987, Philippe Mouret, a French surgeon working mainly in the private sector, initiated the “Second French Revolution”, with true laparoscopic cholecystectomy. Helped

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internationally by Jacques Périssat and by François Dubois, who did a strong presentation and spread of the knowledge of this technique, soon it became “globalised” and a “gold standard” for cholecystectomy.

In the particular case of acute cholecystectomy, its generalised application did not occur the same fast way, but it is also, in the great majority of Hospitals, the technique used for this situation. This pathology is the main reason why this approach is not yet used in all cases. The “classic”, “open” approach is still used worldwide in a not know rate, not only in cases of acute cholecystectomy but also in other cases, depending on availability of equipment and expertise.

Despite having passed now more than 25 years after the introduction of this new technique and this worldwide “explosion” of application, its main problem still exists with strong incidence: iatrogenic lesions of the common bile duct and of the biliary tract are frequent and, probably, underrated as it is difficult to be aware of the real rate. Possibly, many of these complications are not reported for several reasons.

Bile duct injuries (BDI) keep having high incidence, despite all interest given and “calls for attention” which are being done so frequently. Its rates vary between less than 0.2 and 0.8% and even more, in “normal” cholecystectomies. However, if surgery was performed because of acute cholecystitis, these values are higher.

Very few surgeons – still active – exist, who are not performing laparoscopy, for several reasons. This makes it very difficult to “impose” a major change in culture about laparoscopic cholecystectomy, as this is a technique most surgeons believe they master.

Even more so in the difficult cases, leading to “conversion to open”; these, interestingly, show very high rates despite this conversion which would, in theory, give better “view” to the operating field.

*Lesions of the Biliary Tract***Incidence**

In “classic” cholecystectomy	0.2% (Davidoff et al. 1993)
In laparoscopic cholecystectomy	2% (Strasberg et al. 1995)
Laparoscopic × “classic”	>0.5% (McMahon et al. 1995)
Laparoscopic × “classic”	5–10 × (Davidoff et al. 1992)
Diminished in the last years (in general) (Richardson et al. 1996)	0.4–0.8%

Laparoscopy	France (24 300 p.)	0.27%
	USA (77 600 p.)	0.6%
	Portugal (14 455 p.)	0.25%
	Italy (13 718 p.)	0.24%
	Meta-analyses	0.8–1%
Laparotomy	Johns Hopkins (H. Pitt)	0.1–0.2%
	San Diego (A.R. Moossa)	0.5%
	Paul-Brousse (H. Bismuth)	0.2%
	Cornell Univ. (L. Blumgart)	0.2%
	Portug Soc Surg (B.Castelo)	0.55%

Some, more recent figures show different, better values (0.08), coming closer to pre-laparoscopy ones (Halbert et al. 2015).

Several reasons may be culprit of the not-so-good rates, the main one being a lack of in-depth surgical education and training. In many areas, laparoscopic cholecystectomy is considered basic, common laparoscopic surgery, not needing special attention and being considered possible to be practised in the hands of any surgeon, even at their first surgical steps.

The global use of laparoscopic cholecystectomy has changed some of the indications for cholecystectomy itself.

Acute cholecystitis keeps having the same indications, but simple chronic lithiasic cholecystitis has changed: it is now accepted to be done under more liberal approach, and even patients are the ones coming to ask for it because of several dif-

ferent reasons like asymptomatic stones in patients with sickle cell disease, diabetes mellitus disease, receiving immunosuppressive therapy, patients who live far from adequate surgical facilities, asymptomatic gallstones larger than 2 cm, small stones which may give to acute pancreatitis, non-functioning or calcified gallbladder, typhoid carrier with positive bile cultures and trauma of the gallbladder.

All of this is because of the minor aggression caused by the minimally invasive approach; unfortunately, the danger of the iatrogenic lesions needs to be considered.

In fact, acute cholecystitis, usually lithiasic (only up to 18% are alithiasic), can in almost all cases have laparoscopic approach; in alithiasic cases, patients in bad condition or high risk (4–20% of patients with acute cholecystitis in intensive care units) need careful decision regarding approach and surgical decision.

Some of these cases present evolutionary complications of the acute situation: gallbladder gangrene, empyema, cholangitis and perforation with peritonitis, for example. Especially in patients with immunosuppressive situations, all these can lead to complex developments.

In acute cholecystitis, indications like the recommendation for early surgery (within 48–72 h) stay and true “non-operable” patients, with coexistent serious morbidity, can be cases for the consideration of conservative treatment with surgical approach left for later.

In this area, we can also consider clear attention to indications and contraindications: patient’s condition to be submitted (or not) to general anaesthesia, previous surgeries in the upper abdomen and possible implications regarding adhesions; all these can make surgery more difficult and complicated.

Contraindications are, for instance, peritonitis, cirrhosis with portal hypertension, serious acute pancreatitis, cholangitis with septic shock, non-corrected coagulopathy, fistulae and some pregnancy-related situations.

Another important point, related with the safety of patients and prevention of iatrogenic lesions, has to do with timing of

conversion. Many times mentioned, conversion is not a defeat, and the surgeon has to understand it precisely like that. When the surgeon has difficulties both in understanding anatomy and in obtaining the “critical view of safety”, both of which occur frequently in the setting of acute cholecystitis, the surgeon is facing possible indication for conversion, and this shall not be much delayed; another important point is that several series show that even under conversion, many of these cases have a higher incidence of bile duct iatrogenic lesions.

Other indications for conversion are a friable gallbladder, difficult to manipulate, a haemorrhage problematic to an easy control and long time without progression in surgery.

Many of these are situations that show frequently in cases of acute cholecystitis.

Other causes exist for lesions.

A multitude of technical mishaps are cause for complications, most of them being present for ineptitudes from of the surgical team and from some of its technical options.

Bad port positioning, in the case of laparoscopic approach, is an evident cause, but many times these are not properly weighed. By itself or because of the above, bad field exposition and bad illumination are also reasons for higher incidence of complications. Too much smoke or too much blood in the field can hamper visibility to a point of danger.

There is a need to have the ports correctly positioned regarding the possible location of the gallbladder, patient’s BMI and configuration and size of the instruments being used.

In the same line, bad anaesthesia is a well-known factor contributing to bad visualisation of the operating field; patient’s lack of relaxation will “close” the operating field.

Surgeon’s (or team’s) inexperience, as well as surgeon’s (or team’s) tiredness, is very often disregarded or not recognised. In connection with these factors is the lack of knowledge of eventual anomalies; these anomalies are frequent, well known and defined and represent a serious situation which, if not recognised, does not give excuse to the surgeons involved. No surgeon shall under-

take any kind of surgery without minimal theoretical and practical preparation, much more so in the biliary field where important anomalies are so frequent. On another hand, also related with “experience”, surgeon’s overconfidence can be a cause of BDI, by “simplifying” some cases or some technical steps of surgery.

Inappropriate traction of structures, supposedly for “better exposition”, can alter anatomical relations and be a cause for lesions; the same goes for undue use of diathermia, which, unfortunately, we see too often, either by using it too strong or too long.

Let us look at an important point related with these issues: human error. The so-called learning curve, with its associated human error, is so often used nowadays in surgery as an “explanation” for some complications, but would never be accepted in high-technology industries or in some sensitive areas like airlines or military. Many mandatory preparation steps have been designed by these groups to impose rules and protocols in order to minimise those problems; soon we may have to do the same and follow, for instance, a complete checklist procedure before and during each surgical operation; checklists are a controversial point to be discussed under a different approach. Training, in the other hand, is a capital issue, and it is absolutely necessary to keep full attention to this sector.

Human errors can happen, nevertheless, despite all efforts to avoid it; we have to minimise it to the extreme. More often, they are based on technical, training or knowledge failures (ignorance) and by not complying with established rules. These are the ones more “easy” to control. Others are related to a complex and not well-known phenomenon: visual failure or misguidance.

Included amongst processes called “heuristic”, human brain can induce visual errors that, no matter further obvious changes in the visual field, become stable and understood as reality, staying like that for the whole surgery. This means that, under certain circumstances, anatomic structures are percept as different ones in the beginning of the surgery (the most common one being interpreting the CBD as being the cystic duct) and the brain “keeps telling the surgeon” that this first perception is the correct one, leading to the crucial iatrogenic lesion [8].

On a more practical example, this process can also be called “optical illusion” and is well exemplified in all drawings showing this curious effect. When one looks at an image and sees a different image than other people looking at it, or when one is able to see two (or more) different images in a single drawing. Another interesting vision impairment problem has recently been called to attention: the so-called inattention blindness”, when the concentration on the target field makes the surgeon lose track of what is happening around the vision field, even if some serious problem is there (Archie Hughes et al. 2015).

Way and Lawrence have shown, in 2003, that the great majority of iatrogenic lesions of the biliary tract (97%) are caused by errors in visual perception and only 3% because of technical errors.

Another cause, still, maybe the evolution seen in the work of many surgeons, with the idea of reducing the number of ports used; three, or even single port, are being used. Nevertheless, this is a dangerous territory in acute cholecystitis when in many cases there exist already difficult local conditions. Transvaginal approach, used by some, in some countries mainly, is definitely not indicated for acute cholecystitis!

A.R. Moossa very well defined the “three dangers” regarding the risk of having biliary tract lesions during surgery, the first one being particularly important in the setting of acute cholecystitis:

1. Dangerous disease – relating to situations where local surgical conditions convert the “surgical territory” into an area of difficult management because of inflammation, sclerosis, fibrosis or exuberant vascular territory as it happens in cases of late acute cholecystitis or portal hypertension.
2. Dangerous anatomy – in the cases (about 10–15%) where there are anatomic anomalies, it is necessary that the surgeon is well aware of the incidence and of the types of anomalies. While some are of no surgical importance, others can lead to catastrophe.
3. Dangerous surgery – although technical deficiencies can happen without warning, some other can be anticipated and preventive measures shall be applied. Surgery performed by

surgeons or teams without proper physical or training conditions is another scenario leading to disaster.

Other situations shall call surgeon's attention to possible "problematic" cases as it is mentioned later on.

Some other possibilities for lesions, where something can be done, reside in the use, more and more widespread, of some technologies:

One of these technologies, with great impact towards patient's safety, relates with the control of haemorrhage and with the rationale of using instruments of electrosurgery. It is clear that a great number of iatrogenic lesions are connected, in what concerns cause and seriousness, with inadequate use of electrosurgery. Although used quite commonly and frequently, the laparoscopic "hook", utilised by many also as a dissection instrument, besides haemostatic, shall be considered as a dangerous instrument, needing always-intense attention while being used. One of its most dangerous actions is the "jump" it may do, when cutting a structure, under activation, occasions when the surgeon many times loses control of the surgical tool.

It can be considered, in some occasions, its substitution by radio frequency or ultrasonic devices; these have clear advantage in terms of efficiency and safety during haemostasis and can also be used for dissection.

However, even these devices need a strong word of caution. Not being general knowledge – commercial notes mention "without high temperatures" – the temperatures which, in reality, are reached at its extremities, if used continuously for a few seconds, can reach 280°! This temperature only lowers to 30° after about 30 s switched off. This is a very long time during an operation and for a surgeon! Besides there is a clear lateral transmission of this heat, and it is mandatory to have great attention to this.

Dissection shall be done, mainly, by specific instruments and, either these very some ones, either scissors can be used in the phase of cutting and liberation of structures.

Causes for lesions, which need to be very well considered at the time of each surgery, trying to minimise its incidence can be grouped:

Variable issues: patient, pathological condition (acute cholecystitis being one of the most important), surgeon, equipment and environment.

General factors: bad field exposition (bad positioning of trocars), bad light, inappropriate anaesthesia, surgeon's (or team's) inexperience, surgeon's (or team's) tiredness and surgeon's lack of knowledge of eventual anomalies.

Local factors: inflammation, fibrosis, adhesions and urgent operations (all four of which one can face treating surgically acute cholecystitis), bowel distension and reoperations (changed anatomy).

The surgeon: ignorance of possible anomalies, bad vision (smoke, blood), and bad vision angle.

Wrong technique, wrong use of instruments and wrong use of technologies.

Technical failures: inappropriate traction, undue use of diathermia (too strong, too long, etc.) and instrumental mishaps; this adds to instrumental failures, like deficient isolation; "coupling"; broken instrument; point tipped, inappropriate for the task; and even mechanical malfunction.

Talking specifically about patient's possible causes, one must not forget who are the high-risk patients for iatrogenic lesions:

Male patients.

Patients with cirrhosis or liver steatosis.

Obese ones.

Having had previous upper abdominal surgeries.

Those having delayed treatment of acute cholecystitis.

Besides acute cholecystitis, male and obese patients are cases where extra attention is necessary because of high incidence of problems.

Another, very much stressed is the so-called learning curve.

But, although this "learning curve" can be responsible for many things and has to be eliminated or minimised as much as possible, it has no defined causal relation to BDI; the "learning curve" for

laparoscopic cholecystectomy goes well beyond 50 cases, and, although operating time keeps lowering till 200 cases, improvement in cognitive skills to deal with difficult cases continues [5].

It has also been shown that the risk goes beyond “first cases” as demonstrated in the following series from the same institution: first 1284 cases – 0.58% BDI/following 1143 cases – 0.50% BDI [6].

An enquiry done to 1500 surgeons reports that about 30% of BDI occur after the first 200 cases [7].

We can only conclude that surgeon’s experience does not minimise the risk.

This persistence of high rates of BDI after the initial training curve shows that there is a difference in these; it is considered that there is a difference between “experienced” surgeons and “experts”; “experts” are surgeons with “consistent better outcomes” (namely, BDI rates consistent and very low or close to zero).

The choice of wrong timing to operate acute cholecystitis is a common cause for surgical difficulties and, eventually, surgical accidents. There is evidence that performing cholecystectomy more than 5 or 6 days after the onset of the acute inflammation will make surgery much more difficult, facing a great number of serious inflammatory adhesions, causing much more bleeding than usual and making difficult to recognise proper anatomy and surgical landmarks. Some even place this time limit at 72 h.

Summarising causes is a task Strasberg took in hands some time ago.

Classification of causes (Steven Strasberg JACS, 1995):

Wrong perception (confusion) of cystic duct and main biliary ducts

Confusing:

Cystic duct with common hepatic duct

Cystic duct with anomalous right hepatic duct

Cystic duct with common bile duct

Technical causes:

Badly performed occlusion of the cystic duct

Too deep plan of liver dissection

Inappropriate use of “energy”

“Tenting” (or “camel hump”) of the cystic duct

Inappropriate placement of clips for haemostasis

Direct lesion exploring CBD

What shall not be forgotten, either, is that 87% of biliary tract lesions happen when dissecting Calot’s triangle, generally by failure to identify the main bile duct (J. F. Gigot, personal communication, 2005).

As general and basic principles to reduce the incidence, we can recommend:

Use of 30° optic

Surgical access adapted to morphology

Use of clear and proper methods to retract and expose the surgical field giving good exposure of the hepato-duodenal space

Dissection of Calot’s triangle starting close to the gallbladder, with appropriate dissection

Finding the cystic duct by starting dissection at Calot’s triangle

Tracing the cystic duct on an uninterrupted line into the base of the gallbladder

Trying to obtain the “critical view of safety”

Unequivocal identification of cystic duct and artery before they are divided

Clearing the medial wall of infundibulum

There are factors that may suggest that the structure being dissected is the CBD instead of the cystic duct (> \emptyset duct, course behind duodenum, unexpected duct, large artery, etc.)

Clamping of the pedicle if big haemorrhage
If necessary, direct cholecystectomy
Great care with the use of electro-surgery
Selective cholangiography

Some other “principles” have been widely mentioned as rules to prevent lesions. However, some may prove wrong or, at least, dangerous to implement:

“Clearly identify the junction of the cystic duct and CBD”. Too much dissection work in here can lead to problems such as devascularisation with late strictures.

“Use routine IO cholangiography”. Although useful in many situations (confusing anatomy, difficult dissection, anomaly suspected), it brings another possible problem: there is a danger of misinterpretation, giving false sense of security. The “tricks” the brain does before (“heuristic” processes) can also happen with cholangiography. In addition, many lesions happen after cholangiography!

Perioperative cholangiography led to the development of many devices trying to make it “easier”. In truth, the great majority of such devices are not necessary; it is enough to use a simple catheter for adequate diagnostic examination. Nevertheless, although its use routinely has not been proven as definitely beneficial, it is important that all surgeons performing laparoscopic cholecystectomy are trained and know how to perform it.

There are alternatives to intraoperative cholangiography (IOC), like ultrasonography or fluorescent cholangiography. Although in this specific setting (prevention of lesions) US does not seem to be interesting, the new modality, fluorescent cholangiography, seems to have a future, mainly by giving important views and by eliminating the “aggressiveness” and “invasion” of classic cholangiography.

One question is important: can intraoperative cholangiography (IOC) contribute for prevention?

Looking at the rationale of both attitudes (routine or selective IOC), we can realise that routine cholangiography gives some

safety guard by providing bile tree mapping and helping to clarify “unclear” anatomy allowing, possibly, less iatrogenic injuries.

Defenders of selective cholangiography claim that for safety, it does not need to be systematic, and one can rely on surgeon’s criteria to know when there is “unclear” anatomy and to be realistic knowing that absolutely correct interpretation is mandatory!

On the other hand, it can give a false sense of security by providing inadequate images, and it can be “dangerous” by the mentioned misinterpretation (“heuristic” processes) of images (both in film and in dynamic views).

Cholangiography is not a substitute for meticulous dissection, and injuries to CBD can occur before cystic duct dissection reaches the point at which cholangiography can be performed (Fried et al. 2002)

Another issue is that IOC has complications like duct tearing; duct injury, clipping the wrong structure before complete definition; pancreatitis; and infection or cholangitis

All this also needs another perspective:

Are lesions from laparoscopic surgery more serious?

The fact is, in laparoscopic surgery, some lesions started to show, which did not happen before; these are lesions caused by total destruction of the CBD, for instance, because of extensive or wrong use of electrosurgery, and the ones which imply removal, erroneously, of long lengths of the biliary tract. There has been implication, by some authors, that some more recent surgical series, regarding treatment of CDI, have a slightly inferior success rate than older ones; this might be due to the existence of worse lesions in the laparoscopic era. As a matter of fact, about 30% of lesions, nowadays, are extensive burn lesions; resection (or excision) of extensive length of biliary tract and lesions often very close to the hilum happens a lot, as well as coexistence with biliary fistulae with consequent increase of inflammation because of bile action and the fact that in many other cases we see bile ducts, with small calibre, increase the seriousness of the injuries and the difficulty on repairing it.

Alternatively, some other lesions are also more benign, like tangential lesions, clip falling or puncture lesions, allowing treatment by minimally aggressive endoscopic methods. Injuries can be graded, from less to more severe, as:

1. Puncture
2. Partial laceration
3. Complete section
4. Obstructing clip
5. Enlarged section (tissue removal)
6. Thermal lesion
7. Thermal Necrosis

Some of the less severe can be approached and treated only by endoscopic methods like placement of a prosthesis after eventual balloon dilatation. This can be very effective and fully resolve the situation in minor lesions, not so in major ones which, in general, need extensive major surgery. Surgeons performing this corrective surgery need to have proper experience (repairs attempted by the primary surgeon have a dismal success rate!) and are always challenged with strong issues:

1. Injuries involving the confluence
2. High stenosis with previous repair attempts
3. Association with vascular injury (up to 30% in some cases)
4. Association with portal hypertension or biliary cirrhosis

Association with vascular injury is frequent and happens, in general, when, during surgery, there are attempts to control haemorrhage when the clip in the cystic artery falls and the artery, bleeding, retracts behind the CBD; in there with clipping or forceps attempts to control haemorrhage, the main artery is often caught and has lesions which can be serious.

Another important point to stress is in evaluating results one shall not forget that "... only 10 year or longer follow-up can show whether treatment results, surgical or other, are good ..." (H. Bismuth – personal communication).

Finally, which suggestions and recommendations on measures to minimise the problem can be given besides the notes previously mentioned?

Mainly two: following guidelines and improving training and education

Some guidelines are well established and have good grades of recommendation: optimal exposure to reach the critical view of safety is highly recommended (grade of recommendation (GoR B)); although this can be achieved with the 0°, 30° or 45° optics, the CBD is more difficult to see with the 0° because it lies parallel to the scope. Rotation of angled scopes provides different visualisations of the surgical field.

This was already proposed long ago [12].

Inability to reach critical view of safety and/or to identify the source and safely control bleeding are indications for conversion (grade of recommendation (GoR A)).

It is recommended a supervised structured training starting with skills courses (GoR B).

Clearly, although BDI does occur in the hands of expert surgeons, inadequate experience is a risk factor.

New educational methods shall be put in practice and enough experience already exists to understand what shall be done.

Besides known exercises and properly structured courses, some more recent ideas have been understood. Expert surgeons can be identified, and their techniques have to be put in practice in teaching methods; they must share their practical preventing measures. This may bring learners faster to the level of “experts”.

Experts have more knowledge and, consequently, superior performance although some of the reasons for this cannot be clearly understood; the most important is called “hidden knowledge”. This has three types:

Informal knowledge – from experience but unwritten – does not exist in textbooks. An example is something many have experienced: the expert tells the resident

to stop and look “here” or “there”. Looking, one finds that there was a reason for that. But all the experts can say is “it just didn’t look right”.

Impressionistic knowledge – experts are always, even unconsciously, looking back into “past experiences”. They have some “impressions” of some situations, with a “feeling of possible danger” when in presence of certain signs, non-described.

Self-regulation knowledge – deep knowledge about themselves and about how they act.

Other evident but not so often followed principles – these can be generalised for any surgical approach – are the constant use of the highest human and surgical good sense as well as keeping an adequate knowledge of the anatomy and of anomalies. Last, but by no means least, whenever in doubt, stop and re-evaluate; one will be surprised by the number of times this line of acting will change options and attitudes. And keep always a humble position; be aware of the situations and of the capacities, human and technical, existing. Every time it feels advisable, do not hesitate in asking for help.

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Chapter 16

Adhesive Small Bowel Obstruction (ASBO)

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16.1 Introduction

About 2 % of total patients with abdominal pain admitted in the emergency department have a diagnosis of adhesive small bowel obstruction (ASBO) [1–5].

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The clinical presentation of SBO is some combination of signs and symptoms with different grades of intensity which includes colicky abdominal pain and distension, diffuse tenderness, nausea, vomiting, and progressive failure to pass stool and flatus, associated with specific radiological findings of air/fluid levels.

Abdominal adhesions are responsible for the majority of ASBO so it is mandatory to discuss about this cause.

Other causes include hernia (the most frequent cause in a virgin abdomen), cancer, inflammatory bowel disease, intussusception, radiation, endometriosis, infections, and foreign bodies (include gallstone ileus).

The diagnosis is primarily related to the past medical history of a patient, his physical examination, the findings on the radiological studies, and the consideration of the likely causes.

Definitive treatment is related to the cause and degree of obstruction (complete vs partial), duration of symptoms, and failure to medical therapy.

16.2 Clinical Presentation

ASBO results like the combination of different clinical signs and symptoms. The degree of intensity in clinical presentation gives an indication about the severity and the level of obstruction, sometimes the cause, even related to the past history of the patient.

Therefore, the degree of abdominal pain, tenderness and distension, amount and feature of vomiting, or nasogastric tube outputs (bilious vs feculent) could address to the level of the obstruction.

Abdominal plain radiograph could help us to better identify it.

Recent or past medical events are really important to detect the cause. Prior abdominal and pelvic surgery, a history of malignant tumors, and inflammatory or infectious abdominal ill are suggestive of the cause.

Because hernias represent the most frequent cause of obstruction in nonoperated patients, a meticulous inspection of all possible orifices potentially the site of hernias is mandatory in every patient with clinical feature of intestinal obstruction.

It is well known that passage of stools is not against the diagnosis of ASBO, because the portion of the bowel distant to obstacle can pass feces even if the obstruction is complete.

Partial obstruction is a less severe clinical presentation of ASBO.

16.3 Laboratory Findings

Any laboratory finding alone is an independent predictive factor for severity of ASBO.

Often patients who arrive in the ED present a volume deficit because of vomiting, ascites, and the so-called third spacing. Resulting hemoconcentration gives elevated values of hematocrit and hemoglobin levels. Either white blood cell (WBC) count could be spuriously elevated, but leukocytosis greater than 20,000/mL should prompt concern for bowel compromise or perforation in cases of ASBO, but any operation should be initiated or should be delayed only on the basis of WBC count alone.

Metabolic alkalosis and acidosis are common dysfunction in ASBO, due to loss of electrolytes and renal reabsorption. Hypokalemia is the most common electrolyte abnormality, and it should be replaced by isotonic solution with additional potassium. Even blood urea nitrogen and creatinine levels are

commonly increased, due to low renal perfusion, but a replacement of intravenous volume should improve the values.

Resulting to global hypoperfusion, lactate level may be elevated, even considering that segmental bowel infarction or perforation is not necessarily related to an elevated serum lactate level.

16.4 Radiologic Diagnosis

The most useful radiologic test in ASBO is abdominal series of the abdomen, consisting in upright chest and upright abdominal radiograph. This test allows to evaluate the presence of air/fluid levels, gas in large bowel, and free air in the peritoneum. If upright radiographs are not possible for the patient, a decubitus cross table lateral radiograph of the abdomen may be substituted to look for free air. Abnormal gastric distension with air/fluid level associated with poor air in the remaining abdomen is an indication of high level of obstruction. Contrarily a full air-/fluid-level radiogram is more favorable to a low level of obstruction. Usually in cases of complete ASBO, gas in the large colon and rectum is absent, contrarily to cases of ileus. Studies demonstrate that the majority (about 80%) of cases of radiologic complete ASBO implicate a surgical treatment, against a little minority of cases (10–15%) operated for partial obstruction.

In some cases pneumatosis in the bowel wall or portal venous gas may be seen on plain radiograph, and it's mandatory to urgent surgery.

Oral administration of 100 mL of Gastrografin followed by plain abdominal radiographs could help diagnosis. If the contrast arrives at the cecum in about 24 h, the obstruction is partial and is presumable that it will resolve without operation.

The use of barium for the risk of a non-demonstrated perforation of the bowel is not recommended.

Furthermore, it has been suggested that the oral administration of a small amount of Gastrografin can lead to the resolution of ASBO [2–10].

Ultrasound may yield information in cases of ASBO. Thanks to a noninvasive procedure, the presence of fluid-filled loops with or without peristalsis can be established. Gallstones and air in the gallbladder suggest gallstone ileus. A new ascites is an ominous sign of prognosis and a positive factor for operation need.

CT scan is the best investigation in ASBO and it has supplanted abdominal series. A double-contrast CT scan is the imaging procedure of choice because of its high sensitivity and specificity in the diagnosis of ischemia and degree of obstruction (partial vs complete) [3–12]. It can be helpful to determine the etiology of obstruction, such as internal or abdominal wall hernias, intussusception, tumor or mass, Crohn's disease, metastatic cancer, ischemia, and sign of intestinal compromise such as pneumatosis intestinalis or pneumoperitoneum. In cases of ASBO in patients who underwent laparoscopic gastric bypass, an early CT scan is indicated because of the risk of internal hernia.

The presence of a transition point is not a prognostic factor for failure of nonoperative management [3–14].

In the study of Duda and colleagues, the presence of “whirl sign,” defined like the swirl of mesenteric soft tissue and fat attenuation with adjacent loops of the bowel surrounding rotated intestinal vessels, represents a positive factor to predict the need of an operation.

Another useful CT scan finding is small bowel feces sign, which associated with other clinical factors could represent a predictive factor for intervention [5–20].

It is established by a multivariate analysis that the following factors predict the need for resection: free peritoneal fluid at CT

scan (more than 500 mL), reduction of CT bowel enhancement, abdominal pain persisting for 4 or more days, abdominal tenderness with guarding, WBC count $>10,000/\text{mL}$, and C-reactive protein $>75 \text{ mg/L}$. In the analysis all the patients with four or more variables required resection.

In another multivariate analysis conducted to evaluate factors and predictors of the need of operation in ASBO, it was found that vomiting, mesenteric edema on CT scan, and the lack of the small bowel feces sign are independent factors [20–30].

16.5 Nonoperative Management

16.5.1 Patients' Selection

For patients presenting with acute adhesive small bowel obstruction [AASBO] without signs of strangulation, peritonitis, or severe intestinal impairment, there is good evidence to support nonoperative management [NOM].

Free intraperitoneal fluid, mesenteric edema, lack of the “small bowel feces sign” at CT scan, history of vomiting, severe abdominal pain [VAS >4], abdominal guarding, raised white cell count [WCC], and devascularized bowel at CT scan predict the need for emergent laparotomy [31].

Moreover, patients with repeated AASBO episodes, many prior laparotomies for adhesions, and prolonged conservative treatment should be cautiously selected to find out only those who may benefit of early surgical interventions [31].

At present, there is no consensus about when conservative treatment should be considered unsuccessful and the patient should undergo surgery: in fact the use of surgery to solve AASBO is controversial, as surgery induces the formation of new adhesions [31].

Level I data have shown that NOM can be successful in up to 90% of patients without peritonitis [32].

As a counterpart, a delay in operation for AASBO places patients at higher risk for bowel resection. A retrospective analysis showed that in patients with a ≤ 24 -h wait time until surgery, only 12% experienced bowel resection, and in patients with a ≥ 24 -h wait time until surgery, 29% required bowel resection [33].

Schraufnagel et al. showed that in their huge patient cohort complications, resection, prolonged length of stay, and death rates were higher in patients admitted for AASBO and operated on after a time period of ≥ 4 days [34].

The World Society of Emergency Surgery [WSES] 2013 guidelines stated that NOM in the absence of signs of strangulation or peritonitis can be prolonged up to 72 h. After 72 h of NOM without resolution, surgery is recommended [31].

There are no objective criteria that identify those patients who are likely to respond to conservative treatment. Less clear, in fact, is the way to predict between progression to strangulation and resolution of AASBO. Some authors suggested strong predictors of NOM failure: the presence of ascites, complete AASBO [no evidence of air within the large bowel], increased serum creatine phosphokinase, and ≥ 500 ml from nasogastric tube on the third NOM day [31].

However, at any time, if there is an onset of signs of strangulation, peritonitis, or severe intestinal impairment, NOM should be discontinued, and surgery is recommended.

It's really difficult to predict the risk of operation among those patients with AASBO initially undergone to NOM [31].

16.5.2 Tube Decompression, WSCA, and Other Treatments

Randomized clinical trials showed that there are no differences between the use of nasogastric tubes and the use of long-tube decompression [35].

In any case, early tube decompression is beneficial in the initial management, in addition to required attempts of fluid resuscitation and electrolyte imbalance correction. For challenging cases of AASBO, the long tube should be placed as soon as possible, more advisable by endoscopy, rather than by fluoroscopic guide [36].

Several studies investigated the diagnostic-therapeutical role of water-soluble contrast agent [WSCA] [37]. Gastrografin® is the most commonly utilized contrast medium. It is a mixture of sodium diatrizoate and meglumine diatrizoate. Its osmolarity is 2150 mOsm/L. It activates movement of water into the small bowel lumen. Gastrografin® also decreases edema of the small bowel wall, and it may also enhance smooth muscle contractile activity that can generate effective peristalsis and overcome the obstruction [38].

The administration of WSCA showed to be effective in several randomized studies and meta-analysis [30]. Three recent meta-analyses showed no advantages in waiting longer than 8 h after the administration of WSCA [30] and demonstrated that the presence of contrast in the colon within 4–24 h is predictive of AASBO resolution. Moreover, for patients undergoing nonoperative management, water-soluble contrast decreased the need for surgery and reduced the length of hospital stay [39, 40].

Oral therapy with magnesium oxide, *L. acidophilus*, and simethicone may be considered to help the resolution of NOM in partial AASBO with positive results in shortening the hospital stay [41].

Lastly hyperbaric oxygen therapy may be an option in the management of high anesthesiologic risk patients for whom surgery should be avoided [42].

No agreement exists about the possibility to predict the recurrence risk. Factors associated with a higher risk of recurrence are age <40 years, matted adhesion, and postoperative surgical complications [43]. Compared to traditionally conservatively treated patients, Gastrografin® use doesn't affect either the AASBO recurrence rates or recurrences needing surgery [29].

16.6 Surgery

16.6.1 *Open Surgery*

Until recently open surgery has been the preferred method for the surgical treatment of AASBO [in the case of suspected strangulation or after failed conservative management], and laparoscopy has been suggested only in highly selected group of patients, preferably in the case of the first episode of ASBO and/or anticipated single-band adhesion [i.e., small bowel obstruction after appendectomy or hysterectomy] using an open-access technique and the left upper quadrant for entry [31].

More recently, the use of laparoscopy is gaining widespread acceptance and is becoming the preferred choice in centers with specific expertise.

A meta-analysis from Ming-Zhe Li et al. found that there was no statistically significant difference between open versus laparoscopic adhesiolysis in the number of intraoperative bowel injuries, wound infections, or overall mortality. Conversely there was a statistically significant difference in the incidence of overall and pulmonary complication rate and a considerable reduction of prolonged ileus in the laparoscopic group compared with the open group. The authors conclude that laparoscopic approach is safer than the open procedure but only in the hands of experienced laparoscopic surgeons and in selected patients [44].

However, no randomized controlled trial comparing open to laparoscopic adhesiolysis exists to date, and both the precise indications and specific outcomes of laparoscopic adhesiolysis for acute ASBO remain poorly understood. The only RCT aiming to provide level Ib evidence to assess the use of laparoscopy in the treatment of adhesive small bowel obstruction is currently ongoing, having as a primary endpoint the length of postoperative hospital stay and as secondary and

tertiary endpoints the passage of stools, commencement of enteral nutrition, 30-day mortality, complications, postoperative pain, length of sick leave, rate of ventral hernia, and the recurrence of small bowel obstruction during long-term follow-up [45].

16.6.2 Laparoscopy

Laparoscopic adhesiolysis for small bowel obstruction has a number of potential advantages: less postoperative pain, faster return of intestinal function, shorter hospital stay, reduced recovery time, allowing an earlier return to full activity, fewer wound complications, and decreased postoperative adhesion formation [46].

In a recent large population-based propensity score-matched analysis involving 6762 patients [47], laparoscopic treatment of AASBO was associated with lower rates of postoperative morbidity, including SSI, intraoperative transfusion, and overall lower resource use compared with laparotomy as well as shorter hospital stay. Laparoscopic treatment of surgical AASBO is not associated with significant difference in operative time, rates of reoperation within 30 days, or mortality.

Further recent reports confirmed that laparoscopic surgical management of acute ASBO is associated with quicker GI recovery, shorter LOS, and reduced overall complications compared to open surgery, without significant differences in operative times [48]. Furthermore, following the exclusion of bowel resections, secondary outcomes continued to favor laparoscopy.

Although laparoscopic adhesiolysis requires a specific skill set and may not be appropriate in all patients, the laparoscopic approach demonstrates a clear benefit in 30-day morbidity and mortality even after controlling for preoperative patient characteristics [lower major complication and incisional complication

rate as well as shorter postoperative LOS and shorter mean operative times]. Given these findings in more than 9000 patients and consistent rates of ASBO requiring surgical intervention in the United States, increasing the use of laparoscopy could be a feasible way to decrease costs and improve outcomes in this population [49].

Patient selection is still a controversial issue. From a recent consensus conference [50], a panel of experts recommended that the only absolute exclusion criteria for laparoscopic adhesiolysis in ASBO are those related to pneumoperitoneum [e.g., hemodynamic instability or cardiopulmonary impairment]; all other contraindications are relative and should be judged on a case-to-case basis, depending on the laparoscopic skills of the surgeon.

Nonetheless, it is now well known that the immune response correlates with inflammatory markers associated with injury severity and, as a consequence, the magnitude of surgical interventions may influence the clinical outcomes through the production of molecular factors, ultimately inducing systemic inflammatory response, and the beneficial effect of minimally invasive surgeries and of avoiding laparotomy is even more relevant in the frail patients [51].

Laparoscopic adhesiolysis is technically challenging, given the bowel distension and the risk of iatrogenic injuries if the small bowel is not appropriately handled. Key technical steps are to avoid grasping the distended loops and handling only the mesentery or the distal collapsed bowel. It is also mandatory to fully explore the small bowel starting from the cecum and running the small bowel distal to proximal until the transition point is found and the band/transition point identified. After the release of the band, the passage into distal bowel is restored, and the strangulation mark on the bowel wall is visible and should be carefully inspected.

As a precaution and in the absence of advanced laparoscopic skills, a low threshold for open conversion should be maintained when extensive and matted adhesions are found [52].

Reported predictive factors for a successful laparoscopic adhesiolysis are number of previous laparotomies ≤ 2 , non-median previous laparotomy, appendectomy as previous surgical treatment causing adhesions, unique band adhesion as pathogenetic mechanism of small bowel obstruction, early laparoscopic management within 24 h from the onset of symptoms, no signs of peritonitis on physical examination, and experience of the surgeon [53].

Because of the consistent risks of inadvertent enterotomies and the subsequent significant morbidity, particularly in elderly patients and those with multiple [three or more] previous laparotomies, the lysis should be limited to the adhesions causing the mechanical obstruction or strangulation or those located at the transition point area: some authors have attempted to design a preoperative nomogram and a score to predict risk of bowel injury during adhesiolysis, and they found that the number of previous laparotomies, anatomical site of the operation, the presence of bowel fistula, and laparotomy via a preexisting median scar were independent predictors of bowel injury [54, 55].

16.7 Prevention

16.7.1 *Surgical Technique*

Small bowel obstruction has been the driver of research in adhesion prevention measures, barriers, and agents. Recent data from cohort studies and systematic reviews point at major morbidity and socioeconomic burden from adhesiolysis at reoperation, which have broadened the focus of adhesion prevention [56]. Applying adhesion barriers in two-stage liver surgery and cesarean section, to reduce the incidence of adhesions and associated adhesiolysis-related complications, are examples of the

change in paradigm that reducing the incidence of adhesions is clinically more meaningful than only aiming at preventing adhesive small bowel obstruction [57]. Increasing the number of patients without any peritoneal adhesion should be the general aim of adhesion prevention.

“Good” surgical technique and anti-adhesion barriers are the main current concepts of adhesion prevention. From a recent systematic review and meta-analysis on the impact of different surgical techniques on adhesion formation, it was concluded that laparoscopy and not closing the peritoneum lower the incidence of adhesions [1].

However, the burden of adhesions in laparoscopy is still significant most likely due to the necessity to make specimen extraction incisions in addition to trocar incisions and the unavoidable peritoneal trauma by surgical dissection and the use of CO2 pneumoperitoneum [intraoperative pressure and desiccation]. Reduced port laparoscopy and specimen extraction via natural orifices may theoretically further reduce peritoneal incision-related adhesion formation [58].

16.7.2 Anti-adhesion Barriers

Since all abdominal surgeries involve peritoneal trauma and potential healing with adhesion formation, additional measures are needed to reduce the incidence of adhesions and related clinical manifestations. These measures consist of systemic pharmacological agents, intraperitoneal pharmaceuticals, or adhesion barriers [59]. Most clinical experience is with intraperitoneal adhesion barriers, applied at the end of surgery with the aim to separate injured peritoneal and serosal surfaces until complete adhesion-free healing has occurred. Efficacy of anti-adhesion barriers in open surgery has been well established for reducing the incidence of adhesion formation [60]. For one type of barrier [hyaluronate-carboxymethylcellulose, HA-CMC,

Seprafilm®, Sanofi, Paris, France], the reduction of incidence for adhesive small bowel obstruction after colorectal surgery has also been established [RR 0.49, 95% CI 0.28–0.88] without patient harm [60, 61]. Oxidized regenerated cellulose [Interceed®, Ethicon, West Somerville, NJ, USA] reduces the incidence of adhesion formation following fertility surgery [RR 0.51, 95% CI 0.31–0.86], but the impact on small bowel obstruction after gynecological surgery has not been studied [60, 62]. Drawback of both products is the difficulty to use in laparoscopic surgery underlining the need to develop gel, spray, or fluid barriers that are easy to apply via a trocar.

In the POPA study, authors randomized 91 patients to have 2000 cc of icodextrin 4% and 90 to have the traditional treatment. The authors noted no significant difference in the incidence of small bowel leakage or anastomotic breakdown; operative times, blood losses, incidence of small bowel resections, return of bowel function, LOS, early and late morbidity, and mortality were comparable. After a mean follow-up of 41.4 months, there have been two cases of AASBO recurrence in the icodextrin group and ten cases in the control group [$p < 0.05$] [63].

Consistent safety and efficacy evidence has not led to routine application of barriers in open or laparoscopic surgery. Reasons might be the lack of awareness, the question if the “effect size” is large enough for routine application, or the belief that adhesion formation even may benefit the patients, e.g., reinforcing intestinal anastomosis or walling off peritoneal infection. However, most used argument against routine use is the doubt regarding cost-effectiveness of adhesion barriers. The direct hospital costs in the United States in 2005 of adhesive small bowel obstruction alone were estimated at \$3.45 billion. Costs associated with the treatment of an acute ASBO are estimated to be \$3000 per episode with conservative treatment and \$9000 with operative treatment. The additional costs incurred by operative treatment are partially due to complications of adhe-

siolysis. The incidence of bowel injuries during adhesiolysis for ASBO is estimated to be between 6% and 20%. Inadvertent enterotomy due to adhesiolysis in elective surgery is associated with a mean increase in costs of \$38,000 [59, 64].

In a model, counted for in hospital costs and savings resulting from adhesive ASBO based on UK price data from 2007, Wilson showed that a low-priced barrier at about \$160 with 25% efficacy in preventing ASBO would result in healthcare savings. Another concept with a \$360 price barrier would result in a net investment on the long term unless a higher efficacy of 60% could be achieved. In this model treatment costs of small bowel obstruction were substantially lower than more recent cost calculations. Recent direct healthcare costs associated with treatment of major types of adhesion-related complications [small bowel obstruction, adhesiolysis complications, and secondary female infertility] within the first 5 years after surgery are \$2350 following open surgery and \$970 after laparoscopy. Application of an anti-adhesion barrier could save between \$678 and \$1030 following open surgery and between \$268 and \$413 following laparoscopic surgery on the direct healthcare costs related to treatment of adhesion-related complications [data not published]. Benefits from reduction in ASBO were \$103 in open surgery and \$32 in laparoscopic surgery, using a high [\$360]-priced product and only taken into account reoperations for adhesive small bowel obstruction. From this cost modeling, it seems that even routine use of anti-adhesion barriers is cost-effective in both open and laparoscopic surgery [65, 66].

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Chapter 17

Large Bowel Obstruction

Chasen Croft, Doug Kwazneski, and Frederick Moore

17.1 Introduction

Acute mechanical bowel obstruction is a common surgical emergency frequently encountered by the acute care surgeon. While nearly 80% of mechanical bowel obstructions occur in the small bowel, approximately 20% present in the large bowel [1, 2]. Because of the potential life-threatening complications, timely recognition and management are crucial.

Large bowel obstruction (LBO) classically describes any physical or mechanical obstruction to the flow of intraluminal contents through the colon or rectum. It is well recognized that not all obstructions are completely mechanical; some can be functional, and this should be recognized in the differential diagnosis. LBOs are an important subject for acute care surgeons, as they have been classically recognized as a surgical emergency [2]. Due to the varied etiology and symptoms on

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presentation, it is imperative for the treating physician to recognize the symptoms, formulate a differential diagnosis, perform correct diagnostic testing, and institute prompt treatment.

17.2 Etiology

Large bowel obstruction may be caused by a variety of etiologies. In general, the causes of large bowel obstruction may be categorized as either mechanical or physiological. Mechanical large bowel obstruction refers to the physical obstruction to the flow of feces through the colon or rectum. This may be due to luminal, mural, or extramural obstruction of the bowel. As a physiological response to the obstruction, intestinal contractility increases in an attempt to relieve the obstruction. The majority of mechanical LBOs are due to neoplasms, with colorectal cancers accounting for nearly 50% of all LBOs. Incidentally, 10–30% of colorectal cancers present with LBO as the chief presenting symptom. Other common causes of mechanical LBO include colonic volvulus and diverticulitis, which combined account for roughly 25% of LBOs. Unlike mechanical small bowel obstruction, adhesive bowel disease rarely causes LBO. Less common causes of mechanical LBO include ischemic stricture, intussusception, fecal impaction, hernias, and foreign objects. Physiological causes of LBO are conditions which mimic mechanical LBO; however, no physical obstruction exists. Causes include acute colonic pseudo-obstruction (ACPO or Ogilvie's syndrome), colonic ischemia, ileus, toxic megacolon, and inflammatory etiologies such as ulcerative colitis and Crohn's disease (Table 17.1). Although most LBOs occur in the elderly, in general, no age or sex differences have been identified for LBO; rather, incidence is affected by the underlying etiology.

Table 17.1 Causes of acute colonic pseudo-obstruction

Common (>95 %)	Colorectal cancer (60–80%)
	Volvulus (11–15 %)
	Sigmoid
	Cecum
	Transverse colon
	Diverticulitis
	Acute colonic pseudo-obstruction (Ogilvie's syndrome)
Uncommon (<5 %)	Toxic megacolon
	Hernia
	Intussusception
	Inflammatory/ischemic bowel disease
	Extrinsic compression from abscess or mass
	Fecal impaction
	Foreign body

17.3 Clinical Presentation of Acute Large Bowel Obstruction

The signs and symptoms of large bowel obstruction vary greatly and depend on the cause, chronicity, and location of the obstruction. Regardless of the cause of obstruction, patients with LBO commonly present with complaints of generalized colicky abdominal pain, progressive abdominal distention, and failure to pass stool or flatus. As the disease process evolves, constipation may progress to obstipation. Unlike small bowel obstruction, nausea and vomiting are not common presenting symptoms. If they do occur, it is often far later in the course of the disease. Symptoms may present acutely, as in the case of colonic volvulus and intussusception, or may be chronic in cases of more indolent etiologies, such as colorectal cancers.

In large bowel obstruction, the colon becomes distended with air, fluid, and stool proximal to the site of blockage, leading to increased intracolonic pressure. As this pressure increases, the

intramural pressure within the colonic wall may exceed the capillary pressure, leading to mucosal ischemia. If venous occlusion occurs, localized bowel wall edema and transudation of fluid ensues [3]. This may occur in conditions which cause a twist in the mesentery, such as colonic volvulus, or in conditions causing direct pressure on the mesenteric vessels. Following Laplace's law, the cecum is more susceptible to vascular compromise and perforation due to the greater wall tension [4]. As such, patients frequently complain of right lower quadrant pain, even in the presence of left-sided lesions.

A closed loop obstruction occurs when both the proximal and distal portions of the bowel are occluded. This may occur in colonic volvulus or if the ileocecal valve is competent, which occurs in about 75% of patients, preventing decompression of colonic contents into the distal small bowel [5, 6]. When present, a closed loop obstruction increases the risk of ischemia and perforation.

Patients with untreated LBO may have signs of dehydration, septicemia, a distended abdomen, and a palpable mass. Any signs of sepsis, such as high fever, persistent tachycardia despite resuscitation, shock, or peritonitis, should raise the suspicion for an acute surgical process and most often requires emergent surgical intervention [7, 8].

17.4 Diagnosis

As with any surgical disease, a thorough history and physical exam should be undertaken, with special attention focused on the abdominal and rectal exams. The symptomatology of LBO varies widely, and as such, chronicity of symptoms plays an important role in narrowing the differential diagnosis. The etiology of the LBO may be suggested by the signs and symptoms at the time of presentation. Patients who present with

mechanical causes, such as volvulus, often describe a specific beginning of symptoms, which can be delineated on history. In contrast, malignant obstructions often present after a protracted course, with symptoms of partial obstruction which spontaneously resolve prior to presentation with complete obstruction. A review of symptoms should focus on timing of symptoms, recent alterations in bowel habits, changes in stool caliber, presence of melena or hematochezia, changes in weight, and abdominal pain or pain with defecation. Pertinent medical history should include whether the patient has had constipation or diarrhea, history of chronic laxative or narcotic use, and previous surgical history. A family history of colorectal cancers should be noted.

Important findings on physical exam include abdominal distention, tympany, palpable abdominal mass, and symptoms of peritonitis, such as abdominal rigidity, rebound tenderness, and guarding. A digital rectal exam should be performed assessing for rectal mass, impacted stool in the rectal vault, and blood.

Initial blood work should be obtained to include a complete blood count with differential (CBC) and a basic metabolic panel (BMP). A marked leukocytosis suggests possible ischemia or perforation. A basic metabolic panel (BMP) helps determine the degree of dehydration and aids in the correction of electrolyte and acid-base abnormalities. Lactic acid may be helpful in identifying colonic ischemia; however, elevated lactate is a late sign and may be falsely normal if venous obstruction prevents entry of lactic acid into the systemic circulation. If malignancy is suspected, a carcinoembryonic antigen (CEA) level should be obtained.

Plain abdominal radiography is usually the first diagnostic imaging performed in patients suspected of having LBO [5, 7, 8]. Plain films have the advantage of being quick, inexpensive, and may be done as a portable series if needed. The examination should include supine and upright imaging to detect the presence of pneumoperitoneum and exclude small bowel

obstruction. However, an incompetent ileocecal valve will allow decompression of the LBO into the distal small bowel, and the resultant small bowel distention may mimic a distal small bowel obstruction [8]. Although plain radiographs may confirm a clinical diagnosis of LBO, they often cannot accurately determine the site or cause of the obstruction [9]. The reported sensitivity for the detection of LBO is similar to that for the detection of small bowel obstruction; however, the specificity is considerably lower [5, 10, 11]. The presence of intraperitoneal free air, a cecal diameter greater than 12 cm indicating impending perforation, or the diagnosis of large bowel volvulus often warrants emergent surgical exploration, and additional imaging may be unnecessary.

In cases where urgent surgery is not indicated or plain radiography is nondiagnostic, further imaging is warranted. While water-soluble contrast enema (CE) had previously been viewed as a valuable study for LBO to differentiate mechanical from functional issues, it is less often utilized with the proliferation of multi-detector computed tomography (MDCT). It does, however, remain an important clinical tool for select patients. Water-soluble iodinated contrast is given as a retention enema and multiplanar fluoroscopic films are obtained. CE has been proven to be a sensitive (63–96%) and specific (80–96%) examination for the diagnosis of LBO [7, 9, 12, 13]. CE does, however, have its drawbacks, namely, patient discomfort and the need for a procedural radiologist, which may not be available 24 h a day. Additionally, while CE provides information on the degree and anatomic location of the obstruction, it often does not determine the cause of the obstruction nor the degree of inflammation or ischemia present.

Multi-detector computed tomography has now become the imaging modality of choice, with a reported sensitivity of 93% and a specificity of 96% [8, 14, 15]. MDCT has the advantage of being rapid and well tolerated and provides accurate large bowel morphology. In the absence of acute kidney injury, chronic kidney disease, or allergy, MDCT should be performed

with the addition of intravenous contrast, which improves the ability to identify the presence of a mass, inflammation, or bowel wall ischemia. The MDCT, when associated with multiplanar reconstruction and volume rendering, has a documented sensitivity of 83 %, specificity of 93 %, and an accuracy of 91 % in identifying ischemic complications [14, 15]. In cases of malignancy, MDCT has the added benefit of detecting local and regional metastases. Oral contrast is often unnecessary and not well tolerated in the setting of obstruction. Rectal water-soluble contrast may be administered to aid in the identification of distal colonic obstruction. MDCT also has the added benefit of identifying any complicating features, such as pneumatosis intestinalis and a cecal diameter greater than 12 cm, both indicative of impending perforation.

17.5 Initial Management of Large Bowel Obstruction

Although the definitive management of LBO depends on the underlying etiology, the initial treatment generally remains the same. Patients with LBO are usually intravascularly depleted and may sequester large volumes of fluid in the interstitial space. Additionally, patients may present with vomiting, leading to further electrolyte and volume losses. As such, initial treatment should focus on volume resuscitation and minimizing ongoing ischemia. These efforts should be implemented even before the definitive diagnosis is made. The choice of resuscitation fluid will depend on serum electrolyte analysis and clinical assessment. An indwelling urethral catheter allows for measurement of urine output and helps guide resuscitation. Patients with hemodynamic instability may require central venous access in order to assess central venous pressure and response to resuscitation. Should vomiting be present, nasogastric decompression should be instituted.

17.6 Specific Causes and Treatment

17.6.1 *Colorectal Cancer*

Colorectal cancers are the most common cause of large bowel obstruction in the United States and Western Europe. Between 7 and 29% of patients with colorectal cancers present with acute large bowel obstruction [16, 17]. The typical presentation is of a more insidious onset. Most patients report long-standing constipation and colicky abdominal pain. Emergency presentation of colorectal cancer more commonly occurs in advanced stages of the disease, frequently occurring in elderly patients with significant comorbidities [18]. Morbidity and mortality are extremely variable. American Society of Anesthesiologists (ASA) grades 3–4, preoperative renal failure, and the presence of proximal colon perforation with or without peritonitis have been identified as predictors of unfavorable outcome following surgery for malignant LBO [19–21]. The management of colorectal cancers involves a complex, multimodal approach and is far beyond the scope of this chapter. However, some key concepts will be discussed in detail.

17.6.1.1 **Obstructing Right Colon Lesions**

For obstructing cancers involving the proximal colon, situated between the cecum and splenic flexure, right hemicolectomy with primary ileocolic anastomosis is considered safe in the emergent setting, as long as this can be performed following the rules of oncologic resection [17, 18]. Published anastomotic leak rates of 2.8–10% have been reported and are similar to those reported for elective resections [17, 22–24]. Single stage operation has several advantages. It allows for resection of the obstructing lesion and proximal distended colon; it provides

immediate restoration of bowel continuity, obviating the need for a second operation for ostomy takedown, and alleviates the psychosocial impact associated with an ostomy. In patients with a mechanical obstruction, preoperative bowel preparation is contraindicated due to lack of benefit and risk of harm, and it should not preclude primary anastomosis [25]. Similarly, on-table lavage lengthens the operation, increases the risk of spillage, and does not have any beneficial effects on primary anastomosis [25, 26]. Primary resection and anastomosis should only be used in the absence of hemodynamic instability and generalized peritonitis secondary to free perforation as the risk of mortality rises significantly. In this situation, proximal diversion with mucous fistula should be performed.

17.6.1.2 Obstructing Left Colon Lesions

Traditionally, the management of left-sided lesions has differed from that of right-sided lesions because colocolonic and colorectal anastomoses have been regarded as more susceptible to leakage [27]. Surgical management of obstructing lesions of the left colon has evolved from a three-stage procedure (proximal colostomy, second-stage tumor resection, and third-stage stoma closure) to management with a single stage operation. While the three-stage operation has fallen out of favor, opinion is still divided as to the optimal management of left-sided lesions. Frequently performed in the emergency setting, the Hartmann's procedure (primary resection with end colostomy), first described in 1921, has been advocated as it is technically less complex, can be performed quickly, and avoids the morbidity associated with an anastomosis. This should be considered the procedure of choice in high-risk patients. There are disadvantages of this operation. Subsequent stoma reversal is only performed in approximately 60% of patients, usually due to advanced age or significant comorbidities [28, 29]. Additionally,

stoma reversal is associated with significant morbidity and mortality, up to 60 % and 35 %, respectively.

The single stage operation, when used in appropriate patients, has proven feasible in the management of left-sided malignant obstructions [30–32]. This procedure combines the treatment of the disease and restores intestinal continuity in a single operation, thus avoiding the morbidity and mortality associated with colostomy and its reversal. Optimal patient selection is of utmost importance, as the single stage operation should not be performed in the setting of peritonitis or shock.

More recently, the use of colonic stent placement to relieve obstruction and avoid emergency surgery has been utilized. Since their introduction in the early 1990s, colonic stents have been used for palliation or as a bridge to surgery for obstructing lesions of the left colon. The procedure involves fluoroscopic or endoscopic placement of a metallic stent at the site of obstruction. The stent is allowed to self-expand, thus maximizing the patency of the bowel. Colonic stents are indicated in patients who are deemed non-operative candidates due to the extent of malignant disease or those who are considered high-risk operative candidates due to underlying comorbidities [27, 33]. The use of colonic stents and endoscopic management of LBO will be discussed in detail in the ensuing chapter.

17.6.2 Colonic Volvulus

Colonic volvulus is the axial rotation of the colon around its mesentery. Volvulus is thought to be an idiopathic condition, probably with an anatomical basis. The condition results in complete or partial obstruction of the colon and causes impingement of the blood supply. Although colonic volvulus is relatively rare in the United States and Western Europe, accounting for only 1–7 % of all large bowel obstructions, it is much more common in parts of Africa, South Asia, and South America and is the most common cause for large bowel obstruction [34–36].

17.6.2.1 Cecal Volvulus

Colonic volvulus may occur in any segment of the colon which is mobile and attached to a long mesentery that is fixed to the retroperitoneum by a narrow base; however, the mesenteric anatomy is such that colonic volvulus is most common in the sigmoid colon. Volvulus may also involve the right colon and terminal ileum (cecal volvulus), the cecum alone (cecal bascule), and, rarely, the transverse colon. Cecal volvulus occurs when the cecum is poorly fixed and highly mobile or when there is anomalous fixation of the right colon to the retroperitoneum. However, anatomic variation alone does not account for the wide variation in incidence of cecal volvulus throughout the world. Factors such as previous abdominal operations, chronic constipation, high-fiber diets, ileus, distal colon obstruction, and late-term pregnancy have all been identified as predisposing factors in the development of cecal volvulus [37–39]. Cecal bascule, though considered by most as a volvulus, is actually the anterosuperior folding of the cecum over the ascending colon, without axial rotation. This occurs less commonly than true rotational volvulus and is less likely to cause vascular compromise [37, 39]. Cecal volvulus and sigmoid volvulus exhibit different patient demographics. While sigmoid volvulus presents more commonly in elderly men, the majority of patients with cecal volvulus are younger women [40].

The typical presentation of patients with cecal volvulus is the acute onset of abdominal pain and distention. Depending on the acuity of symptoms, patients may be able to identify the exact time of onset. Cecal volvulus may resolve spontaneously; thus, many patients give a history of chronic, intermittent symptoms. Because cecal volvulus involves both the cecum and terminal ileum, symptoms of distal small bowel obstruction may also be present. In the early stages of the disease, patients may complain of mild abdominal pain; however, as the obstruction progresses, vascular compromise may lead to gangrene and peritoneal signs. On physical exam, a palpable mass may be noted in right lower quadrant.

If the presentation is suspicious for cecal volvulus, plain abdominal radiography is often diagnostic. The classic finding is the “coffee bean” sign, whereby the distended cecum is displaced out of the right lower quadrant into the left upper quadrant. Should contrast enema be performed, a classic “beak” sign, representing the point of torsion, will be demonstrated. Occasionally, contrast enema may reduce the volvulus, negating the need for emergent operation.

The patient with cecal volvulus may initially be managed with decompression; however, surgical resection remains the mainstay of treatment. Colonoscopic decompression, while commonly used for sigmoid volvulus, has not shown long-term efficacy as the sole treatment for cecal volvulus [41–43]. Although it is rarely advocated as a definitive treatment because of its high recurrence rates of 20–70%, colonoscopic decompression is considered a temporizing measure, allowing surgical intervention to be performed on an elective or semi-elective basis [41, 44–47].

Operative management of cecal volvulus can be divided into two broad categories: resective versus non-resective procedures. In general, non-resective procedures are not advocated due to the exceedingly high recurrence rates, reported up to 75% for cecal detorsion and 20–30% with the addition of cecopexy [38, 48–50]. Primary resection of the cecal volvulus is the primary method of management. At operation, untwisting of the torsed cecum is not recommended, as septic shock may result from rapid intravascular influx of toxins from the gangrenous segment [8, 51]. The majority of patients can be resected and primarily anastomosis in the same setting. When gangrenous changes, perforation, or hemodynamic instability is encountered, resection with end ileostomy and mucous fistula or a planned second-look operation may be necessary.

17.6.2.2 Sigmoid Volvulus

Volvulus of the sigmoid colon is the most common form of volvulus in the United States. Sigmoid volvulus occurs when the sigmoid colon is significantly elongated (dolichosigmoid) and the mesocolon is narrow at its base. Sigmoid volvulus most commonly affects elderly males. Similar to cecal volvulus, it is generally agreed that sigmoid volvulus does not occur with dolichosigmoid alone; otherwise, children, who have a baseline redundancy in their sigmoid colon, would be most affected [52, 53]. The two most common predisposing factors are chronic constipation and the use of psychotropic medications, as evidenced by the high incidence among chronically institutionalized, elderly patients, with poor intake of fluids and dietary fiber [52–54].

The clinical presentation depends on the duration and degree of colonic torsion, but, in general, sigmoid volvulus presents in a similar fashion as cecal volvulus; abdominal pain, cramping, distention, and obstipation are hallmark signs. However, as previously noted, patients with sigmoid volvulus are typically elderly, chronically ill individuals. This may preclude their ability to provide a useful history and physical exam may be of limited use. These patients are often brought in by their caregivers who note that the patient has not had a bowel movement, appears distended, and may be obtunded. This leads to a delay in the diagnosis, sometimes for up to 48–72 h. Approximately 30–60% will report previous similar episodes [37, 55, 56]. If an incomplete obstruction exists which may occur with torsion of less than 180°, allowing liquid stools to pass, paradoxical diarrhea may be a presenting feature.

In 60–90% of cases, plain abdominal radiographs alone are sufficient to establish the diagnosis [5, 57, 58]. The massively distended ahaustral sigmoid loop will be oriented with its apex

in the right upper quadrant, giving rise to the classic “bent inner tube,” “coffee bean,” or “omega” configuration of the bowel, being specific to sigmoid volvulus, and is present in up to 60% of cases [58, 59] (see Fig. 17.1). In the emergency setting, MDCT is often the diagnostic examination of choice for non-specific gastrointestinal problems and is often performed first. A “beak” sign may be seen at the point of obstruction and, if necessary, may be confirmed with the addition of rectal contrast. Similar findings are generally found on barium or water-soluble contrast enema; however, these studies should not be utilized if gangrene or perforation is suspected.

Unlike cecal volvulus, the primary strategy for treating sigmoid volvulus is early sigmoidoscopic or colonoscopic decompression followed by elective surgery [35, 44, 53, 60–62]. An alternative to decompression by flexible sigmoidoscopy is the

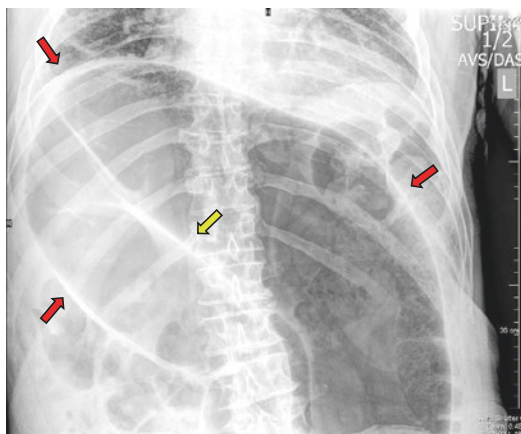


Fig. 17.1 65-year-old man with a sigmoid volvulus. Supine anteroposterior radiograph of the abdomen demonstrates the classic “coffee bean” sign (red arrows) in sigmoid volvulus. The apex of the volvulus points toward the right upper quadrant. Note also the central cleft (yellow arrow) of the coffee bean

use of rigid proctoscopy with the placement of a rectal decompression tube. However, the location of the obstruction is often beyond the limit of the rigid proctoscope. Because the recurrence rate following decompression approaches 90%, elective or semi-elective resection should be performed during the same hospital admission [55, 61, 63, 64]. Definitive surgery should be performed within 2–7 days following decompression, barring complications. If, at the time of surgical exploration, the colon is viable, a number of treatment options exist. Detorsion alone should be avoided as it carries an unacceptable recurrence rate. Rectopexy, both laparoscopic and open, have been shown to be reliable approaches in acute and elective settings. If the bowel appears only mildly compromised, primary resection and anastomosis should be considered. However, in the setting of gangrene or perforation, a Hartmann procedure or damage control operation should be performed. Occasionally, patients present with long-standing or recurrence sigmoid volvulus with resultant megacolon, in which case a subtotal colectomy with ileorectal anastomosis may be warranted.

17.6.3 Acute Colonic Pseudo-Obstruction

Acute colonic pseudo-obstruction (ACPO) refers to a syndrome defined by abnormal colonic distention in the absence of mechanical obstruction. Ogilvie's syndrome is an eponym for acute colonic pseudo-obstruction. Ogilvie's syndrome is believed to be a functional disturbance of colonic motility often observed in hospitalized patients as a result of hemodynamic, metabolic, pharmacologic, inflammatory, or postoperative conditions. Although not a mechanical obstruction, ACPO may present with features similar to mechanical LBO. The clinical features of Ogilvie's syndrome include abdominal distention, with or without abdominal pain, in hospitalized or institutionalized patients with serious underlying medical and

surgical conditions. Patients usually present with constipation; however, passage of flatus or stool is reported in up to 40% of patients. In a large retrospective series of 400 patients, Vanek et al. reported the most common predisposing conditions associated with Ogilvie's syndrome were non-operative trauma (11%), infections (10%), and cardiac disease (10%) [65]. Additional predisposing [65, 66] factors, such as severe metabolic derangements, sepsis, gastrointestinal infections, medications, and spinal cord injuries, have also been implicated in the development of Ogilvie's syndrome [65–67]. Although the diagnosis of ACPO may be suggested by the clinical presentation, mechanical obstruction must be ruled out. Plain abdominal radiographs will show varying degrees of colonic dilation (see Fig. 17.2). In contrast to mechanical LBO, air will be noted in the rectum. If the diagnosis is in question, mechanical obstruction can be excluded by performing a water-soluble contrast enema or rectal contrast-enhanced MDCT scan. This has the added benefit of creating an osmotic effect which may be therapeutic in decompressing the colon. MDCT may also identify signs of impending perforation, such as a cecal diameter of greater than 9 cm, or signs of colonic ischemia.

Treatment of ACPO is primarily medical. Nasogastric decompression should be used in patients with concomitant paralytic ileus. Electrolyte and metabolic abnormalities must be corrected, and offending medications, such as opioids, anticholinergic agents, norepinephrine, and dopamine, should be minimized or discontinued if possible. Success of conservative management is variable, ranging from 20 to 92%. If these measures are ineffective, intravenous neostigmine should be administered. Neostigmine is highly effective in inducing colonic decompression; however, relapse is common and occurs in 40% of patients [68, 69]. In patients whom medical management has failed, colonoscopic decompression should be performed [4, 70]. This may be performed without the use of bowel preparation and advancement to the hepatic flexure usually results in



Fig. 17.2 47-year-old man with developmental delay and Ogilvie's syndrome. Computed tomography scout film demonstrates massive gaseous distention of the colon overlying dilated loops of small bowel (paralytic ileus)

adequate decompression [70]. To increase therapeutic benefit, decompression tube placement at the time of colonoscopy may reduce recurrence, but controlled trials with this intervention are not available.

Surgical management is rarely necessary and should be reserved for patients who have failed pharmacologic and endoscopic management or those who have clinical signs of colonic ischemia or perforation. Surgical options include a venting stoma (cecostomy) or colectomy. Ogilvie's syndrome is one of the few conditions where cecostomy is indicated. Tube

cecostomy should be performed only in patients without evidence of ischemia or perforation. It can be performed laparoscopically or through a limited right lower quadrant incision. A large Foley catheter is left in place for 2–3 weeks to allow venting of the colon. Cecostomy can be performed under local anesthesia. In cases of ischemia or perforation, laparotomy is indicated. Segmental or subtotal resection may be performed, as dictated by the extent of colon involvement. In the event a colectomy is needed; an end stoma and mucous fistula should be performed and anastomosis avoided.

17.7 Summary

Acute large bowel obstruction is a complex syndrome frequently encountered by the acute care surgeon. In the United States, the majority of large bowel obstructions are caused by colorectal carcinoma, colonic volvulus, and diverticulitis. The treating physician must include physiological causes of LBO in the differential diagnosis. Acute colonic pseudo-obstruction may mimic mechanical bowel obstruction; however, the treatment is drastically different. The astute surgeon must rapidly evaluate the patient and implement the appropriate treatment algorithm so as to limit morbidity and mortality. Ultimately, the treatment needs to be tailored to the individual situation.

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Chapter 18

Endoscopic Management of Large Bowel Obstruction

Marco Bassi, Stefania Ghersi, Carlo Fabbri, Anna Larocca, and Vincenzo Cennamo

18.1 Introduction

About 8–29% of patients with colorectal malignancy present with acute colonic obstruction [1, 2].

Emergency surgery procedures required for the treatment of large bowel obstruction (LBO) are associated with a mortality rate of 15–20% and a morbidity rate of 40–50% [3, 4].

Colonic stent placement for malignancy was first used in the early 1990s and has been then proposed either as palliative strategy in inoperable patients or as a bridge to surgery; Tejero et al. first reported the use of self-expanding metal stents (SEMSs) as a bridge to surgery in two patients with colonic obstruction in 1994 [5].

In the setting of operable patients, the temporary placement of a colonic stent, allowing colonic decompression, should

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avoid the need of emergency surgery and shift to elective surgical strategy, avoiding colostomy and providing the possibility of subsequently performing an elective segmental resection with a primary anastomosis, even laparoscopically [6, 7].

Furthermore the SEMS placement erasing the need of emergency surgical treatment enables clinical management of patients with volume resuscitation and treatment of underlying comorbidities allowing to improve general conditions before undergoing to elective surgery [6–8].

Finally, in patients with rectal cancer, preoperative neoadjuvant therapy can be administered with the stent in place after relief of obstruction [9].

Although SEMS placing is currently used widely in daily clinical practice, the scientific evidence for SEMSs in the colorectum is not yet sufficient; the debate regarding the advantages and limitations of SEMS is still ongoing.

18.2 Indications

SEMS has been used in both malignant and benign obstructions. Neoplastic obstruction is still the main indication for SEMS in the colon. The purposes of SEMS insertion in malignant colon obstruction can be divided either in a bridge to surgery or palliation in inoperable patients.

18.2.1 SEMS as a Bridge to Surgery

The strategy of bridge to surgery by using a stent placement includes three steps, each with specific aim and outcomes. Any step contributes as a part to the whole process of bridging.

First step is the SEMS placement, allowing to avoid the need of emergency surgery. The second step is the medical treatment

during the time between stenting and surgery. During this time the effort should be addressed to manage the comorbidities and to provide the best condition for the elective surgery. The third step is the elective surgical strategy that should be performed in accordance with the guideline of minimal invasive surgery.

Colonic stent placement as bridge to surgery purpose has been shown to be effective in large nonrandomized series.

The first data regarding the knowledge of efficacy and safety of colonic stent in bridge to surgery came from uncontrolled trials and individual case series. In a polled analysis from Sebastian et al., including stent placement series for both palliative and bridge to surgery strategy, the percentage success rates in individual series varied from 64 to 100% (median 94%, i.q.r. 90–100). The technical success in the palliative group was 93.35% and in the bridge to surgery group was 91.9% ($p=0.34$).

In the bridge to surgery group, including a total of 407 patients in 21 series of nonrandomized reports, the procedure was technically successful in 374 (91.9%) patients. Clinical success in this subgroup, defined as the ability to perform a single-stage surgery with primary anastomosis, was achieved in 292 patients. The overall percentage of clinical success was 71.7%. The mortality rate from stent insertion was 0.5% and is significantly lower than the reported figures for emergency surgery [10].

Subsequently several reviews including nonrandomized and randomized studies comparing the outcomes of SEMS followed by elective surgery to those of emergency surgery without prior stenting have been published.

A review including only nonrandomized studies on 363 patients with stents placed as a bridge to surgery showed that rates of primary anastomosis after elective surgery following stenting were at least twice that of those undergoing emergency surgery. Colostomy rates were notably higher in cases of emergency surgery than elective surgery. SEMS followed by elective surgery was effective both on mortality and morbidity, and the

length of hospital stay was shorter after SEMs and elective surgery than emergency surgery [11].

However, the preliminary results came from uncontrolled trial. Therefore, in the last years, several RCTs have been performed [6, 12–17].

The study included several variables, such as the type of stent, the definition of stenting clinical success, the kind of elective surgery employed, and the emergency surgery.

The mean technical success rate of SEMs placement was 76.9%. Morbidity ranged from 8 to 53% in the stent group and 53–71% in the emergency surgery group. Mortality ranged from 0 to 19% in the stent group and from 0 to 18% in the emergency surgery group.

The high rate of complications and failure in the stenting groups among these trials affected the results of derived meta-analysis. Indeed the review and meta-analysis published including some of these trials, together with other nonrandomized trials or only with randomized trials, showed results not in accordance with previously published meta-analysis.

Indeed, overall complications (RR, 0.42; 95% CI, 0.24–0.71; $p=0.001$), including anastomotic leakage (RR, 0.31; 95% CI, 0.14–0.69; $p=0.004$), were reduced by stent insertion in the meta-analysis by Zhang et al. [18].

These results have been confirmed by meta-analysis including three randomized trials together with five nonrandomized trial studies involving 444 patients in which there was significant difference (RR, 0.57; 95% CI, 0.44–0.74; $P<0.0001$) in the overall morbidity [19].

By contrast, the meta-analysis including only randomized trials comparing SEMs vs surgery for intestinal obstruction from left-sided colorectal cancer as a bridge to surgery showed no benefits by using stenting to reduce the morbidity rate. Tan et al., including four RCTs with 234 patients, showed that there was no significant difference in anastomotic leak, 30-day reoperation, and surgical-site infection rate.

All but one reviews and meta-analysis, irrespective of number of randomized trials included, agreed on the advantage of bridge to surgery to achieve an increased rate of primary anastomosis.

Indeed the primary anastomosis rate in the stent group was higher (RR, 1.62; 95 % CI, 1.21–2.16; $p=0.001$) in the meta-analysis comparing 232 patients underwent stent insertion and 369 underwent emergency surgery [18].

Similar results were reported by another meta-analysis including mixed papers and only randomized trials. In one study, there has been reported difference of the one-stage stoma rates between the two groups (RR, 0.60; 95 % CI, 0.48–0.76; $P<0.0001$) [19].

Furthermore, SEMS has been shown to lower overall stoma (RR 0.71, 0.56–0.89; $P=0.004$) rates in the short-term follow-up. By contrast there was no significant difference in the permanent stoma rate [20].

This result has been confirmed in meta-analysis with only randomized controlled trials included, with no significant differences between the two groups as to permanent stoma rate [19, 21].

However, the overall stoma rate was significantly lower in the stent group (45.3 %) compared with the emergency surgery group (62 %) ($p<0.02$) [21].

The most recent systematic review and meta-analysis that evaluated the efficacy and safety of colonic stenting as a bridge to surgery ($n=195$) compared with emergency surgery ($n=187$) and considered only randomized controlled trial for inclusion have been studied. All randomized controlled trials that focused on the postoperative outcome of stent placement and emergency surgery were included in this meta-analysis. The mean technical success rate of colonic stent placement was 76.9 % (range 46.7–100 %), and SEMS placement as a bridge to surgery followed by elective surgery showed a lower overall postoperative morbidity (33.1 % vs. 53.9 %, $p=0.03$), higher primary anastomosis rate (67.2 % vs. 55.1 %, $p<0.01$), and lower stoma rate

(9% vs. 27.4%, $p < 0.01$) when compared to emergency surgery in left-sided colorectal cancer obstruction.

Despite these favorable immediate postoperative clinical courses, the overall postoperative mortality after SEMS insertion as a bridge to surgery was similar to that after emergency surgery (10.7% vs. 12.4%) [22].

Furthermore, the long-term oncological outcome, such as disease recurrence, was worse in the group with SEMS as a bridge to surgery than in the emergency surgery group, as shown in the Table 18.1 [15, 17, 23, 24].

Based on these unexpected long-term oncological outcomes, the recent guidelines by the European Society of Gastrointestinal Endoscopy (ESGE), endorsed by the Governing Board of the

Table 18.1 Oncological outcome after SEMS insertion as a bridge to surgery

	Study population	Study design	Results
Sloothaak et al. [24]	Preoperative SEMS = 26 Emergency surgery = 32	Follow-up data of RCT	5-year overall recurrence rate ($p = 0.027$): SEMS as a bridge to surgery: 42% (11/26) Emergency surgery: 25% (8/32)
Tung et al. [17]	Preoperative SEMS = 24 Emergency surgery = 24	Follow-up data of RCT	Overall recurrent disease ($p = 0.4$): SEMS as a bridge to surgery: 50% (11/22) Emergency surgery: 23% (3/13)
Alcantara et al. [15]	Preoperative SEMS = 13 Emergency surgery = 13	RCT	Tumor reappearance ($p = 0.055$): SEMS as a bridge to surgery: 53% (8/15) Emergency surgery: 15% (2/13)

American Society for Gastrointestinal Endoscopy (ASGE), do not recommend routine SEMS insertion as a bridge to surgery in potentially curable left-sided colorectal obstruction. Therefore, emergency surgery should be considered the first option in left-sided colorectal obstruction rather than SEMS as a bridge to surgery, unless new scientific evidence will emerge. However, surgical resection is suggested as the preferred treatment for malignant obstruction of the proximal colon in patients with potentially curable disease [25].

The timing of elective surgery after stent placement and the clinical strategy during intercurrent period between stenting and elective surgery should be considered as a part of strategy and assessed to evaluate the whole efficacy of process. Furthermore, the patients could be submitted to surgery after improving their general conditions. Although there are limited data to determine an optimal time interval to operation after SEMS placement as a bridge to surgery, an interval to operation of 5–10 days is suggested when SEMS is used as a bridge to elective surgery in patients with potentially curable left-sided colon cancer [25].

18.2.2 Palliative SEMS

A meta-analysis that reviewed 13 studies regarding palliative SEMS for incurable neoplastic colorectal obstruction ($n=404$) in comparison to palliative surgery ($n=433$) showed a shorter duration of admission (10 days vs. 19 days) and a lower frequency of admission to the intensive care unit (0.8 % vs. 18 %).

The technical success of stent placement in the studies included ranged from 88 to 100%. Chemotherapy could also be started earlier after palliative SEMS insertion than after palliative surgery (16 days vs. 33 days). In addition, colostomy/ileostomy was required less frequently after palliative SEMS insertion (13 % vs. 54 %). The long-term complications were more common in the palliative SEMS group, which included colonic perforation (10 %), stent migration

(9%), and re-obstruction (18%). However, the overall morbidity was similar (34% in the SEMS group vs. 38% in the surgery group) [26, 27].

Thus, considering all these findings, the ESGE guidelines recommend SEMS placement as the preferred treatment for palliation of incurable CRC obstruction.

However, also in the strategy of treatment for malignant obstruction of the proximal colon, colonic SEMS can be an alternative to emergency surgery [25, 26].

Palliative SEMS insertion has been also attempted in patients with colonic obstruction due to extra-colonic malignancy. A technically successful SEMS insertion was achieved in 67–96%, while clinical success was obtained in 20–96% of patients. Although these outcomes may be slightly worse than those of SEMS insertion for primary CRC obstruction, palliative SEMS can still be indicated in patients with colonic obstruction from extra-colonic malignancy, especially in those with a relatively short expected survival time and those who are poor surgical candidates [23, 28].

There are insufficient data regarding the outcome of stent placement in patients with peritoneal carcinomatosis. Despite the lower probability of success, SEMS placement may be an alternative to surgical decompression in the setting of peritoneal carcinomatosis [25].

Stent re-obstruction (4.0–22.9%) and stent migration (1.0–12.5%) are two common late complications, and perforation may also occur as a late complication [26, 27, 29].

Most migrations are distal, and spontaneous expulsions of the stents per anus occur occasionally. Risk factors for migration include colonic stents with a small diameter less than 24 mm and balloon dilation [30].

Covered SEMS is also a risk factor for migration (5.5% vs. 21.3% in the uncovered SEMS group) [31, 32].

The most common late complication is re-obstruction. Most re-obstructions are caused by tumor ingrowth.

However, tumor overgrowth, fecal impaction, and mucosal prolapse can also lead to stent re-obstruction. According to a meta-analysis of 54 case series, the re-obstruction rate in the covered SEMS group was lower than that in the uncovered SEMS group (4.7% vs. 7.8%, $p=0.003$). The lower rate of re-obstruction in the covered SEMS group is believed to be related to the lower rate of tumor ingrowth (0.9% vs. 11.4%) [10, 31, 32].

Both migration and re-obstruction can be managed with endoscopic intervention. Stent re-obstruction can be treated by balloon dilation, argon laser therapy, and additional stent insertion [23].

Recently a warning about stent placement and concomitant chemotherapy has been proposed; furthermore, a multicenter retrospective study explored the relationship among K-ras mutation status, antitumoral treatments, and SEMS-related complication rates [33, 34].

The results of this study confirm the increased risk of stent perforation in case of concomitant bevacizumab-based chemotherapy.

Finally, a recent meta-analysis has shown that the perforation risk of bevacizumab-based chemotherapy was significantly increased when compared with non-concomitant therapy group [35].

However, given the high risk of colonic perforation, it is not recommended to use SEMS as palliative decompression if a patient is being treated or considered for treatment with antiangiogenic therapy (e.g. bevacizumab) [25].

18.3 Technical Considerations

Abdominopelvic computed tomography (CT) scan is usually performed to determine the etiology of colonic obstruction. CT scan can also provide information on the anatomy of the

patient's colon, the length and severity of the obstruction, and any other concurrent problems, such as perforation [36].

Colonic stenting should be avoided for diverticular strictures or when diverticular disease is suspected during endoscopy and/or CT scan.

Prophylactic stenting for patients with colonic malignancy but no evidence of symptomatic obstruction is strongly discouraged because of the potential risks associated with colonic SEMS placement [25].

Although some argue that prophylactic antibiotics should be considered in patients with complete obstruction and dilated proximal colon because of the increased risk of perforation during SEMS placement, the administration of prophylactic antibiotics prior to colonic SEMS insertion is not recommended [25, 37].

Stent delivery device could be either placed over the wire or through the scope, under endoscopic alone, endoscopic and radiologic, or radiologic alone guidance; however, colonic SEMS placement is recommended with the combined use of both endoscopy and fluoroscopy [25].

Briefly, a low-traumatic hydrophilic guidewire inserted in a 5- or 7-French endoscopic retrograde cholangiopancreatography (ERCP) catheter or a sphincterotome is introduced to cannulate the stricture; the guidewire is then withdrawn, and a contrast agent is injected to delineate the location, length, and anatomy of the stricture (Figs. 18.1 and 18.2). A 0.035-in. super-stiff guidewire is then inserted after removing the hydrophilic guidewire, and the catheter is withdrawn.

The SEMSs selected should have a length adequate to cover the entire stricture at least 2 cm beyond both stricture edges.

Stricture dilation has been commonly considered a risky technique leading to perforation as shown in several papers.

Perforation risk also appears to be heightened by balloon dilation as assessed by a systematic review on efficacy and safety of colorectal stents [38].

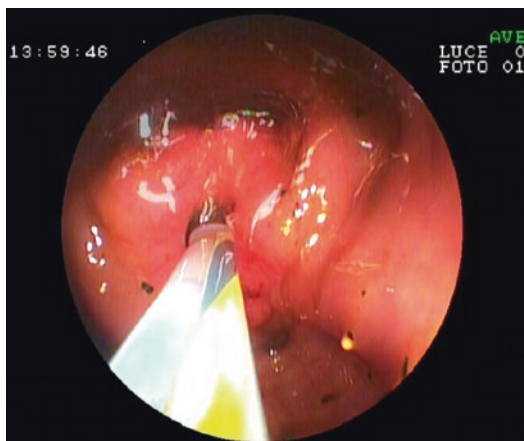


Fig. 18.1 Endoscopic view of stricture cannulation

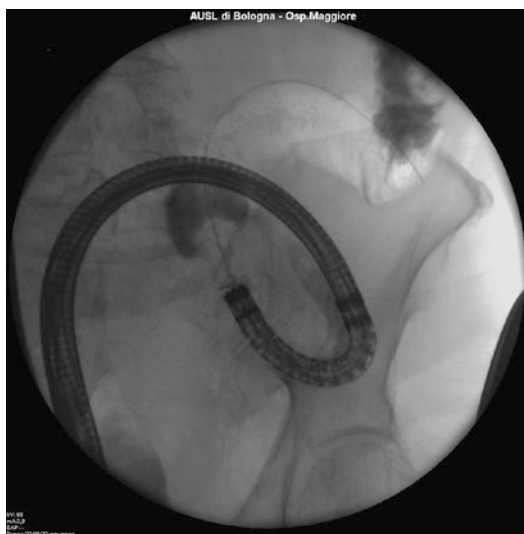


Fig. 18.2 Endoscopic colonography showing colonic stricture and proximal colon dilation

Although these considerations are based on low-quality evidence with small patient numbers, according to the guidelines above cited, stricture dilation either before or after stent placement is discouraged [25].

The diameter of the SEMS should be 24 mm or larger so that decompression can be effective [31].

The SEMS is then deployed through the endoscope, and the deployment of the proximal portion of the SEMS should be monitored by fluoroscopy (Figs. 18.3 and 18.4).

At the end of the procedure, a contrast agent is injected again through the endoscope to rule out the presence of a perforation [39].

As the main limitation in placing the stent is the passage of guidewire through the stricture, several technical tricks are suggested to improve the rate of successful placement. The use of hydrophilic biliary guidewires helps to achieve a way to pass through the stricture; a clear cap and a sphincterotome are also used to orient the catheter in the direction of the

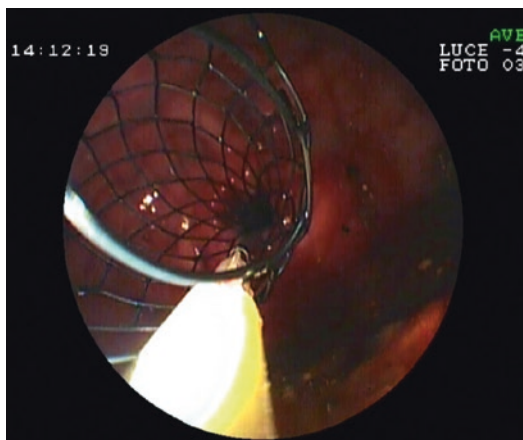


Fig. 18.3 Successful stent deployment



Fig. 18.4 X-ray view of successful stent deployment

lumen, particularly for lesions at flexures or corners [40]; in these cases, the use of a side-viewing endoscope has been proposed [41].

All these tricks, useful to reach a high rate of technical success in colorectal stenting, are borrowed from the ERCP's cannulation techniques. Indeed, endoscopists with pancreaticobiliary experience seem to have higher success rates and lower complication rates than those without [42].

18.4 Conclusions

Colorectal stenting is safe and effective for the decompression of neoplastic colonic obstruction. Therefore, for the past

two decades, colorectal SEMSs have been used safely and effectively, thus providing a bridge to surgery of potentially curable CRC malignancies and palliation for patients with incurable CRC or extra-colonic tumors. However, recent data suggest that the long-term oncological outcome after SEMS insertion as a bridge to surgery is less favorable than that following emergency surgery.

Therefore, current indications for colorectal SEMS placement include palliation for colonic obstruction by primary CRC or extra-colonic malignancy and placement, as a bridge to surgery, only in those at high surgical risk, such as those with an ASA classification \geq III and elderly patients aged over 70 years. Because colorectal SEMSs have a variety of clinical benefits, further investigations are needed to overcome its limitations.

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Chapter 19

Management of Colonic Diverticulitis

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19.1 Introduction

Acute left colonic diverticulosis (AD) is common in Western countries, but its prevalence is increasing throughout the world [1]. On one side left colonic diverticulosis is more common

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among elderly patients; on the other side, however, a dramatic rise of its incidence has been seen in the younger age groups during recent years [2]. Data from Western populations suggest that up to one fifth of patients with acute diverticulitis are under the age of 50 years old [3–5]. The lifetime risk of developing acute left colonic diverticulitis (AD) is about 4% among patients with diverticulosis [6].

The management of this common disease depends principally on the local and systemic severity of inflammation/infection and on the presence or not of diverticular perforation. Even with some remaining concerns, nowadays several high level of evidence [8, 9] data exist driving to a better management of AD.

19.2 Classification Systems

AD ranges in severity from uncomplicated inflammatory diverticulitis to complicated diverticulitis (abscess formation or perforation). Several classification systems have been published during the last three decades as shown in Table 19.1 [7, 10–14].

The advent of computed tomography (CT) imaging as a primary diagnostic and staging tool in patients with AD led to several modifications of the Hinchey classification [7, 11–14]. The main introduction since Hinchey's grading system publication has been the progressive increase in free air consideration. In fact the distinction between the different kinds of free fluid (i.e., purulent or fecaloid) based only on the CT scan images has been demonstrated to be hard. However, taking into consideration the importance of this distinction as it is one of the major issues in leading the treatment decision, the needing for accurate CT-based grading system is high. The most recent classifications introduced the evaluation of free air as a sensible proxy in evaluating the presence of perforation associated to the patient clinical conditions. This can more precisely help in planning a tailored diagnostic-therapeutical path (Fig. 19.1).

Table 19.1 Classifications of acute diverticulitis

Author [Ref.]	Year	Disease grading	Based on
Hinchey et al. [10]	1978	1 Pericolic abscess 2 Pelvic, intra-abdominal, or retroperitoneal abscess 3 Generalized purulent peritonitis 4 Generalized fecal peritonitis	CT ^a finding
Neff and vanSonnenberg [11]	1989	0 Uncomplicated diverticulitis (diverticula, thickening of the wall, increased density of the pericolic fat) 1 Locally complicated with local abscess 2 Complicated with pelvic abscess 3 Complicated with distant abscess 4 Complicated with other distant complications	CT ^b finding
Ambrosetti et al. [12]	2002	<i>Moderate diverticulitis:</i> Localized sigmoid wall thickening of ≥ 5 mm Pericolic fat stranding <i>Severe diverticulitis:</i> Abscess formation Extraluminal air Extraluminal contrast	CT ^b finding

(continued)

Table 19.1 (continued)

Author [Ref.]	Year	Disease grading	Based on
Mora Lopez et al. [13]	2013	0 Uncomplicated diverticulitis (thickening of the wall, increased density of pericolic fat) 1 Locally complicated diverticulitis A Localized pneumoperitoneum in the form of air bubbles B Abscess (<4 cm) 2 Complicated diverticulitis with pelvic abscess (>4 cm) 3 Complicated diverticulitis with distant abscess (abscess in abdominal cavity outside pelvis) 4 Complicated diverticulitis with other distant complications (abundant pneumoperitoneum and/or intra-abdominal free liquid)	CT ⁿ finding
Sallinen et al. [14]	2015	1. Uncomplicated diverticulitis 2. Complicated diverticulitis with small abscess (<6 cm) 3. Complicated diverticulitis with large abscess (≥6 cm) or distant intraperitoneal or retroperitoneal air 4. Generalized peritonitis without organ dysfunction 5. Generalized peritonitis with organ dysfunction	CT ⁿ – clinical – physiological findings

Sartelli et al. [7]	2015	<p><i>Uncomplicated diverticulitis:</i></p> <ol style="list-style-type: none"> 1. Diverticula, thickening of the wall, increased density of the pericolic fat <p><i>Complicated diverticulitis:</i></p> <ol style="list-style-type: none"> 2. A Pericolic air bubbles or little pericolic fluid without abscess <ol style="list-style-type: none"> B Abscess ≤ 4 cm 3. A Abscess > 4 cm <ol style="list-style-type: none"> B Distant air (> 5 cm from inflamed bowel segment) 4. Diffuse fluid without distant free air (no hole in the colon) 5. Diffuse fluid with distant free air (persistent hole in the colon) 	CT ^a finding
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^aComputed tomography

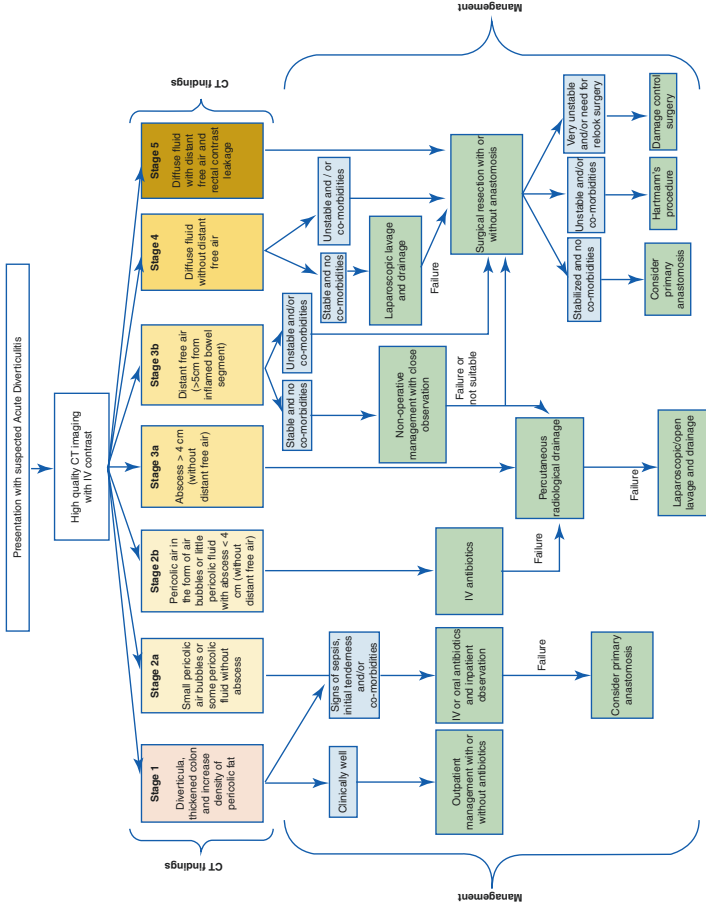


Fig. 19.1 Acute diverticulitis management algorithm (Modified from Sartelli et al. [7])

19.3 Diagnosis

Clinical findings of AD usually include acute pain or tenderness in the left lower quadrant which may be associated with increased inflammatory markers including C-reactive protein (CRP) and white blood cell count (WBC).

Clinical diagnosis of AD usually lacks in accuracy; ultrasound and CT had superior diagnostic accuracy; however, according to some authors, these examinations rarely lead to a change in the initial management decision [15].

Lameris et al. proposed three criteria for the diagnosis of AD: (1) direct tenderness in the left lower quadrant, (2) CRP > 50 mg/L, and (3) absence of vomiting [16]. This three criteria method showed an efficacy in diagnosis of AD in a quarter of patients with suspected diverticulitis [16].

Andeweg et al. proposed a clinical scoring with a diagnostic accuracy of 86 % [17]. It was based on (1) patient's age, (2) one or more previous episodes, (3) localization of symptoms in the lower left abdomen, (4) aggravation of pain on movement, (5) the absence of vomiting, (6) localization of abdominal tenderness in the lower left abdomen, and (7) CRP > 50 mg/L.

Serological markers have been largely studied in AD [18, 19]. A retrospective study showed a cutoff value of 170 mg/L significantly discriminated episodes prone to become either severe or mild AD (87.5 % sensitivity, 91.1 % specificity, area under the curve 0.942, $p < 0.00001$) [18]. Another study showed the optimal threshold was reached at 175 mg/L with a positive predictive value of 36 %, negative predictive value of 92 %, sensitivity of 61 %, and a specificity of 82 % [19].

Recently Makela et al. showed a CRP value over 150 mg/L and old age were independent risk factors for acute complicated diverticulitis [20]. Moreover a CRP value over 150 mg/L and free abdominal fluid at CT scan were independent variables predicting postoperative mortality [20].

CT imaging is becoming the gold standard in the diagnosis and staging of patients with AD. CT imaging with intravenous contrast has excellent sensitivity and specificity [21–23].

CT scan criteria may be used either to diagnose AD or to determine the grade of severity and may also drive treatment planning of patients [7].

Ultrasound (US) is a real-time widely available and easily accessible dynamic examination that may be useful when only mild diverticulitis is suspected [24]. Its limitations include operator dependency, poor assessment in obese patients, and difficulty in detecting free air and deeply located abscesses [25]. A systematic review and meta-analysis reported a summary sensitivity for US of 90% (95% CI: 76–98%) versus 95% (95% CI: 91–97%) for CT ($p=0.86$) and a summary specificity for US of 90% (95% CI: 86–94%) versus 96% (95% CI: 90–100%) for CT ($p=0.04$) [26].

Some authors, however, proposed a step-up approach with CT performed after an inconclusive or negative US as safe and alternative approach for patients with suspected AD [26, 27]. Magnetic resonance imaging (MRI) is hardly performed in emergency setting, even if not exposing patients to radiation and not constrained by the operator dependency limitation of ultrasound [28, 29].

19.4 Management

19.4.1 *Immunocompromised Patients*

Immunocompromised patients (i.e., patients with kidney failure, diabetes, ongoing chemotherapy, hematological disorders, organ transplant, and patients using corticosteroids) are at higher risk to have complicated diverticulitis [30–33] and are prone to fail

non-operative treatment requiring urgent surgical intervention with a significantly higher mortality [34]. As a consequence these patients must raise a high level of suspicion, and an early empirical aggressive antibiotic treatment associated to an immediate surgical intervention in case of non-immediate response has to be actuated in order to prevent negative outcomes.

19.4.2 Uncomplicated Acute Diverticulitis

The utility of antimicrobial therapy in acute uncomplicated AD is debated. Several studies demonstrated that antimicrobial treatment was not superior to withholding antibiotic therapy [35].

Uncomplicated AD seems to be a self-limiting condition in immunocompetent patients.

A multicenter randomized trial involving 623 (antibiotic vs. no antibiotic) patients with CT scan-verified uncomplicated AD showed antibiotic treatment neither accelerated recovery nor prevented complications or recurrence [36]. However, the high mortality associated with sepsis requires a high index of clinical suspicion, in conditions predisposing to sepsis. WSES expert panel routinely recommends antimicrobial therapy in patients with radiological documented uncomplicated AD and signs of systemic inflammatory response syndrome. Oral antibiotic administration seems to be as effective as intravenous one, as showed by randomized trials [37].

Patients with uncomplicated AD symptoms without significant comorbidities, who are able to take fluids orally and manage themselves at home, can be treated as outpatients with adequate follow-up. Patients with significant comorbidities and unable to take fluids orally should be treated in hospital with intravenous fluid. A recent retrospective analysis demonstrated that outpatient treatment is effective for the vast majority (94%)

of patients [40]. Moreover a systematic review concluded that a more progressive, ambulatory-based approach to the majority of cases of uncomplicated AD is justified [41]; the same concept has recently been shown effective for elderly patients either with comorbidities [42].

The DIVER trial confirmed the aforementioned concepts and showed outpatient management can also reduce the costs (saving an average of €1124.70 per patient) without negatively influencing the patient's quality of life [43].

Recurrence after an uncomplicated episode of AD has been reported since now in almost one third of patients [44, 45] within the first year. Recently a prospective 5-year follow-up trial reported a recurrence rate of 1.7% [46–48]. A systematic review concluded that the general principle to proceed with elective colectomy after 2 episodes of diverticulitis is no longer acceptable. Decisions to proceed with elective colectomy should be made taking into consideration frequency of recurrent diverticulitis, surgical morbidity, ongoing symptoms, disease complexity, and operative risk [48].

19.4.3 Localized Complicated Diverticulitis

CT findings of pericolic air in the form of air bubbles or little pericolic fluid without abscess and distant air indicate a complicated AD and antimicrobial therapy, and hospital admission should always be recommended [7]; no studies have examined the value of dietary restriction or bed rest [39] (Fig. 19.1).

19.4.4 Diverticular Abscess

Abscesses on CT scan are present in 15–20% of patients with AD [49]. Several authors consider 3–6 cm in diameter the cutoff between antimicrobial therapy and percutaneous drainage [14, 49–54].

Others defined a size of 4/5 cm as the cutoff size [7].

The CT scan is the pivotal examination to be repeated in cases of clinical and laboratory improvement failure after drainage catheter placement. Generally the drainage catheter should be removed when the patient conditions have improved and the output has ceased. Cancer abscesses mimicking AD were described [50]. Systematic review of routine colonic evaluation after radiologically confirmed AD found a cancer incidence of 0.01 % [55].

A retrospective analysis of CT-studied AD showed a 2.7% of colon cancers in patients with an initial diagnosis of complicated AD; no cancers were found in patients with uncomplicated AD [56]. In case of diverticular abscesses of less than 4/5 cm in diameter, IV antibiotics should be immediately started, and hospital admission is required, and in case of antibiotic therapy failure, percutaneous drainage should be considered (Fig. 19.1).

In case of diverticular abscess of more than 4 cm in diameter, IV antibiotics should be immediately started, hospital admission is required, and percutaneous drainage should be the first attempt of source control; in case of drainage failure/impossibility, laparoscopic lavage and drainage should be considered (Fig. 19.1).

19.4.5 Diffuse Peritonitis

Up to 25% of admitted patients may require urgent surgical intervention [57]. Patients with diffuse peritonitis may present with severe sepsis or septic shock requiring prompt fluid resuscitation, antibiotic administration, and adequate source control without delay [58].

Antimicrobial therapy is necessary and important and should be administered immediately after the admission. Initially an empiric broader-spectrum regimen is recommended to be changed according to definitive antimicrobial susceptibility.

In managing AD with peritonitis, the demonstration at the CT scan of distant free air (with a distance >5 cm from the inflamed bowel segment) without diffuse fluids should be considered as a major issue. In fact, on one side, distant free air has been considered a known predictor of failure of non-operative treatment [23]; however, on the other side, some recent studies described high success rate of non-operative management in patients with AD and localized pneumoperitoneum, excluding those with hemodynamic instability, diffuse peritonitis, and free fluid in Douglas's pouch [59, 60].

These data showed highly selected patients at this stage may be treated by conservative treatment. However, strict CT monitoring is mandatory [7].

Suggested intervention for patients at this stage should be surgical resection and anastomosis with or without stoma in stable patients without comorbidities and Hartmann's resection in unstable patients or in patients with multiple comorbidities [7] (Fig. 19.1).

Conservative approach using laparoscopic peritoneal lavage and drainage has been debated in recent years as an alternative to the aforementioned treatments with the aim to avoid a stoma in patients with diffuse peritonitis [61]. The first doubts arose with a retrospective analysis of 38 patients treated by laparoscopic lavage [62]. In fact among seven patients undergoing laparoscopic lavage, the abdominal sepsis was not controlled (two died and five required further surgical interventions). Great debate is still open on this topic; three prospective randomized trials (DILALA, SCANDIV, and Ladies) gave contrasting conclusions in comparing laparoscopic lavage and resection [63–65].

The DILALA trial published in 2014 reported similar results in terms of morbidity and mortality between laparoscopic lavage and Hartmann's procedure in Hinchey III AD. Moreover laparoscopic lavage resulted in shorter operating time, shorter time in the recovery unit, and shorter hospital stay with the avoidance of a stoma [63].

Results from SCANDIV study were published in 2015 reporting no reduction in severe postoperative complications and worse outcomes in secondary end points with laparoscopic lavage in Hinchey III AD [64]. In the same year, the results of Ladies trial were consistent with SCANDIV trial: laparoscopic lavage was not superior to sigmoidectomy for the treatment of purulent perforated diverticulitis. However, survival was not compromised and stoma formation could be avoided in the majority of patients [65].

Hartmann's resection has been considered the procedure of choice in patients with generalized peritonitis and remains a safe technique for emergency colectomy in diverticular peritonitis, especially in critically ill patients and in patients with multiple comorbidities. However, restoration of bowel continuity after a Hartmann procedure has been associated with significant morbidity [66]. Many patients cannot undergo reversal surgery due to comorbidities; therefore, they remain with permanent stoma [67]. A recent retrospective administrative Australian study confirmed the common use of Hartmann's resection in treating diverticular perforation [68]. Among 2829 emergency admissions for AD, 724 were for complicated AD. One third of the admissions for complicated AD required surgical intervention. Hartmann's procedure accounted for the 72% of resections. Another administrative retrospective cohort study from Ontario (Canada) showed consistent results describing a 64% of Hartmann's procedures showing also that the frequency of use of this procedure remained unchanged within the whole study period (2002–2012) [69].

Some authors gave an increasing role to primary resection and anastomosis (PRA) with or without a diverting stoma in AD, even in diffuse peritonitis [70]. The studies comparing mortality and morbidity of Hartmann's procedure versus primary anastomosis didn't show any significant differences. It must be said that most of these studies have relevant selection biases [71–74].

A published comparison of primary resection and anastomosis (PRA) with or without defunctioning stoma to Hartmann's procedure (HP) as the optimal operative strategy for patients presenting with Hinchey stages III–IV considered 135 PRA, 126 primary anastomoses with defunctioning stoma (PADS), and 6619 HP [74]. Morbidity and mortality was 55 % and 30 % for PRA, 40 % and 25 % for PADS, and 35 % and 20 % for HP. Stomas remained permanent in 27 % of HP and in 8 % of PADS. The author's conclusions were that PADS may be the optimal strategy for selected patients with diverticular peritonitis and may represent a good compromise between postoperative adverse events, long-term quality of life, and risk of permanent stoma.

A randomized trial comparing primary anastomosis plus ileostomy vs. Hartmann's procedure in patients with diffuse diverticular peritonitis reported no statistically significant differences in initial mortality and morbidity, but a reduction in length of stay, lower costs, serious complications rate, and a greater stoma reversal rates in the primary anastomosis group [75].

In unstable patients with diverticular peritonitis, “damage control surgery” has been progressively more adopted in the last years [76]. Damage control with lavage, limited bowel resection, laparotomy, and scheduled second-look operation represents a feasible strategy in severely ill patients to enhance sepsis control and improve rate of anastomosis. A treating algorithm with damage control operation, lavage, limited closure of perforation, and second-look surgery to restore intestinal continuity, after a period in ICU, has been recently proposed to improve outcome and reduce the stoma creation rate in critically ill patients (i.e., patients with severe sepsis and septic shock) presenting with hypotension and myocardial depression, combined with coagulopathy [76–78]. The aforementioned strategy has been investigated in a prospective study by Kafka-Ritsch et al. [76]. All patients ($N=51$; Hinchey III ($n=40$, 78 %) and IV ($n=11$, 22 %)) were initially managed with limited resection,

lavage, and temporary abdominal closure followed by second, reconstructive operation 24–48 h later. Bowel continuity was restored in 38 (84%) patients, of which four were protected by a loop ileostomy. Five anastomotic leaks (13%) were encountered requiring loop ileostomy in two patients or Hartmann's procedure in the remaining three patients. The overall mortality rate was 9.8% and 35/46 (76%) of the surviving patients left the hospital with reconstructed colon continuity. Fascial closure was achieved in all patients.

In patients presenting with diffuse peritonitis and diffuse fluid at the CT scan, the pivotal roles in deciding the strategy are the condition of the patient, comorbidities, and presence or absence of distant free air. In fact if the patient presents with no comorbidities, in stable condition with free fluid but no evidence of distant free air, the possibility to attempt a laparoscopic lavage and drainage still exists with the alternative of surgical resection in case of failure. However, if the conditions of the patients are unstable, if comorbidities coexist, or if there is evidence of distant free air, the laparotomy becomes mandatory. The decision to perform or not a direct anastomosis, once again, depends on the comorbidities and on the clinical conditions of the patient (Fig. 19.1).

19.4.6 Antimicrobial Therapy

Antimicrobial therapy plays an important role in the management of complicated AD and it's typically an empiric antibiotic treatment. The empirically designed antimicrobial regimen depends on the underlying severity of infection, the pathogens presumed to be involved, and the risk factors indicative of major resistance patterns [79]. Several recommendations have been recently published in intra-abdominal infections [38, 79]. However, consideration of local epidemiological data and regional resistance profiles is essential for antibiotic selection.

Considering intestinal microbiota of large bowel, generally AD requires antimicrobial coverage for gram-positive and gram-negative bacteria, as well as for anaerobes.

Uncomplicated Acute Diverticulitis

Antimicrobial regimens with beta-lactamase-inhibiting antibiotics such as amoxicillin/clavulanic acid are appropriate for community-acquired AD. In case of allergy to penicillin, the association of ciprofloxacin with metronidazole may be administered although high rates of resistance to quinolones have been reported in many countries [38].

In immunocompromised patients antibiotics with a broader spectrum should be used. An appropriate antimicrobial regimen administered for an adequate duration has minimal impact on the emergence of antimicrobial resistance [38].

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Most of them are community-acquired infections. In these and in other forms of intra-abdominal infections, the main resistance threat is posed by extended-spectrum beta-lactamase (ESBL)-producing *Enterobacteriaceae*. These bacteria in fact are increasing in community-acquired infections worldwide [78]. The most significant risk factors for ESBL-producing infection include prior exposure to antibiotics, comorbidities requiring concurrent antibiotic therapy, and chronic-care admissions [79]. Anti-ESBL-producer coverage should be warranted for patients with these risk factors. Empiric coverage for *Enterococcus* species is not recommended in patients with mild or moderate community-acquired intra-abdominal infection [66].

In patients with hospital-acquired infections, an aggressive broader-spectrum antimicrobial therapy is mandatory. Intra-abdominal cultures are always recommended for patients with healthcare-associated infections or with community-acquired

infections at risk for resistant pathogens. In case of complicated AD with concrete risk of ESBL-producer bacteria, larger spectrum antibiotics should be utilized (i.e., ertapenem, tigecycline, piperacillin/tazobactam). Empirical antifungal therapy for *Candida* species would be recommended for patients with nosocomial infections and for critically ill patients with community-acquired infections [38]. An echinocandin regimen would be recommended for critically ill patients with nosocomial infections [38].

Although discontinuation of antimicrobial treatment should be based on clinical and laboratory criteria such as fever and markers of inflammation, a period of 5–7 days for adult patients is generally sufficient in patients with AD who have been treated with proper source control and prompt surgical intervention [38, 79].

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Chapter 20

Laparoscopy for Perforated Acute Diverticulitis

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20.1 Introduction

The surgical approach to perforated diverticulitis has radically changed over the last few decades. In fact, during the last century, open Hartmann's procedure has been the undisputed primary procedure for perforated diverticulitis. Recently, however, several studies have compared non-restorative resection with primary anastomosis [6, 16, 18, 21, 23, 25], and interestingly, significant differences have resulted in favor of primary anastomosis in terms of the stoma reversal rate and other complications [12]. In addition, studies have investigated the role of laparoscopic resection and laparoscopic lavage as an alternative to the standard open procedures for the treatment of complicated diverticulitis [4]. Laparoscopy in perforated diverticulitis has emerged early on not only to confirm the diagnosis but also to be therapeutic by allowing the lavage of the peritoneal cavity in cases of purulent peritonitis or the resection with or without primary anastomosis if a perforation is clearly identified.

According to the Hinchey Classification [7], perforated diverticulitis can be separated into stage III or stage IV disease states. Hinchey III diverticulitis occurs when a peridiverticular abscess has ruptured and caused purulent peritonitis [8]. Hinchey IV diverticulitis refers to the presence of a feculent peritonitis due to the rupture of an uninflamed and unobstructed diverticulum into the free peritoneal cavity with fecal contamination [8]. Hinchey stage III and stage IV diverticulitis, the presence of a large inaccessible abscess as well as the lack of improvement or deterioration within 3 days of conservative management, are both well-accepted indications for emergency operative treatment [8]. Currently, different surgically valid and laparoscopically feasible options exist in cases of Hinchey III or IV diverticulitis. Hartmann's procedure is still the most preferred and performed worldwide and consists of a two-stage operative approach [12]. During the first operation, the diseased colonic segment is resected, the distal rectal stump is oversewn,

and an end colostomy is performed [8]. Months later, a second operation is then performed in order to reestablish the colonic continuity [8]. However, restoration of bowel continuity is not performed in up to 55% of patients owing to operative risks [23]. Alternatively, resection with primary anastomosis can be performed [1]. A combining diverting loop-end ileostomy should be considered in order to prevent a second operation in case of anastomotic leakage occurrence [17]. All these surgical procedures are validated and widely discussed primary procedural choices in cases of perforated acute diverticulitis, irrespective of the Hinchey stages III and IV. Recently, an increasing number of studies have investigated the possible role of laparoscopic peritoneal lavage for Hinchey III diverticulitis. Clear indications for laparoscopic lavage in the treatment of acute diverticulitis have not yet been accepted [1, 4]. Notwithstanding, current data suggest that laparoscopic lavage could potentially become the definitive treatment for perforated diverticulitis in selected cases [4].

20.2 Laparoscopic Peritoneal Lavage for Hinchey III Diverticulitis

Perforation is a rare complication in cases of acute diverticulitis. The incidence of patients with free perforation during urgent evaluation is estimated to be only 1–2% [8]. Although Hartmann's procedure has been the standard of care for the treatment of perforated acute diverticulitis over the past decades, this procedure is associated with high morbidity and mortality rates along with a high incidence of unreversal stomas [12]. As a resulting operative risk, many patients will never restore bowel continuity. Therefore, laparoscopic peritoneal lavage, first described back in 1996 by O'Sullivan et al. [13], has emerged as a promising alternative to sigmoidectomy in patients with purulent peritonitis

secondary to perforated diverticulitis [2]. As a direct consequence of the improvement in the knowledge of this disease, the rationale for laparoscopic treatment is that Hinchey stage III diverticulitis, determined by the rupture of a large abdominal abscess inside the peritoneal cavity thus causing a diffuse purulent peritonitis, indicates related general symptoms referring to perforated acute diverticulitis. Notwithstanding, a site of diverticular perforation is very often not identified in such cases, and on some occasions, eventual perforations are already sealed when surgery is performed. Allegedly, in such cases, previous clear indications of emergent colonic resection diminish, whereas laparoscopic lavage and drain of the peritoneal cavity from the disseminated purulent material seem to be the only necessary procedures to be performed.

Although early studies showed positive results [2], the effective role of laparoscopic peritoneal lavage is still under review, and some crucial points are raising discussion among authors. To date, only four randomized clinical trials (RCT) have been set with the intent to assess the outcome of laparoscopic peritoneal lavage in comparison to primary resection for perforated acute diverticulitis with purulent peritonitis. One of them is still ongoing and early results are still anticipated [24]. The first to be published is the DILALA trial [2]. It found laparoscopic peritoneal lavage feasible and as safe as Hartmann's procedure for the cure of Hinchey III diverticulitis. A second RCT, the LADIES trial [20], is still underway. It is composed of two groups. The DIVA group compares Hartmann's procedure to sigmoidectomy with primary anastomosis in cases of fecal perforated diverticulitis (Hinchey IV). The LOLA group makes a comparison between laparoscopic peritoneal lavage and sigmoidectomy, with or without primary anastomosis, in cases of purulent perforated diverticulitis (Hinchey III). The LOLA part of the LADIES trial has been prematurely stopped by an independent monitoring board for safety reasons, due to the high rate of reinterventions in the lavage group [22]. Finally, the

SCANDIV trial has reached the same conclusions demonstrating a significantly higher rate of reinterventions in the lavage group [19]. Consequently, laparoscopic peritoneal lavage seems to be affected by an increased 30-day reintervention compared with sigmoidectomy in patients with Hinchey III diverticulitis. The reason for these results might be explained by the failure in discerning Hinchey III from Hinchey IV diverticulitis stages that would require a primary resection. The intraoperative distinction between Hinchey III and IV is not always obvious. Both surgical experience and technical skills are required for meticulous irrigation, for appropriate positioning of drains able to effectively discharge, and for reliably locating a not always obvious and easy-to-find free perforation which would change the case from a Hinchey III to a Hinchey IV. Lack of these abilities reduces the chances of successful outcomes for a non-resectional strategy to almost zero. Therefore, laparoscopic lavage needs careful patient selection and assessment for occult perforations by conducting an initial diagnostic laparoscopy by expert operating surgeons. Moreover, an incorrectly performed procedure may be the cause of iatrogenic tears during the lavage. In fact, laparoscopic lavage must consist only of the exploration of the abdominal cavity for occult perforations, the abundant lavage of all four quadrants with at least 4 L of saline solution, and the final position of a drain in the Douglas's pouch. In this procedure, the sigma should never be mobilized in order to avoid involuntary damage to the inflamed colonic segment. Doing so may lead to a failure of the lavage. Also, mistakes may occur during the positioning of the drain. A passive drain has to be placed in a strategic position in the pelvis and left inside for at least 24 h.

A second disappointing issue concerns the risk of missed colon carcinomas when performing laparoscopic peritoneal lavage [19, 22]. Discerning a colon cancer from acute diverticulitis may be difficult during emergency surgery for suspected perforated diverticulitis. Because resection is not performed in

cases of a laparoscopic peritoneal lavage procedure, a short-term colonoscopy after discharge is recommended as to permit a correct diagnosis of the disease. In some related cases, laparoscopic lavage has been shown to potentially lead to a delay in the diagnosis of primary colon cancer and then to retard its most opportune treatment [19].

Finally, another point of concern regards the need for delayed elective resection for patients undergoing laparoscopic lavage. Even though laparoscopic lavage would have been able to solve the acute phase of diverticulitis in some patients without the necessity of invasive surgery, because of the residual diseased colon, the high risk of a second acute diverticulitis event during their lifetime suggests a colon resection of those patients in elective surgery after the first recovery for perforated acute diverticulitis.

20.3 Laparoscopic Sigmoid Resection for Hinchey III and IV Diverticulitis

Whenever a perforation site is identified, resection of the diseased colon is undoubtedly mandatory. In such cases an open approach has always been regarded as the only possible method. Currently, two alternatives can be considered: non-restorative colectomy or resection with primary anastomosis. Over the past century, the usual intervention has been represented by a two-stage approach, better known as Hartmann's procedure. This process consists of the resection of the inflamed and perforated colon with the creation of a diverting left-sided end colostomy for fecal diversion and drainage of infection during the first procedure. In a second delayed elective operation, the bowel continuity is then restored and the colostomy reversed. This type of intervention is the most performed worldwide and still considered the best, secure

way to treat such a lethal emergency condition. Notwithstanding, non-restorative colectomy carries high morbidity and mortality rates along with a significant percentage of patients not eligible for stoma reversal due to operative risk according to their age or comorbidities [12]. Quite recently, several authors have been proposing resection with primary anastomosis as an effective treatment of choice for Hinchey III or IV diverticulitis [6, 16, 18, 21, 23, 25]. Surprisingly, good results have been shown in favor of primary anastomosis with protective ileostomy as noted in a recent RCT on this subject [14].

In 1991, the first laparoscopic sigmoid colectomy for diverticular disease was reported by Jacobs [9]. Thus far, many studies [4] and a few randomized trials [5, 10, 15] have been published on elective laparoscopic resection for diverticular disease. Despite an increased operative time, laparoscopy has emerged to be feasible and safe as well as associated with fewer postoperative complications and shorter hospital stays as compared to standard open colectomy [4]. Nevertheless, sparse data exists about laparoscopic resection for diverticulitis in an emergency scenario. In this interest, a large retrospective study was conducted from the American College of Surgeons National Surgical Quality Improvement (ACS-NSQIP) database by Mbadiwe et al. [11] This study analyzed a total of 11,981 colonic resections performed for complicated diverticulitis from 2005 to 2009 in 237 hospitals nationwide. Of the total, 1,946 (16%) interventions were performed in emergency conditions, of these emergencies, 138 (7%) underwent a laparoscopic approach, 64 cases (46%) involved a laparoscopic primary anastomosis, and in 74 cases (54%) a laparoscopic colostomy was performed. The remaining 1,808 (93%) open procedures consisted of 334 (18%) resections with primary anastomosis and 1,474 (82%) open colostomies. In an emergency context, laparoscopy emerged to be associated with significantly lower rates of respiratory complications as compared to the standard open approach ($p=0.02$), whereas no differences

were found with regard to overall postoperative complications ($p=0.25$). Although some studies exist on elective surgical resections for the treatment of symptomatic diverticulitis [4], robust data is lacking for laparoscopic concerns in emergencies such as in cases of perforated acute diverticulitis. Therefore, indications for laparoscopic colectomy remain uncertain and not yet widely accepted for Hinchey stages III or IV [1].

Despite the absence of emergency-related data, a growing body of evidence suggests that the laparoscopic approach can carry some benefits to emergency surgery in cases of perforated acute diverticulitis—more so than in terms of postoperative outcomes. In fact, colostomy reversal after a laparoscopic Hartmann's procedure (Figs. 20.1, 20.2, and 20.3) will be much easier due primarily to the absence of tissue adherence (common after open Hartmann's procedure). Furthermore, laparoscopic resections for Hinchey IV diverticulitis (Fig. 20.4) might be considered easier than in cases of Hinchey II or III diverticulitis where typically an abscess involves the surrounding viscera and retroperitoneum (Fig. 20.5). Usually thereafter, a free perforation causes diffuse peritonitis requiring the patient to arrive in surgery within 24 h from the onset of symptoms so that anatomical planes are less inflamed. Therefore, mesenteric dissection may be easier because the mesentery is not as thick and edematous as in Hinchey II or III cases where the mesentery is friable, bleeds easily, and is retracted and stuck to the retroperitoneum and surrounding structures. Also small bowel loops can adhere to the inflamed colon leading to a higher risk of iatrogenic tears due to dense inflammatory adhesions.

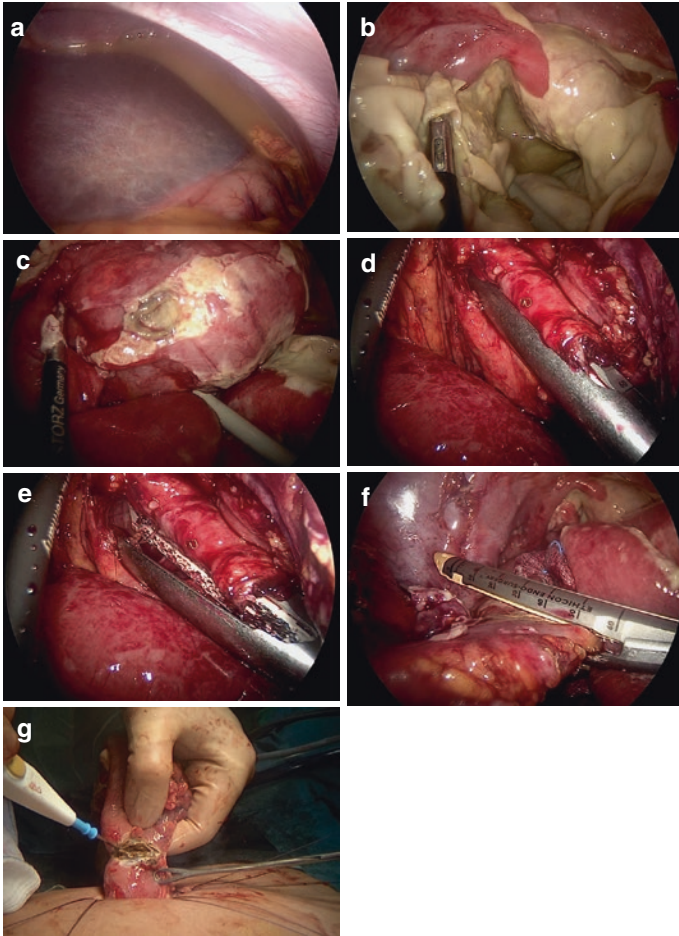


Fig. 20.1 Laparoscopic Hartmann's procedure for perforated Hinchey IV diverticulitis. (a–b) Exploratory laparoscopy showing diffuse peritonitis and fecal free fluid, (c) perforation individuation of the site, (d–e) vascular pedicle resection with an endostapler, (f) stapled sigmoid resection, (g) the sigmoid is extracted from the left flank by enlarging the port to a 4 cm incision; it is then resected after which the colostomy is fashioned on the left flank using the same incision. (Operative pictures provided by Dr. Salomone Di Saverio MD FACS FRCS)

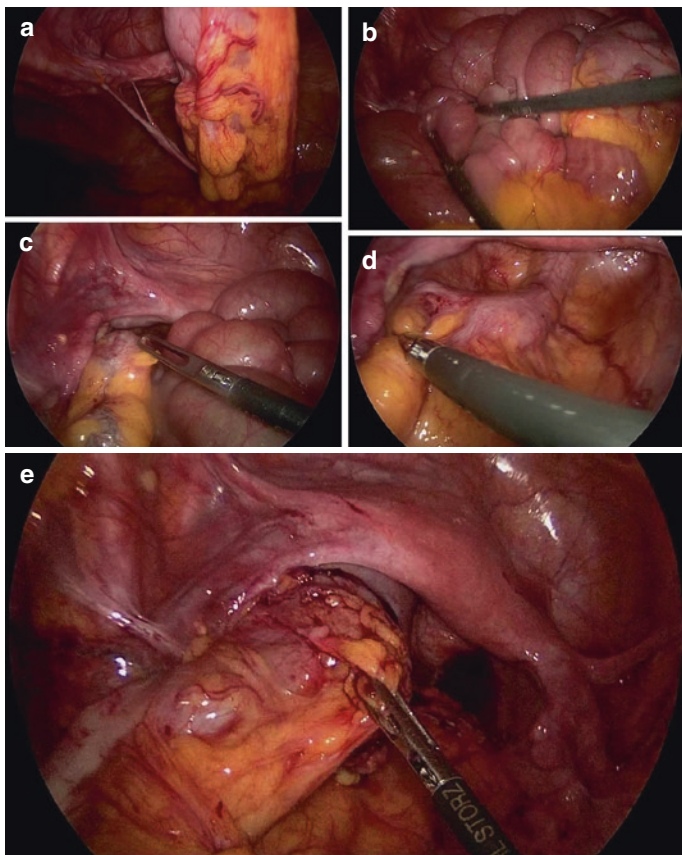


Fig. 20.2 Colostomy reversal after laparoscopic Hartmann's procedure. (a) The diverted colon, (b) exploratory laparoscopy, (c–d) rectal stump, (e) colorectal anastomosis (detail). (Operative pictures provided by Dr. Salomone Di Saverio MD FACS FRCS)

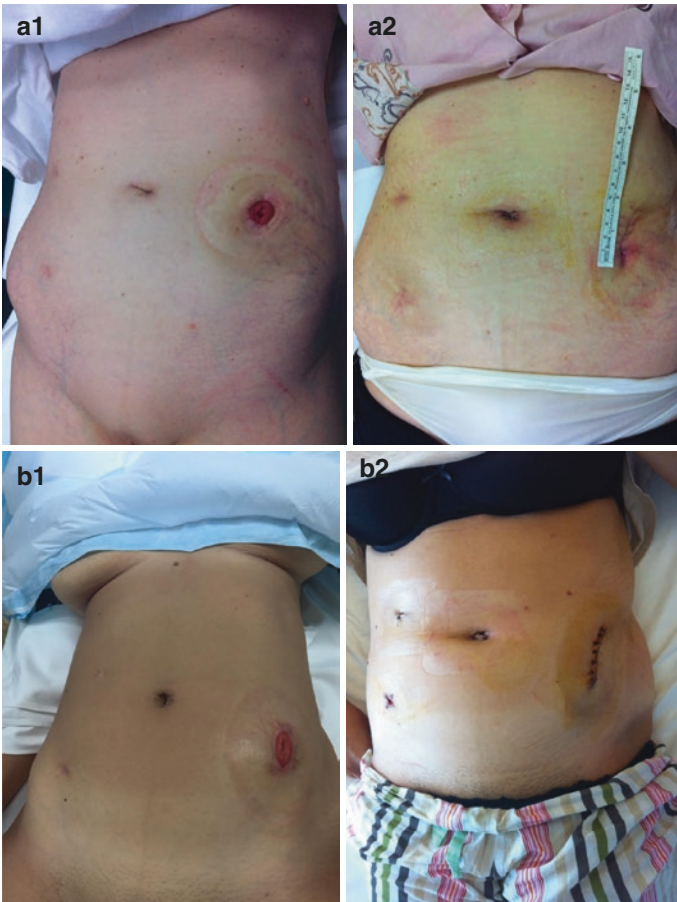


Fig. 20.3 Laparoscopic Hartmann's resection for Hinchey IV diverticulitis and subsequent laparoscopic reversal. Functional and aesthetic outcome. (a1) and (b1) Postop outcomes after Laparoscopic Hartmann for Hinchey IV. (a2) and (b2) Postop outcomes after laparoscopic colostomy reversal (4-6 months later after Hartmann) in the same two patients. (Follow up pictures provided by Dr. Salomone Di Saverio MD FACS FRCS of his own patients)

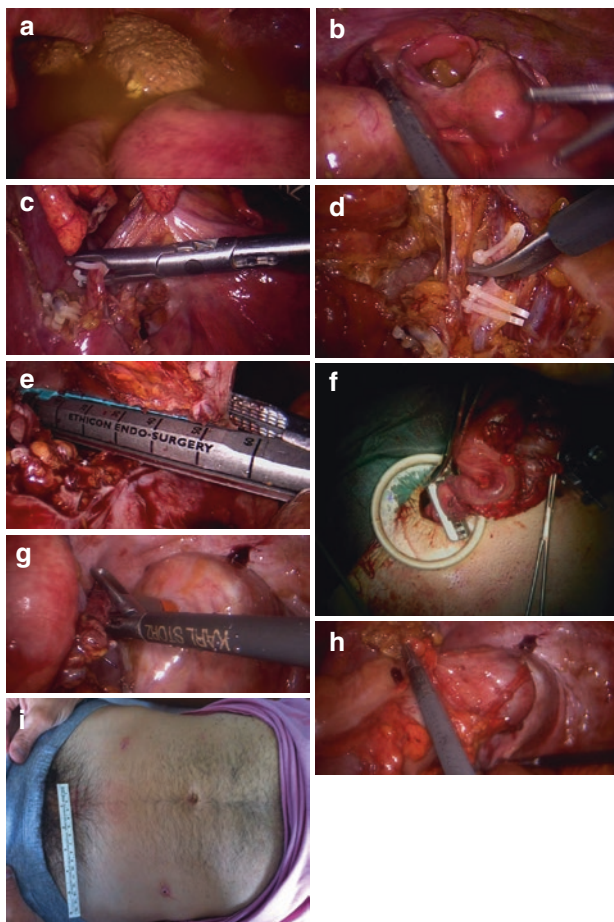


Fig. 20.4 Laparoscopic sigmoid resection with primary anastomosis for Hinchey IV diverticulitis. (**a–b**) Exploratory laparoscopy showing the presence of feculent peritonitis due to the rupture of a diverticulum into the free peritoneal cavity with fecal contamination (**a**) and individuation of the site of perforation (**b**), (**c–d**) positioning Hem-o-Locks for the ligation of the vascular pedicle (**c**) and vascular pedicle resection with scissors (**d**), (**e**) stapled sigmoid resection, (**f**) the sigmoid is extracted through a mini-Pfannenstiel incision and then resected with purse-string forceps, (**g–h**) performing transanal colorectal anastomosis, (**i**) functional and aesthetic outcome. (Operative and follow up pictures provided by Dr. Salomone Di Saverio MD FACS FRCS)

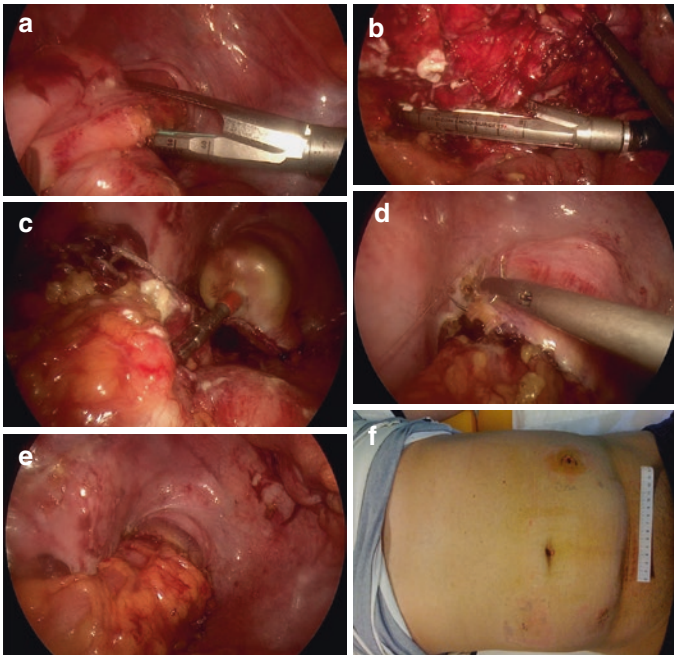


Fig. 20.5 Laparoscopic sigmoid resection with primary anastomosis for Hinchey III diverticulitis. (a) Rectal resection, (b) vascular pedicle resection with an endostapler, (c) performing transanal colorectal anastomosis, (d) oversewing the serous layer with a running suture, (e) colorectal anastomosis, (f) functional and aesthetic outcome. (Operative pictures provided by Dr. Salomone Di Saverio MD FACS FRCS)

20.4 Conclusion

The role of laparoscopy in cases of perforated acute diverticulitis is still debated and remains an important subject of ongoing studies. In cases of Hinchey III diverticulitis, there is some evidence that laparoscopic lavage is feasible as well as safe and effective with the potential advantage of shorter hospital stays as compared to standard open procedures [2]. But more data are still anticipated to substantiate definitive conclusions.

The lack of data regarding laparoscopy for Hinchey IV diverticulitis makes it challenging as to be considered only in selected and hemodynamically stable patients. However, laparoscopic colectomy is likely to be adopted as the standard surgical procedure for complicated diverticulitis when surgeons become more confident with the technique.

Acknowledgment All figures and intraoperative pictures have been provided by Dr. Salomone Di Saverio MD FACS FRCS and belong to his own library of personal surgical procedures performed in emergency colorectal surgery setting.

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Chapter 21

Incarcerated and Strangulated Abdominal Wall Hernias

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Federico Coccolini, and Luca Ansaloni

21.1 Introduction

Incarcerated and strangulated abdominal wall hernias are common problems encountered by surgeons in the acute setting and may be associated with high rate of postoperative complications [1].

Abdominal hernias include both groin hernias (femoral and inguinal) and ventral hernias (umbilical, epigastric, spigelian, and incisional).

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Fig 21.1 Strangulated groin hernia (Picture Courtesy of Dr. Salomone Di Saverio MD): the diagnosis of a strangulated hernia is still mainly based on the clinical exam and inspection of the groin

An incarcerated hernia occurs when the sac contents become constricted such that the contents can no longer be reduced into the abdomen.

In contrast, a strangulated hernia (Fig 21.1) occurs when the blood supply to the contents of the herniated sac becomes compromised.

21.2 Timing of Intervention

Abdominal hernia may become irreducible and cause bowel obstruction; however, strangulation is the most feared complication requiring urgent surgical intervention. Strangulation occurs because of the impairment of the blood supply, leading to venous congestion. Venous congestion impairs arterial blood supply causing necrosis within few hours.

Strangulated hernias can have serious effects such as bacterial translocation to the surrounding tissues making, the surgical field contaminated, and intestinal wall necrosis and gangrene potentially resulting in bowel perforation [2].

Patients should undergo emergency hernia repair immediately when intestinal strangulation is suspected. Early diagnosis of strangulated obstruction may be difficult, and delayed diagnosis can lead to poor outcomes [2]. However, several authors reported that early detection of progression from an incarcerated hernia to a strangulated hernia may be difficult to recognize by either clinical or laboratory means [3–5].

Generally, strangulated hernia is associated with systemic inflammatory response syndrome (SIRS) signs, including fever, tachycardia, leukocytosis, as well as abdominal wall rigidity. Contrast-enhanced CT findings as well as lactate, CPK, and D-dimer levels may be also predictive of bowel strangulation [4, 6–9].

In the case of a strangulated groin hernia, a resection of a segment of the gangrenous bowel and primary anastomosis may be performed through the same incision. However, a midline incision allows to achieve a complete reduction of the hernia sac into the abdomen and to perform a segmental resection of gangrenous bowel with an easy and secure anastomosis (Fig. 21.2).



Fig 21.2 Strangulated groin hernia (Intraoperative picture Courtesy of Dr. Salomone Di Saverio MD) where is clearly visible the hernia sac and the strangulated small bowel loop. The red loop is encircling the spermatic cord

21.3 Treatment

The choice of technique repair is based on the contamination of the surgical field.

Wounds are classified according to the degree of wound contamination during operation [10]. Classification includes:

- Clean wounds
- Clean-contaminated wounds
- Contaminated wounds
- Dirty or infected wounds

Bacteria colonize all surgical wounds, but only in few cases, contaminating bacteria cause a surgical site infection. In most patients, infection does not occur because innate host defenses

are able to eliminate bacteria at the surgical site. Moreover surgeons can minimize the risk of infection and associated complications by routinely administering appropriate antibiotic prophylaxis [2].

However, there is some evidence that the implantation of foreign materials, such as prosthetic mesh, may lead to a decreased threshold for infection [2].

In contaminated and dirty-infected wounds, the use of mesh is generally contraindicated because of the risk of mesh infection.

In clean wounds, the use of mesh is generally recommended [2].

21.3.1 Treatment in Clean Field

Primary suture repair for large hernias can increase the risk of recurrence, thereby leading to subsequent surgery. Numerous studies have demonstrated the reduction of recurrence for incisional hernias treated by mesh repair in elective setting [11–14].

For patients with intestinal incarceration and no signs of intestinal strangulation or concurrent bowel resection, the surgical field is presumed clean and the infectious risk for synthetic mesh is low and synthetic mesh is suggested [2].

21.3.2 Treatment in Clean-Contaminated and Contaminated Field

The use of prosthetic grafts for patients with strangulated hernia has been debated because of the high risk of wound infection.

Studies by Vix et al., Birolini et al., and Geisler et al. reported wound-related morbidity rates of 10.6%, 20%, and 7%,

respectively, with mesh use in both clean-contaminated and contaminated procedures [15–17]. However, these studies did not focus on emergency repair of incarcerated hernias. Kelly et al. reported a 21 % infection rate in a series of emergency and elective incisional hernia repairs [18]. A retrospective multivariate analysis by Nieuwenhuizen et al. showed bowel resection to be a major factor associated with wound infection, but its clinical consequences were relatively low [19]. A retrospective analysis by Zafar et al. on emergency repair of incisional hernia with simultaneous bowel obstruction in potentially contaminated fields demonstrated that the use of permanent prosthetic mesh in these surgeries was associated with high rates of wound infection [20].

In patients with intestinal strangulation and/or concurrent bowel resection, direct suture should be performed when it is possible in the case of a small hernia. Synthetic mesh repair should be performed with caution because of the risk of mesh infection [2].

21.3.3 Treatment in Dirty-Infected Field

The use of biological materials in clinical practice is an innovative method for treating abdominal hernias in contaminated surgical fields. Many retrospective studies have evaluated the role of biological mesh in contaminated fields, but most of these investigations did not focus on emergency repair of incarcerated hernias [21–23]. Although biological mesh in these situations is safe, long-term durability has still not been demonstrated [24]. Biological meshes may be an option in patients with strangulated obstruction and peritonitis by bowel perforation (dirty surgical field) when direct tissue suture is not possible because of a large hernia defect [2] especially in incisional hernias.

Patients with strangulated obstruction and peritonitis caused by bowel perforation are often critically ill due to the development of abdominal sepsis and can benefit of a damage control surgery.

An open abdomen procedure in these patients allows surgeons to abbreviate initial surgery, relook surgery in patients with ongoing sepsis, preventing abdominal compartment syndrome, and delay intestinal anastomosis until the patient is appropriately resuscitated and hemodynamically stable [25].

Following the stabilization of the patient, surgeons should attempt early, definitive closure of the abdomen.

In the event that early definitive fascial closure is not possible, surgeons should resort to progressive closure performed incrementally each time the patient returns for a subsequent procedure. Cross-linked biological meshes may be considered an option in abdominal wall reconstruction. Other options when definitive fascial closure is not possible could be skin-only closure and subsequent management of the eventration with deferred abdominal closure with synthetic meshes after hospital discharge [2].

21.4 Laparoscopic Repair

Prospective studies [26–29] have focused on the laparoscopic approach to hernia repair in an elective setting. By contrast, few studies have focused on the laparoscopic approach to hernia repair in an emergency setting [2]. Laparoscopic repair of incarcerated hernia is a feasible procedure with acceptable results; however, its efficacy needs to be studied further, ideally with larger, multicenter randomized controlled trials [30].

21.5 Conclusion

Strangulated abdominal hernia is one of the most common surgical emergencies dealt with by surgeons worldwide.

Patients should undergo emergency hernia repair immediately when intestinal strangulation is suspected. However, early diagnosis of strangulated obstruction may be difficult, and delayed diagnosis can lead to poor outcomes.

In patients with intestinal strangulation and/or concurrent bowel resection, synthetic mesh repair should be performed with caution because of the risk of mesh infection.

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Chapter 22

Acute Appendicitis: Diagnosis and Nonoperative Management

Roland E. Andersson

Patients with suspicion of acute appendicitis are common, especially among children and young adults and in men. A hospital that serves a population of 100,000 inhabitants will see about 500 patients every year with suspicion of appendicitis. Eventually about 100 of them will be treated for appendicitis. How these patients are diagnosed and managed is controversial and varies considerably. This involves the indication for admission for observation, extent and methods of diagnostic workup, role of diagnostic imaging, indications for abdominal exploration and nonoperative treatment and use of surgical method.

22.1 What is Appendicitis?

Appendicitis is an inflammation of the appendix. An invasion of neutrophils into the muscular layer is needed for the true

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diagnosis of appendicitis, but clinically non-important inflammatory changes are commonly seen in the appendix and may falsely be classified as acute, early or mucosal appendicitis.

What triggers this inflammation is not well understood. Mechanical obstruction of the lumen by a faecalith, lymphoid hyperplasia or kinking is definitely one cause, often associated with perforation, but cannot be demonstrated in most cases. Epidemiological findings suggest that appendicitis is associated with disturbances in the immune system.

The inflammation may give vascular thrombosis, tissue necrosis and eventually perforation. The resulting peritonitis may be walled off by the body's defence mechanisms into an abscess or a phlegmon. If the patients' defence mechanisms are insufficient, a life-threatening free peritonitis can also develop.

22.2 Natural History

Understanding the natural history of appendicitis is essential for defining the optimal management. Traditionally it was thought that all untreated appendicitis will eventually perforate. Patients with suspicion of appendicitis were thought to have a ticking bomb that needed to be detected and disarmed as soon as possible. Early exploration on wide indications in order to prevent perforation has therefore been the leading star for the management of patients with suspicion of appendicitis. The high frequency of negative explorations was not a problem. This attitude still influences our thinking.

There are however indications that perforated and nonperforated appendicitis are two different entities, that perforation occur early before the patient reach hospital and that most cases of untreated uncomplicated appendicitis will resolve spontaneously [1, 2].

The increasing proportion of perforations with duration of symptoms is explained by selection – the advanced cases will remain as the others resolve. In-hospital delay will not increase

the risk of perforation or morbidity as most perforations have occurred early before the patients arrive to the hospital [3]. An increased use of sensitive diagnostic instrument will detect more cases of appendicitis that would otherwise resolve undetected [4].

22.2.1 Implications of Early Perforation and Resolving Appendicitis

If most perforations occur early, and some appendicitis will resolve without treatment, we need to differentiate our approach and focus on early identification and treatment of patients with perforation or progressing inflammation, whereas the detection and treatment of simple appendicitis has lower priority. This is the rationale for a management based on a risk stratification.

A marked decrease in the inflammatory response after some hours of observation or absence of inflammatory response in patients with appendicitis diagnosed by imaging may suggest resolving appendicitis even in patients with a highly probable diagnosis of appendicitis. A period with prolonged observation is then indicated.

A large part of the reported efficiency of antibiotic treatment for uncomplicated appendicitis may also be an effect of spontaneous resolution due to the expectant management rather than of the antibiotic treatment.

22.3 Diagnosis

The diagnosis of acute appendicitis is based on elements from the history, symptoms and signs, laboratory examination of systemic inflammatory response and diagnostic imaging. Signs related to gastric upset, peritoneal irritation and inflammatory response are the most important. It is important to understand

that no single symptom, sign, laboratory or even imaging result in isolation can confirm or exclude the diagnosis. The diagnosis is rather made from a synthesis of the whole picture.

22.3.1 History, Signs and Symptoms

Appendicitis usually starts with a feeling of a *gastric upset* with *nausea* or *vomiting* associated with mid-abdominal, dull discomfort. Some patients may think they are constipated. The patient feels diffuse abdominal pain which is usually not very intense. A strong pain may suggest another condition but can be present in patients with an obstruction of the appendix.

Within a few hours, the pain shifts to the right lower quadrant (RLQ) and becomes localised. This *migration of pain* is the consequence of the local peritoneal irritation as the inflammation in the appendix becomes transmural. Most of the physical findings, like *indirect tenderness*, *direct* and *indirect rebound tenderness* and *guarding*, and the pain on cough or movements are related to the *peritoneal irritation*. Localised peritoneal irritation is more likely seen in nonperforated appendicitis, whereas strong or generalised rebound tenderness or muscular guarding may indicate perforated appendicitis with free peritonitis. However, the signs of peritoneal irritation may be weak or even absent in patients with a retrocaecal appendix or when the appendix is located low in the pelvis. Right-sided rectal tenderness has very low or no diagnostic value in appendicitis but may be useful to detect a pelvic abscess (Table 22.1).

22.3.2 Systemic Inflammation

A systemic inflammation, with fever, leucocytosis, increase in the proportion of neutrophils and the C-reactive protein (CRP) concentration, comes within some hours after the debut of

Table 22.1 Predictive power of elements of history and clinical examination in the diagnosis of appendicitis, expressed as pooled likelihood ratios

Variable	LR+	95 % CI	LR-	95 % CI
<i>Patient details and disease history</i>				
Age \geq 20 years	1.25	(1.10–1.42)	0.74	(0.62–0.89)
Male sex	1.62	(1.49–1.76)	0.62	(0.57–0.68)
<i>Symptoms</i>				
<i>Gastrointestinal dysfunction</i>				
Vomiting	1.63	(1.45–1.84)	0.75	(0.69–0.80)
<i>Pain</i>				
Pain migration	2.06	(1.63–2.60)	0.52	(0.40–0.69)
<i>Signs</i>				
<i>Tenderness</i>				
Indirect tenderness	2.47	(1.38–4.43)	0.71	(0.65–0.77)
Rectal tenderness	1.03	(0.83–1.27)	0.96	(0.85–1.08)
Psoas sign	2.31	(1.36–3.91)	0.85	(0.76–0.95)
<i>Peritonism</i>				
Rebound tenderness	1.99	(1.61–2.45)	0.39	(0.32–0.48)
Percussion tenderness	2.86	(1.95–4.21)	0.49	(0.37–0.63)
Guarding	2.48	(1.60–3.84)	0.57	(0.48–0.68)

From Andersson [5]

appendicitis. It is stronger in patients with perforated appendicitis. The diagnostic value of these tests is very important and should always be included in the diagnostic workup of patients with suspicion of appendicitis (Table 22.2).

22.3.3 Diagnostic Imaging

Graded compression ultrasound (US), computerised tomography (CT) and magnetic resonance imaging (MRI) are increasingly used in the diagnostic workup in patients with clinical suspicion of appendicitis. The sensitivity and specificity for

Table 22.2 Predictive power of laboratory variables and body temperature in the diagnosis of appendicitis, expressed as pooled likelihood ratios

Variable	LR+	95 % CI	LR-	95 % CI
WBC count ($\times 10^9/l$)				
≥ 10	2.47	(2.06–2.95)	0.26	(0.18–0.36)
≥ 15	3.47	(1.55–7.77)	0.81	(0.69–0.95)
Neutrophil count ($\times 10^9/l$)				
≥ 7	1.64	(0.87–3.09)	0.31	(0.23–0.40)
≥ 9	2.66	(1.39–5.09)	0.45	(0.37–0.54)
≥ 11	4.36	(2.83–6.73)	0.60	(0.53–0.69)
≥ 13	7.09	(4.06–12.37)	0.74	(0.68–0.81)
Proportion of neutrophils (%)				
>75	2.44	(1.60–3.74)	0.24	(0.11–0.50)
>85	3.82	(2.86–5.08)	0.58	(0.51–0.66)
CRP concentration (mg/l)				
>10	1.97	(1.58–2.45)	0.32	(0.20–0.51)
>20	2.39	(1.67–3.41)	0.47	(0.28–0.81)
Body temperature ($^{\circ}C$)				
>38.5	1.87	(0.66–5.32)	0.89	(0.71–1.12)

appendicitis is 0.94 and 0.95, with CT, and 0.86 and 0.81 with US, respectively [6]. US performs best in patients with low BMI. MRI is mainly used in children and pregnant women. In recent studies it has shown similar diagnostic properties as CT.

The particular findings associated with the appendicitis diagnosis at US and CT are presented in Table 22.3. Recent reviews of the individual diagnostic properties of these elements suggest that some of them need to be revised, especially the cut-off for the diameter suggesting appendicitis at >6 mm has a very low specificity in many studies. The presence of one single diagnostic criterion should also not be considered diagnostic for acute appendicitis.

CT has become the favoured imaging modality due to the higher diagnostic accuracy. Routine imaging is however not

Table 22.3 Diagnostic characteristics for appendicitis found at graded compression ultrasound and computerised tomography

Characteristics found at graded compression ultrasound

Aperistaltic, noncompressible, dilated appendix (>6 mm diameter)

Distinct appendiceal wall layers

Target appearance (axial section)

Presence of appendicolith

Periappendiceal fluid collection

Echogenic prominent pericaecal fat

Confirming that the structure visualised is the appendix is clearly essential and requires demonstration of it being blind ending and arising from the base of the caecum

Characteristics found at CT

Dilated appendix with distended lumen (>6 mm diameter)

Thickened and contrast-enhanced wall

Thickening of the caecal apex: caecal bar sign, arrowhead sign

Periappendiceal inflammation, including stranding of the adjacent fat and thickening of the lateroconal fascia or mesoappendix

Extraluminal fluid

Inflammatory phlegmon

Abscess formation

Appendicolith

more efficient than traditional clinical management and selective imaging. Imaging as a screening instrument, in patients with low probability of appendicitis, or for confirming a very likely clinical diagnosis, will produce high rates of false positive and false negative diagnoses, respectively (Fig. 22.1).

Extensive use of imaging may increase the total number of operations due to the detection of patients that would otherwise go undetected to spontaneous resolution. [4] There is also an increasing concern for the potential harm from ionising radiation and the side effects of the contrast media, motivating efforts for a more selective and varied pathway with score-based risk stratification and selective, staged US and CT.

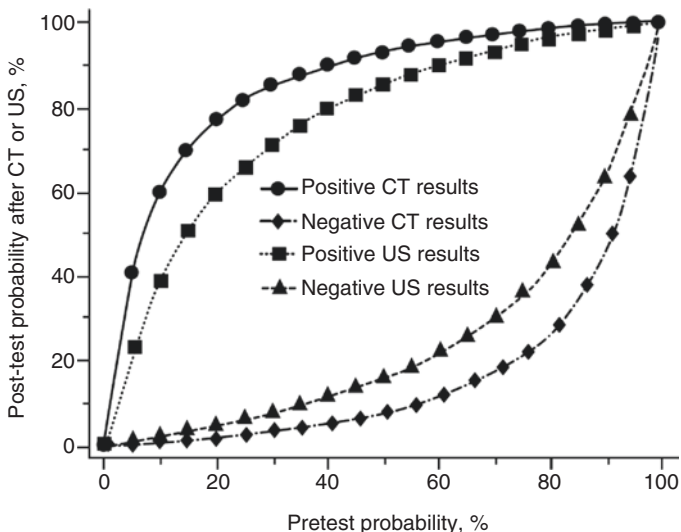


Fig. 22.1 Post-test probability of acute appendicitis after computed tomography (CT) or ultrasound (US). Post-test probabilities are shown as a function of pretest probability for patients with positive results on CT, positive results on US, negative results on CT and negative results on US (From [6])

22.3.4 Clinical Score

The clinical diagnosis is a complicated and subjective process dependent on the surgeon's knowledge and previous experience of similar cases. Pain and tenderness are often given too much attention and the inflammatory response too little [7].

There is no single symptom, sign, laboratory test or imaging result that can confirm or exclude the diagnosis in isolation. The diagnosis is a synthesis of the whole picture. A clinical score can make this process more objective and has an inbuilt experience as it is based on a large number of

Table 22.4 Comparison of the Alvarado and AIR score

Diagnosis	Score	
	Alvarado	AIR
Vomiting		1
Nausea or vomiting	1	
Anorexia	1	
Pain in RLQ	2	1
Migration of pain to the RLQ	1	
Rebound tenderness or muscular defence	1	
Light		1
Medium		2
Strong		3
Body temperature >37.5 °C	1	
Body temperature >38.5 °C		1
Leucocytosis shift	1	
Proportion of neutrophils		
70%–84%		1
≥85%		2
Leucocytes		
>10.0×10 ⁹ /L	2	
(10.0–14.9)×10 ⁹ /L		1
≥15.0×10 ⁹ /L		2
C-reactive protein concentration		
10–49 mg/L		1
≥50 mg/L		2
Total score	10	12

The Alvarado score: sum 0–4=not likely appendicitis, sum 5–6=equivocal, sum 7–8=probably appendicitis, sum 9–10=highly likely appendicitis

Appendicitis inflammatory response score: sum 0–4=low probability, sum 5–8=indeterminate, sum 9–12=high probability

RLQ indicates right lower quadrant

patients. The Alvarado score is the most well-known and widely used clinical score. The more recent Appendicitis Inflammatory Response (AIR) score has better diagnostic properties [8, 9] (Table 22.4).

22.3.4.1 Impact of Time and Repeat Examination

The disease, its clinical presentation and the systemic response in appendicitis are dynamic. Perforation often occurs early, within some hours after the debut of symptoms. In nonperforated appendicitis, resolution may come within a few days. In patients with duration of symptoms of more than 3–4 days, perforated appendicitis is therefore the more likely differential diagnosis, sometimes associated with an abscess or phlegmon.

The inflammatory response comes with a delay, especially for the CRP, where the increase in serum concentration may come after 24–48 h, whereas the WBC count and the proportion of neutrophils may respond within hours. A high WBC count and proportion of neutrophils should therefore be given more attention than a low CRP in a patient with short duration of symptoms. An increase in CRP at a repeat examination can indicate worsening of the inflammatory state but can also only reflect the situation the day before. An increase in CRP is therefore less important if associated with a concomitant marked decrease in the other inflammatory markers. The change in body temperature comes somewhere between in time. Appendicitis is less likely if fever preceded the abdominal pain. The diagnostic value of the elements in the clinical diagnosis increases with duration of symptom. This motivates *repeat clinical and laboratory examination* after some hours of observation in patients with an unclear diagnosis [10].

22.3.5 Diagnostic Laparoscopy

Diagnostic laparoscopy is the final diagnostic instrument. It is an invasive method and should not replace conventional clinical diagnosis and imaging, but has a role in patients with an equivocal

clinical diagnosis where the patient's presentation is such that a condition needing surgical treatment cannot, or has not, been ruled out by the repeated clinical diagnosis with or without imaging. A macroscopically uninflamed appendix does not need to be excised but can safely be left in situ. As a consequence of this praxis, the meaning of the "negative appendectomy rate" has changed. Diagnostic laparoscopy for suspicion of appendicitis where no cause for the abdominal pain is found and the uninflamed appendix is left in situ is still a non-productive abdominal exploration which is associated with unnecessary pain, costs and risks and should not replace noninvasive diagnostic modalities. A non-productive abdominal exploration rate over 10–15% is not acceptable for open surgery and the same standard should be applied to diagnostic laparoscopy.

22.4 Putting It All Together Through a Structured Management

Patients with suspicion of appendicitis are heterogeneous – from the unaffected patient with mild symptoms to the septic patient with generalised peritonitis. We cannot use one single approach for them all but need to differentiate the management depending on the clinical presentation, the probability of appendicitis and if there are indications suggesting perforation or not. Age, comorbidity and duration of symptoms also play a role.

22.4.1 Risk Stratification and Algorithm

A risk stratification, based on the AIR score, is a simple tool to select patients for the different arms in the algorithm (Fig. 22.2).

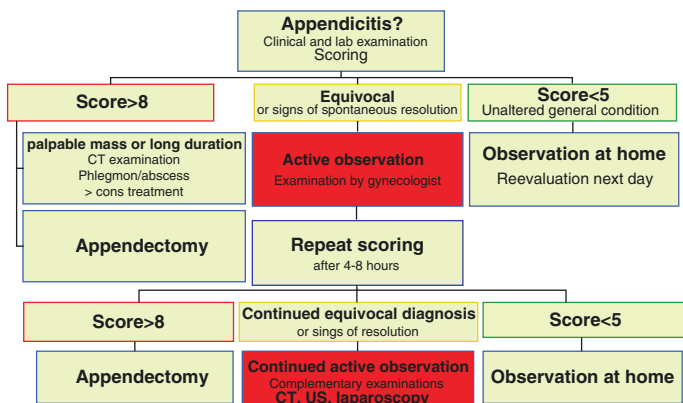


Fig. 22.2 Algorithm for a structured management of patients with suspicion of appendicitis, based on risk stratification using the AIR score

22.4.1.1 Low Probability

An AIR score <5, e.g. a patient with abdominal pain but no alarm symptoms, no signs of peritonism and no inflammatory response, does not exclude appendicitis completely, but there is a very low probability of advanced appendicitis needing emergent appendectomy. An imaging study may only lead to a false positive examination due to the low prevalence of appendicitis or detect a mild appendicitis that may resolve without treatment within a few hours. Most of these patients can be observed at home and return for planned reexamination after some hours if not improved.

22.4.1.2 High Probability

A patient with strong clinical suspicion of appendicitis, e.g. a young man with a history of vomiting, the presence of rebound

or guarding and strong inflammatory response (AIR score >8), has a high probability of appendicitis and needs to be considered for surgical treatment. Imaging may not give any useful information as a negative study cannot rule out appendicitis. Before sending such a patient for imaging, you may ask yourself what your action would be if the imaging turned out negative. You would probably need to do at least a diagnostic laparoscopy anyway.

22.4.1.3 Indeterminate Probability

If the clinical presentation is indeterminate (AIR score 5–8), a planned rescoring after 6–8 h will improve the diagnostic accuracy. Patients with acute appendicitis will have a stronger inflammatory response, and if it is just nonspecific abdominal pain and sometimes also in simple appendicitis, the “attack” will resolve, which is first manifested by a significant decrease in the inflammatory response and then a decrease in other symptoms. Patients rarely perforate under surgical observation. If the diagnosis still remains unclear after re-evaluation, an imaging study or diagnostic laparoscopy may be indicated.

22.4.2 *The Atypical Patient*

Small children, the fragile elderly and patients with atypical presentation (>3-day duration of symptoms, recurrent episodes of abdominal pain, a palpable mass or patients where the intensity of pain does not correspond to the weak clinical or laboratory findings) need special considerations. Imaging may here be indicated to detect an appendiceal phlegmon or abscess, Crohn’s disease, diverticulitis, tumour, strangulated ileus, torsion of the ovary, ureteral calculi or other differential diagnoses.

22.5 Treatment: Surgery or Antibiotics?

In patients with perforation and free-floating abdominal pus, source control by the excision of the appendix and aspiration of pus followed by minimum 5-day antibiotic treatment covering both aerobe and anaerobic bacteria is the standard of care.

Surgical treatment is also the standard for nonperforated appendicitis, but nonoperative treatment with antibiotics has been proposed with estimated efficiency of about 60% to over 80% [11]. Unfortunately, most of these studies have deficiencies in design which decrease their generalisability.

It is evident that some patients with nonperforated appendicitis may heal without surgery, but it is not clear if this is the effect of the antibiotics or the result of spontaneous resolution.

Nonoperative treatment with antibiotics is indicated for treatment of perforated appendicitis with localised abscess or phlegmon as surgical treatment may be difficult and lead to high morbidity [12]. It may also be tried in fragile, high-risk patient for general anaesthesia, like a patient with severe respiratory or cardiac insufficiency or a recent myocardial infarction. Non-surgical treatment with antibiotics and percutaneous drainage of abscesses >5 cm diameter is effective in over 90%. An elective appendectomy is not indicated as the risk of recurrence is <10%. Patients aged >40 years should have a follow-up with colonoscopy or CT colonography because of an increased risk of an underlying malignancy.

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Chapter 23

Management of Complicated Appendicitis: Percutaneous Drainage and Interval Appendectomy or Immediate Surgery? Open or Laparoscopic Surgery?

Kazuhide Matsushima, Kenji Inaba,
and Demetrios Demetriades

23.1 Complicated Appendicitis: Epidemiology, Definitions, and Risk Factors

Patients with acute appendicitis can present at different stages of the disease process, ranging from mild mucosal inflammation to frank perforation with abscess formation. The reported overall incidence of acute appendicitis varies with age, gender, and geographical differences [1, 2]. Interestingly, while the incidence of non-perforated appendicitis in the United States decreased between 1970 and 2004, no significant decline in the rate of perforated appendicitis was observed despite the increasing use of computed tomography (CT) and fewer negative

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appendectomies [3]. Of 32,683 appendectomies sampled from the American College of Surgeons National Surgical Quality Improvement Program (ACS-NSQIP) hospitals between 2005 and 2008, 5,405 patients (16.5%) had a preoperative diagnosis of acute appendicitis with peritonitis/abscess [4].

The definition of complicated appendicitis varies slightly in the literature [5–8]. Clinicopathological diagnoses (gangrenous, perforated, appendiceal abscess/phlegmon) of acute appendicitis are commonly used for its definition [9] (Fig. 23.1a–c). Classically, patients at the extremes of age are more likely to present with complicated appendicitis [1, 10]. Similarly, pre-morbid conditions including diabetes and type of medical insurance are significantly associated with the risk of perforation [11, 12]. The importance of early appendectomy has also been emphasized to prevent perforation of the appendix and the subsequent negative impact on patient outcomes [13]. However, more recent meta-analysis data supports the safety of a relatively short (12–24 h) delay before appendectomy, which was not significantly associated with increased rate of complicated appendicitis [14]. Teixeira et al. also showed that the time to appendectomy was not a significant risk factor for perforated appendicitis but did result in a significantly increased risk of surgical site infection [10].

The outcome of patients with complicated appendicitis is significantly worse than patients with uncomplicated appendicitis. A population-based study from Sweden showed that, in a risk-adjusted model, patients with perforated appendicitis were 2.34 times more likely to die after appendectomy than non-perforated appendicitis patients [15]. Because of its higher mortality and morbidity in patients with complicated appendicitis, the management of complicated appendicitis has evolved significantly over the last few decades. As shown in the current treatment algorithm at our institution, the management of complicated appendicitis should be individualized (gangrenous/perforated appendicitis, phlegmon, and appendiceal abscess) to achieve the best patient outcomes (Fig. 23.2).

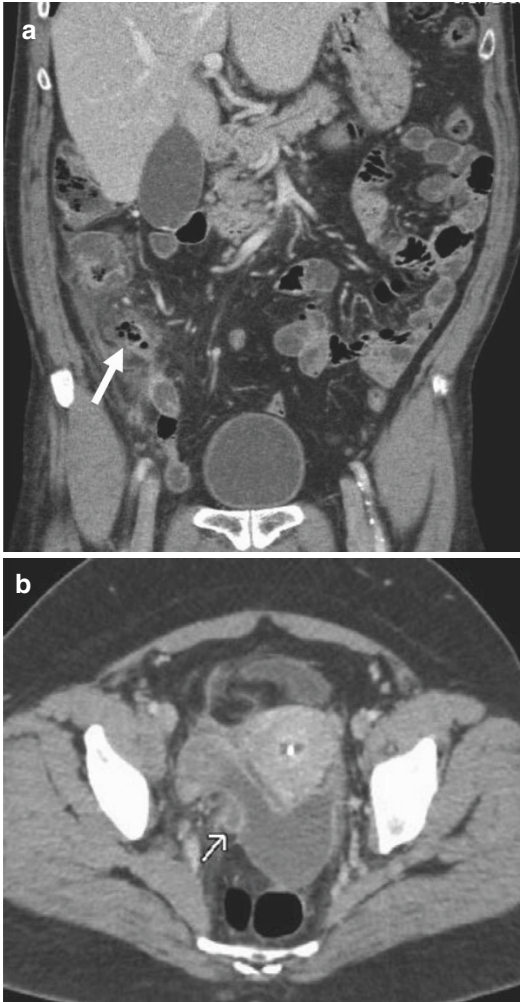


Fig. 23.1 (a) Perforated appendicitis with extraluminal air seen on the abdominal CT (*arrow*) (CT computed tomography), (b) perforated appendicitis with associated abscess formation (*arrow*), (c) acute appendicitis with inflammatory phlegmon (*arrowheads*)

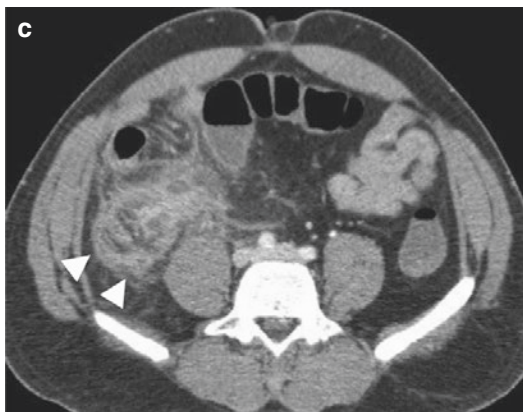


Fig. 23.1 (continued)

23.2 Open Versus Laparoscopic Appendectomy

Since the first laparoscopic appendectomy was described by Semm in 1983, multiple studies have compared operative time, complication rates, length of hospital stay, hospital cost, and other outcomes between open and laparoscopic appendectomy for acute appendicitis [16]. The most recent Cochrane review included 67 studies showing that laparoscopic appendectomy was associated with a lower incidence of wound infection, reduced postoperative pain, shorter postoperative length of hospital stay, and faster recovery to daily activity [17]. In contrast, reduced risk of intra-abdominal abscesses and shorter operative time were found as the advantages of open appendectomy.

Due to increased surgeon experience in uncomplicated appendicitis, laparoscopic appendectomy is more frequently attempted even in complicated appendicitis cases as an alternative approach to open appendectomy [4]. Although the general surgical steps for complicated appendicitis are similar to those for uncomplicated

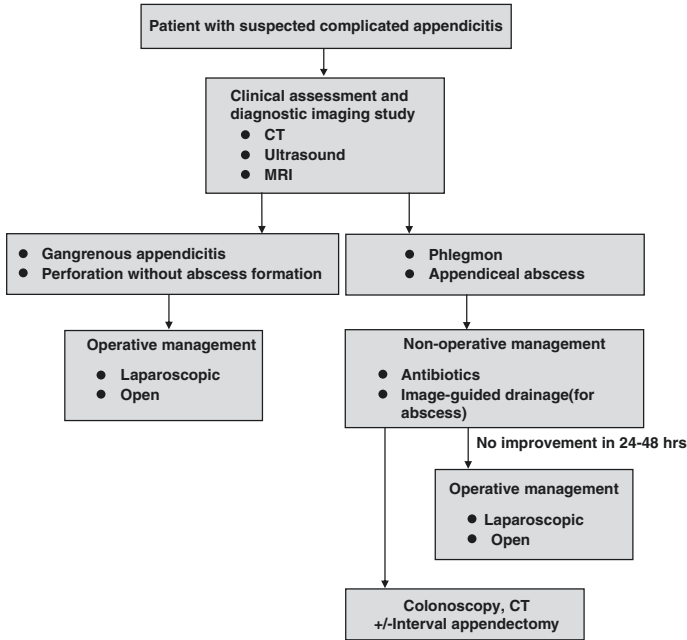


Fig. 23.2 Treatment algorithm for complicated appendicitis at LAC+USC Medical Center (*CT* computed tomography, *MRI* magnetic resonance imaging)

appendicitis, the laparoscopic procedure can be more technically demanding [18]. Therefore, conversion from laparoscopic appendectomy to open appendectomy can be expected [8]. Despite these concerns, the laparoscopic approach in patients with complicated appendicitis has been proven to be safe and comparable to open appendectomy [4–8, 18, 19]. Retrospective studies using a large database in the United States uniformly showed more favorable clinical outcomes (mortality, morbidity, length of hospital stay, readmission rate) and hospital costs in patients who underwent laparoscopic appendectomy when compared to open

appendectomy [4, 5, 19]. The real risk of developing an intra-abdominal abscess after laparoscopic appendectomy remains unclear. A meta-analysis by Markides et al. found no significant difference in the intra-abdominal abscess rate between laparoscopic and open appendectomy for complicated appendicitis, whereas Ingraham et al. showed a higher likelihood of developing an organ-space surgical site infection in patients undergoing laparoscopic appendectomy [4, 6].

23.3 Management of Appendicitis with Phlegmon or Appendiceal Abscess

In previous studies, the proportion of cases with enclosed inflammation (phlegmon or circumscribed abscess) secondary to acute appendicitis ranged from 1.4 to 45.8% [20]. While an emergent surgical intervention is still indicated in patients with gangrenous or perforated appendicitis, primary nonoperative management with antibiotics and image-guided percutaneous drainage of the abscess is more commonly attempted in patients who presented with appendiceal abscess or phlegmon (Fig. 23.3). Immediate surgical intervention used to be considered a treatment option for complicated appendicitis regardless of associated localized abscess or phlegmonous mass formation. However, a recent meta-analysis has shown that an emergent appendectomy in patients with complicated appendicitis, particularly with localized abscess and phlegmonous mass formation, is associated with a significantly higher incidence of complications (odds ratio, 3.95; 95% CI, 1.99–5.44) and unnecessary ileocecectomy [20]. Simillis et al. also showed a reduced risk of complications in patients who received nonoperative treatment compared with acute appendectomy, as well as no significant difference in the duration of hospital stay or intravenous antibiotic days [21].

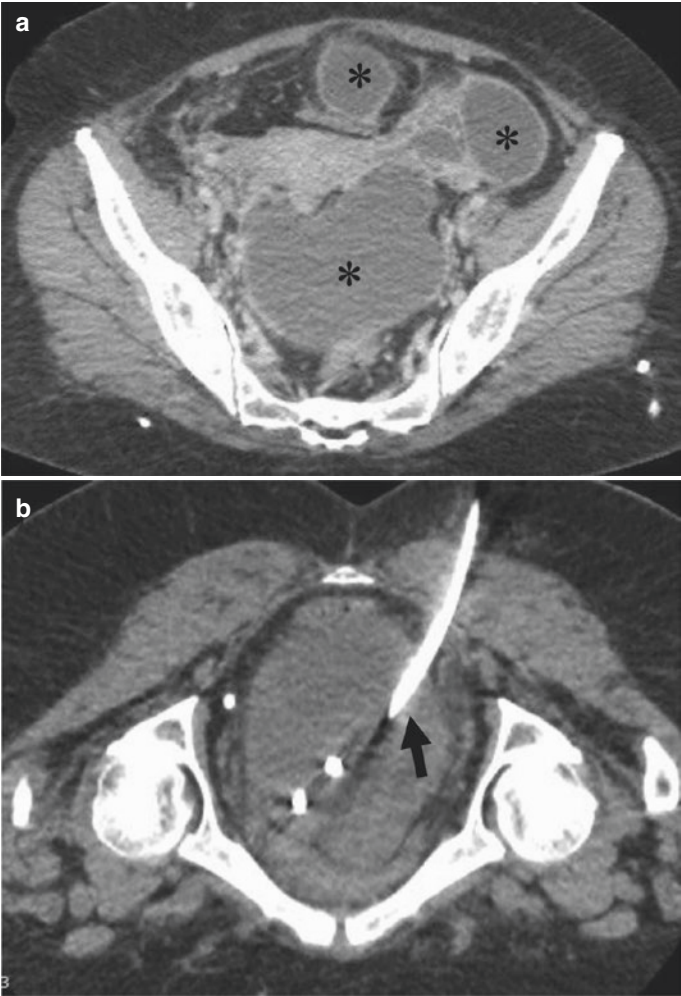


Fig. 23.3 (a, b) Multiple pelvic abscesses (*) were drained percutaneously under CT guidance (arrow: pigtail catheter)

Once nonoperative treatment has been initiated, the patient should be monitored closely for any clinical signs or laboratory data indicative of treatment failure. An urgent consultation by interventional radiology is warranted to discuss percutaneous abscess drainage. In previous studies, there is a significant disparity in the rate of treatment failure (0–55.6%) as well as the use of percutaneous drainage (0–100%) [20]. It remains unknown whether more aggressive use of percutaneous drainage would impact the failure rate. If there is no obvious clinical improvement after 24–48 h of nonoperative management, surgical intervention may still need to be considered.

23.4 Interval Appendectomy After Nonoperative Treatment

In patients who have successfully been managed nonoperatively for acute complicated appendicitis (abscess or phlegmon), the average rate of recurrence has been reported to be 12.4% [22]. Patients with a previous history of appendicitis and calcified appendicolith on CT have a higher likelihood of recurrence [23]. Traditionally, particularly in the pediatric population, the appendix was electively removed to prevent the episodes of recurrence after acute intra-abdominal inflammation subsided in several weeks. However, the necessity of interval appendectomy was recently questioned and remains controversial. To evaluate the necessity of interval appendectomy, Kaminski et al. reviewed the natural history of 864 appendicitis patients initially treated nonoperatively without interval appendectomy [24]. Nearly 80% of the patients had either abscess or peritonitis during their first hospitalization. Over a median of 4 years of observation, 39 (5%) developed recurrent appendicitis. The median length of hospital stay for interval appendectomy was significantly longer

than the one for the admission related to recurrent appendicitis (6 days vs. 4 days, $p=0.006$). Another retrospective study by Willemsen et al. showed that postoperative complications occurred in 17.6% of patients who underwent an interval appendectomy [25].

The potential risk of underlying pathology in addition to acute appendicitis, such as cancer or inflammatory bowel disease, should be ruled out after nonoperative treatment for complicated appendicitis. Cumulative data suggest that malignancy will be found in 1.2% of patients [20]. Despite the recent advances in diagnostic imaging modalities, accurate diagnosis of any underlying disease process can be extremely difficult in patients with an inflammatory appendiceal mass [26]. Patients may therefore require delayed investigations with colonoscopy or repeat CT if they are at risk of malignancy or inflammatory bowel disease. The role of interval appendectomy for this purpose remains unknown.

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Chapter 24

Laparoscopy, SILS, and NOTES for Acute Appendicitis

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24.1 Introduction

In today's world of medical procedure, laparoscopic appendectomy (LA) is well accepted as the gold standard of care in cases of acute appendicitis [15, 23]. Many studies demonstrate LA to be feasible, safe, and effective when compared to the traditional open appendectomy (OA) alternative [18, 19, 26, 27, 32, 33]. These studies show LA to be superior to OA due to shortened hospital stays, lower complication rates, earlier returns to work, and resumption of normal activity [31, 43].

Single-incision laparoscopic surgery (SILS) has recently become a preferred procedure in many centers and has attracted the attention of surgeons worldwide. Growing acceptance of this technique greatly augments its surgical impact. Early data for SILS appendectomy show promising results that are nearly the same as standard LA [17, 29]. However, the increasing costs for SILS and the use of bent instruments requiring advanced laparoscopic skills are still the major disadvantages that limit this practice [7, 41].

More recently, an innovative and challenging technique, natural orifice transluminal endoscopic surgery (NOTES), has been described with the innovative possibility of performing scarless surgery. Since this intervention is performed through natural orifices such as the stomach, rectum, vagina, or urinary bladder, without exterior abdominal scars, this technique may quickly revolutionize surgical procedures.

24.2 Laparoscopic Appendectomy

Laparoscopic appendectomy (LA) was first described back in 1983 [40]. Today, LA represents state-of-the-art care in cases of acute appendicitis worldwide [15, 23]. Innumerable studies have shown that LA has emerged to be feasible, safe, and effective when compared to the traditional open appendectomy (OA) alternative [18, 19, 26, 27, 32, 33]. Major advantages of

LA over OA are based upon superior visualization of the peritoneal cavity, enabling the diagnosis of alternative diseases in cases of normal appendices. The corresponding absence of visible entry ports and scar tissue results in cosmetic benefits as well. Nonetheless, its predominance over OA has not yet been reached, and skepticism still exists among surgeons with regard to the presumed increased risk of intra-abdominal collection [12, 20, 28]. More than one theory has been advanced trying to explain the early findings of higher rates of intra-abdominal abscess recorded for LA. Many ascribe to the way the stump is managed and its manipulation inside the peritoneal cavity [22]. Also, another of the most prevalent hypotheses is that the formation of abscesses is facilitated by exposure inside the peritoneal cavity of the extraverted stump mucosa that is, conversely, divided and oversewn outside the cavity during conventional OA [3]. A randomized control trial comparing open appendectomy with or without introversion of the stump did not find any differences in terms of incidence of intra-abdominal abscess [8].

LA has been associated with a longer operation time [10, 14, 21, 34]. Notwithstanding, operative times depend on surgical experience and laparoscopic skills which demonstrate an indirect correlation between surgical time and an improved learning curve [4].

Even though recent trials show LA to be superior to OA due to shortened hospital stays, lower complication rates, and earlier returns to work/normal activity [18, 19, 26, 27, 32, 33], the higher costs of LA are another criticism and certainly a major consideration for its widespread use thus far.

Contrary to the alleged exorbitant costs of LA, mostly regarding the employment of staplers and disposable devices [11, 30], various accepted and safe options have been described to secure the stump. Metal and nonabsorbable polymeric clips as well as endoloops or intracorporeal knots have been shown to be successful alternative methods [22, 39]. Therefore, costs may vary largely and outcomes can be influenced by both technique and surgical ability.

24.2.1 Surgical Technique

In all surgical procedures, preparation of the patient and all related equipment is of the utmost importance. With the patient in a supine position, arms tucked at the side, the surgeon and the video recording assistant stand on the left side of the patient. To maintain coaxial alignment, the surgeon should be positioned near the patient's left shoulder, and the video monitor facing the surgeon should be placed near the patient's right hip.

The laparoscopic equipment consists of one disposable 10 or 12 mm blunt port and two 5 mm reusable ports. Reusable laparoscopic instruments include a Maryland dissecting forceps with monopolar/bipolar diathermy, atraumatic Johan fenestrated graspers, a hook diathermy, scissors, a reusable irrigation/suction tube, and an endocatch bag to retrieve the appendiceal specimen.

The operation begins with umbilical access achieved using a gasless open technique. The umbilical scar is lifted up and inverted by grasping its deeper apex with a Kocher tooth clamp, and a transumbilical incision is performed to enter the abdominal cavity. The optical trocar is positioned in the umbilicus, and two additional 5 mm trocars are inserted in clear view in the hypogastrium (midline) and in the left iliac fossa (laterally), respectively. Thus, for cosmetic reasons, the three ports should be placed in such a way so that the two 5 mm ports will be below the bikini line. Alternatively, the trocar placed in the hypogastrium port may be inserted in the right iliac fossa or right upper quadrant if preferred. The second and last operative port should be equidistant from the others in order to form a symmetric triangle among the three ports. This will achieve the best triangulation and ergonomics for the operator. A wide examination of the peritoneal cavity is particularly useful in females and in obese patients when diagnosis may be uncertain (Fig. 24.1). After confirmation of diagnosis, the appendix is bluntly isolated from adhesions or fibrin or purulent collection, using a suction tube or atraumatic grasper. The mesentery is coagulated and bluntly

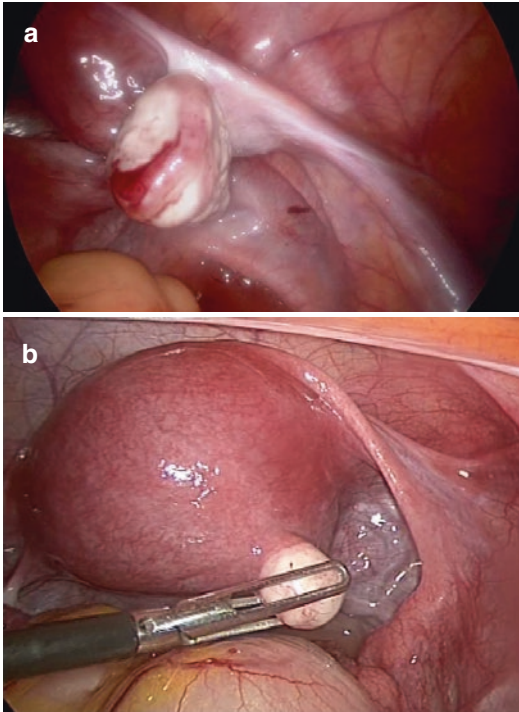


Fig. 24.1 Exploratory laparoscopy in female patients with right iliac fossa pain showing possible alternative diseases in the case of a normal appendix. (a) Hemorrhagic corpus luteum, (b) uterine fibromak, operative and follow up pictures provided by Dr. Salomone Di Saverio MD FACS FRCS

divided by monopolar or bipolar diathermy, connected to a Maryland dissecting forceps (Fig. 24.2). The appendix base is tied with one or maximum two endoloops on the stump (Fig. 24.3). The appendix is then divided between endoloops (Fig. 24.4). The cecal base can be inverted with a laparoscopic purse string if needed (Fig. 24.5). Loops are not recommended in cases of a perforated base appendix or when inflammation has reached the cecum wall. In such cases, a stapler is preferred as a safer method. The specimen is

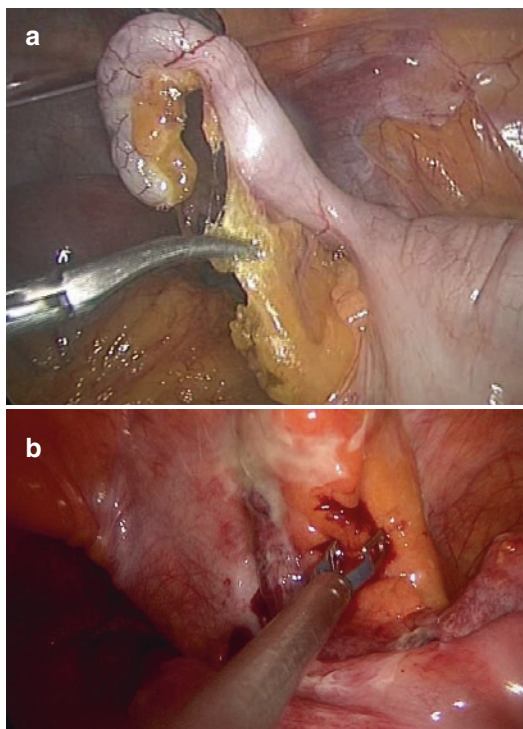
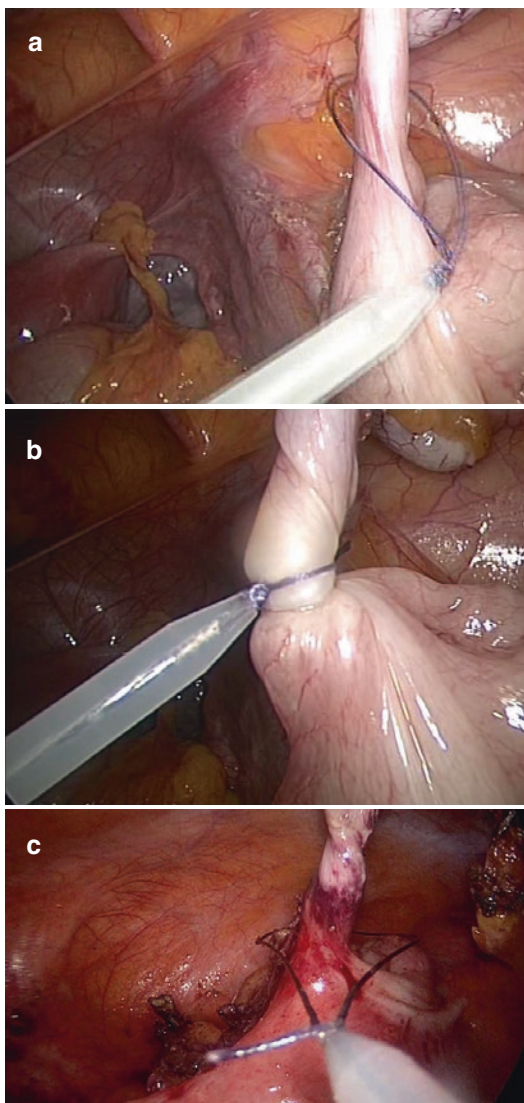


Fig. 24.2 Mesentery coagulation by monopolar (a) and bipolar (b) diathermy. (a) Three-port laparoscopic approach, (b) SILS approach. Note dissection and coagulation should always begin from the mid-mesentery. Also note the absence of triangulation during single-incision procedures. operative and follow up pictures provided by Dr. Salomone Di Saverio MD FACS FRCS

Fig. 24.3 Ligation of the appendix. The endoloop is positioned and tied on the appendix base during three-port laparoscopic (a–b) or SILS appendectomy (c–d), operative and follow up pictures provided by Dr. Salomone Di Saverio MD FACS FRCS



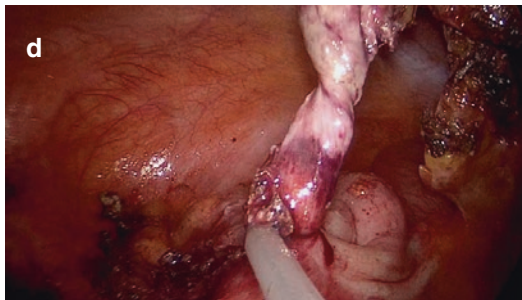


Fig. 24.3 (continued)

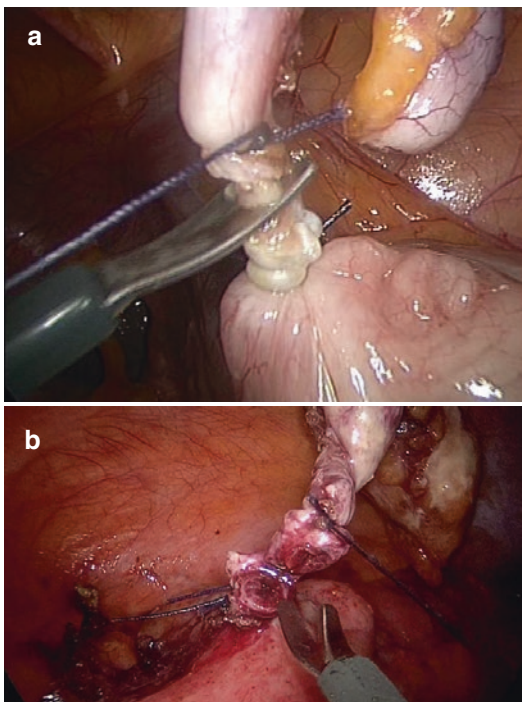


Fig. 24.4 The appendix is divided between endoloops. (a) Three-port laparoscopic approach, (b) SILS approach, (c) appendiceal stump, operative and follow up pictures provided by Dr. Salomone Di Saverio MD FACS FRCS

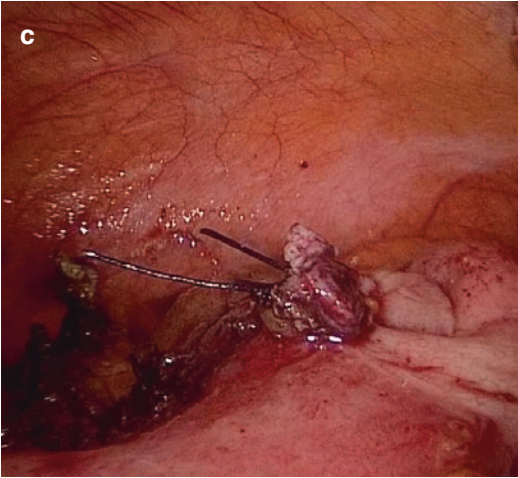


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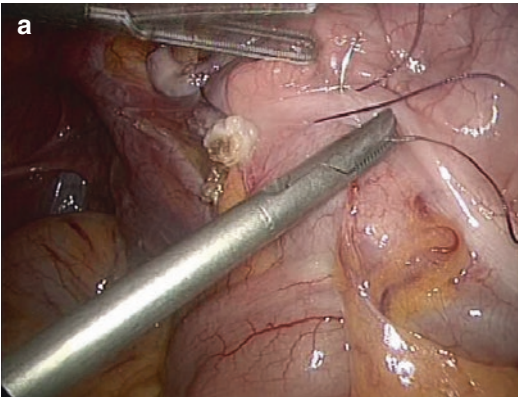


Fig. 24.5 The cecal base is inverted. (a–b) Purse-string suture, (c) appendiceal stump inversion. operative and follow up pictures provided by Dr. Salomone Di Saverio MD FACS FRCS

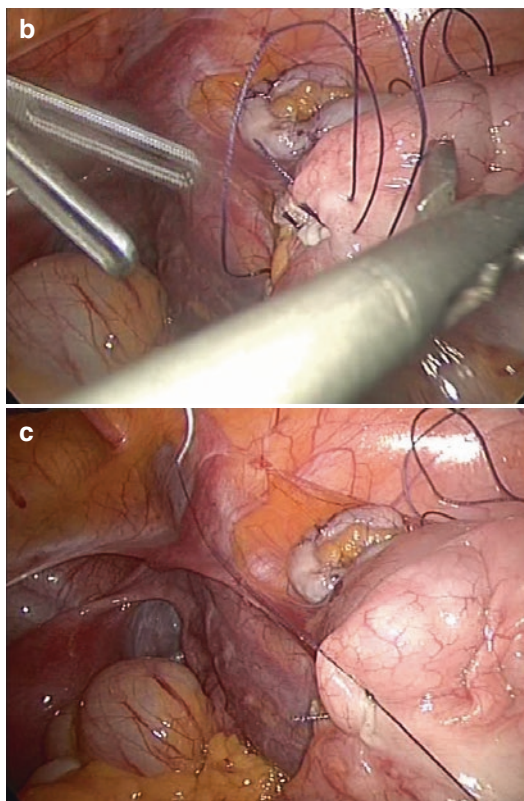


Fig. 24.5 (continued)

retrieved through the umbilical port at the end of the operation preferably using an endocatch bag in cases of gangrenous or purulent appendicitis. Suction and irrigation is recommended for adequate clearance of localized or diffuse peritonitis. Before the extraction of the trocars under direct vision, the insertion of a drain should be considered in cases of complicated appendicitis. Finally, the intervention ends with the closure of the umbilical incision, with absorbable interrupted

stitches for the fascial layer, and the skin sutures corresponding to the laparoscopic accesses.

24.3 SILS Appendectomy

Single-incision laparoscopic surgery (SILS) was first described in 1992 to perform an appendectomy [37]. Thereafter, SILS has been adopted for a vast array of interventions such as cholecystectomies, appendectomies, colorectal resections, and minor liver resections [6]. Through a single incision, usually transumbilical, a multichannel port is applied, and the operation is then performed with curved instruments to permit work within very small operative spaces (Fig. 24.6). A large number of multichannel access ports have recently been introduced differing from each other only in the materials, concepts, the number and



Fig. 24.6 SILS port operative picture provided by Prof. Elio Jovine MD

size of trocar openings, and the incision length required. The main multichannel ports currently are [1]:

The SILS Port (by Covidien, Mansfield, MA, USA) is made of a particular thermoplastic elastomer and consists of four openings, of which three are for 5–12 mm trocars and one is dedicated to insufflation by way of a right-angled tube. Easy to use, this SILS port also accommodates a large variety of laparoscopic instruments and allows for a wide range of maneuverability [1].

In the TriPort (by Advanced Surgical Concepts/Olympus, Co., Wicklow, Republic of Ireland), a sheath is placed through the fascial opening. The peritoneal surface of this sheath has a compressible ring allowing the TriPort to remain inside the peritoneum. The distal portion of the port remains flush with the internal abdominal wall to reduce operative field clutter [1].

The AirSeal (by SurgiQuest, Inc., Orange, CT, USA) uses a pressure barrier created by gas pumped through openings in the housing of the port and allows the use of instruments of any size and shape. Recirculated gas provides improved visibility [1].

The GelPort or GelPoint (by Applied Medical, Rancho Santa Margarita, CA, USA) consists of a combination of a rigid ring with a GelSeal cap and permits the use of standard straight laparoscopic instruments [1].

The Endocone (by Karl Storz GmbH & Co. KG, Tuttlingen, Germany) is a seven-port, multiport bulkhead that can be removed to allow for the removal of organs [1].

The Single-Site Laparoscopy (SSL) Access System (by Ethicon Endo-Surgery, Inc., Cincinnati, OH, USA) is an integrated, low-profile system that obviates the need for trocars and offers a 360° seal cap rotation [1].

The use of curved instruments and the coaxiality due to the absence of triangulation between them associated with the decreased space in which the instruments operate are all factors that not only increase the difficulty level of the operation and correlate with longer surgical times but may also increase the risk of postoperative complications [41].



Fig. 24.7 SILS appendectomy. Functional and aesthetic outcome operative and follow up pictures provided by Dr. Salomone Di Saverio MD FACS FRCS

Because surgical evidence is contained within the umbilicus, transumbilical SILS has the great advantage of leaving no visible exterior abdominal scars (Fig. 24.7). Despite the undisputed cosmetic results related to the reduced number of incisions and trocars, early trials did not provide unanimous opinions regarding postoperative pain and recovery times [9, 24, 36, 41, 42]. In fact, although there is a smaller skin incision in SILS, the total size of fascial defects may be equal to the size required for classic laparoscopy. Also, the instance of postoperative pain may correlate more closely with the inflammatory process around the appendix rather than with the surgical approach [17]. With regard to the length of hospital stay and return to normal activity, SILS recovery time is nearly equal to LA [17, 29] and is, therefore, not a singularly decisive factor for procedural choice.

An important drawback of SILS is the possibility to use a drain. The decision to put a drain at the end of a SILS procedure complicates the advantage of the single umbilical incision and thus requires an additional trocar through which the drain is

inserted. However, the additional trocar can also allow for adequate irrigation of the abdominal cavity in cases of complicated appendicitis with diffuse purulent peritonitis, and therefore it can prevent postoperative intra-abdominal collections [7, 25]. The placement of a drain via umbilicus should be avoided because of the increased risk of wound infection [1, 38].

Although several studies and randomized trials have tested and compared SILS with LA showing similar postoperative results [17, 29], the increasing costs for SILS compared with the already more expensive LA is still the major disadvantage that limits this practice [7, 41]. In this interest, a novel SILS technique modified by the introduction of a single port made of a surgical glove has lately been described by several authors [16, 36], and some benefits are immediately apparent. As commercial expensive triports and proper bent instruments raise the overall costs for SILS procedures in comparison to common multiport laparoscopy, the use of a homemade surgical glove port and standard straight laparoscopic instruments makes SILS equally or less expensive than classic LA [7, 41]. In addition, by improving operating space and reducing coaxiality, the incidence of instrument clash dramatically declines. The implementation of the glove port is very simple (Fig. 24.8). After an umbilical access is provided, a double-ring wound protector is positioned and tightened within the umbilical incision, and a surgical glove (of adequate size to fit the diameter of the external ring) is slipped down onto the external ring in order to get a tight hermetic seal between the wound protector and the glove. Cuts are then made with normal surgical scissors at the tip of the fingers of the glove to provide access for the trocars' cannula. Finally, surgical tape or a tie can be wrapped or knotted around the cannula, and the glove edges to avoid the slippage of the cannula and leakage of CO₂. Although the lack of certification for use may possibly limit its implementation over time, this concept should nonetheless encourage pharmaceutical group concept designers to develop innovative, derivative glove port devices to be contained in kits for SILS procedures to meet surgical needs in a cost-effective way.

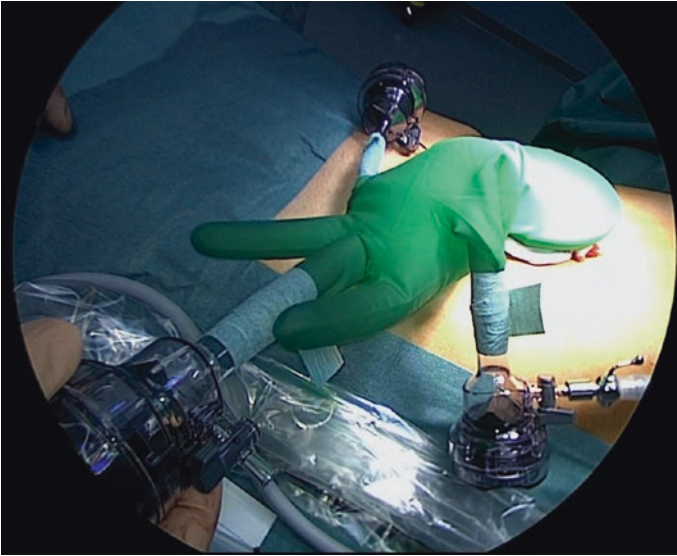


Fig. 24.8 Surgical glove port (intraoperative pic provided by Dr. Salomone Di Saverio MD FACS FRCS)

To date, little data exists on this topic even if the promising early results reported for surgical glove port laparoscopy [13], with its improved feasibility with respect to conventional SILS, might lead to increased use of SILS worldwide.

24.4 NOTES Appendectomy

As with SILS, natural orifice transluminal endoscopic surgery (NOTES) was first described to perform an appendectomy through the stomach [2]. Palanivelu reported the first case of a pure transvaginal NOTES appendectomy [35]. Actually, appendectomies are the second, most frequently performed NOTES procedure after cholecystectomies [5]. The intent of NOTES is to perform abdominal interventions by accessing the cavity through the main natural orifices in order to pursue truly scarless

surgery. This technique thus reduces the overall impact of the surgical process and possibly improves outcomes, especially in terms of pain and recovery time. By introducing an endoscope and instruments (flexible or rigid) through a natural orifice and then entering the abdominal cavity via a hollow organ (such as the stomach, rectus, vagina, or bladder), the choice of the orifice will vary from patient to patient and according to the surgeons' preference. The NOTES procedure can be divided into pure or hybrid NOTES depending on the addition of percutaneous laparoscopy as optical or assistance ports.

The advantage of the transvaginal approach is that it is the easiest entry into the peritoneal cavity and promotes the use of classic laparoscopic rigid instruments. Moreover, the transvaginal approach seems to eliminate the potential risk of causing intestinal fistula [5, 35] thus making the closure of the vaginal wound quite simple. The transgastric or transcolonic routes require dedicated flexible and longer instruments. Also, transgastric NOTES is a more difficult procedure to perform; so high-level endoscopic skills are needed because of the technical difficulties in maneuvering the instruments and the retrieval of the specimen through the esophagus lumen [5]. This procedure is not even comfortable. To its advantage, transgastric NOTES provides a better control of the bacterial load even if the closure of the gastrotomy may be challenging [5]. Consider also that the transcolonic approach provides a more feasible closure of the rectal wound with the transanal microsurgery technique in male patients and serves as a comparable alternative to the transvaginal approach for female patients [5].

At the moment, NOTES appendectomies are still futuristic surgery, performed with consenting and selected patients in a few centers worldwide. So far, only a few cases of NOTES appendectomy reported in literature and scientific data are hereby inadequate to draw preliminary, substantiated conclusions. Further studies are needed to standardize NOTES procedures to prove its safety, feasibility, and effectiveness when compared to other standard mini-invasive surgical techniques.

24.5 Conclusion

LA has superseded OA under all possible clinical/surgical aspects and, despite the slightly higher costs and early skepticism of many surgeons, currently represents the undisputed standard of care for the treatment of acute appendicitis worldwide. By contrast, SILS appendectomies should be considered for the minimally invasive treatment of acute appendicitis in selected cases at centers capable of this technique.

Finally, the NOTES appendectomy is still a limited procedure performed in only few centers with very few cases reported in literature thus far. As the ultimate culmination of minimally invasive surgery, NOTES might soon open a new technological era and definitively change the surgical spectrum.

Acknowledgment All figures and intraoperative pictures have been provided by Dr. Salomone Di Saverio MD FACS FRCS and belong to his own library of personal surgical procedures of laparoscopic appendectomy or SILS appendectomy, performed in acute care surgery setting.

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Chapter 25

Small Bowel Perforations and Small Bowel Miscellaneous Acute Diseases

B. Sakakushev

The small bowel consists of duodenum, jejunum, and ileum, with an average length of usually 6000–7000 mm in vivo having digestive, absorptive, secretory, and immunological functions, the most important of which is processing and absorption of nutrients [1, 2]. It is evidently unique through the sophisticated central but autonomous innervations (“gut-brain axis”), which monitor and integrate gut functions, link emotional and cognitive centers of the brain with peripheral intestinal functions. Small bowel controls immune activation, intestinal permeability, enteric reflex, and enteroendocrine signaling, influencing neuro-endocrine systems associated with stress response, anxiety and memory function. The small bowel has the narrowest indications (intestinal failure) and the lowest sustainability to transplantation, with only 32 small bowel transplants (3.3% of the total) performed from living donor versus 957 from deceased donors [3]. Limited pathological changes are easily curable; extensive ones, like peritonitis

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or segmental necrosis one, can deal with functional consequences, while total small bowel necrosis after acute mesenteric ischemia is unmanageable.

The incidence small bowel perforations is 1 in 300000–350 000/4/. Small bowel perforations can vary from microscopic ones, like fish bone created, to extensive ischemic necrosis induced, leading to an intra-abdominal abscess or total peritonitis, respectively. Clinically they are described as free perforations, causing diffuse peritonitis and/or contained ones, previously covered by adjacent organs [4].

The four basic pathological processes which may result into intestinal perforations are trauma, inflammation, distention, and necrosis. The six clinically important causes of small bowel perforations featured by Roberts in 1957 [5] were recently updated by Vallicelli et al. [6] and Schiessel [7]. For practical use, the clinical classification of Freeman is one of the most relevant, distinguishing two main groups of causes for intestinal perforation – traumatic and those leading to spontaneous perforation [8]. The first group includes blunt abdominal trauma, foreign bodies, and iatrogenic endoscopic and surgical procedures. The second group envisages all the varieties of other reasons, like immune-related and infectious diseases, drugs and biological agents, and congenital, metabolic, and vascular neoplasm.

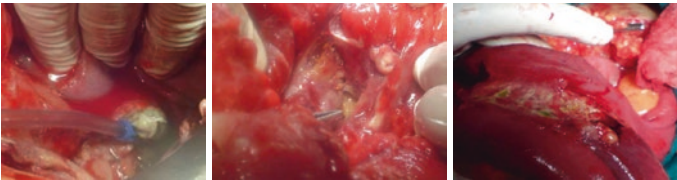
Bowel obstruction is a leading cause of intestinal perforation in industrialized countries, where small bowel obstruction is typically caused by adhesive disease, hernia, or intraluminal mass [9]. Progressing small bowel obstruction leads to proximal bowel dilation, venous outflow obstruction, bowel wall ischemia, and perforation. There are two basic types of small bowel obstruction – dynamic (mechanical) obstruction and adynamic obstruction (paralytic ileus) [10]. Mechanical obstruction can be caused by intraluminal reasons, like foreign bodies (Fig. 25.1), bezoars, dentures, bones [11], and ascaris [12, 13].

The reasons originating from the small bowel wall can be inflammatory or neoplastic. Crohn's disease of the small bowel

[14] and tuberculosis [15] can lead to ileus followed by nontraumatic spontaneous small bowel perforation with local (Figs. 25.2 and 25.3) or diffuse peritonitis (Fig. 25.4).



Fig. 25.1 Foreign body (wooden stick) obstruction (P.S. All images are from the authors' surgical operative practice)



Figs. 25.2, 25.3 and 25.4 Crohn's disease perforation with local (2, 3) and diffuse (4) peritonitis

Small bowel wall neoplasia can rarely lead to obstruction due to benign tumors [16] like lipoma [17] or cysts (Figs. 25.5 and 25.6).

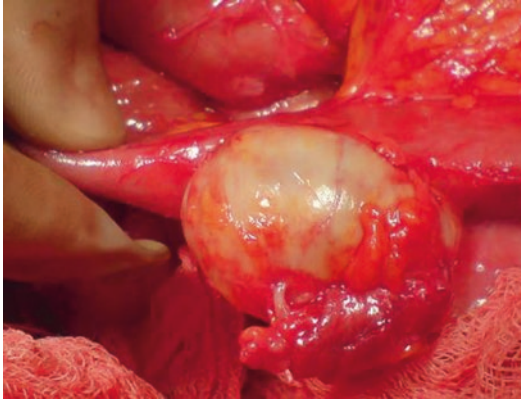


Fig. 25.5 Obstructing simple cyst

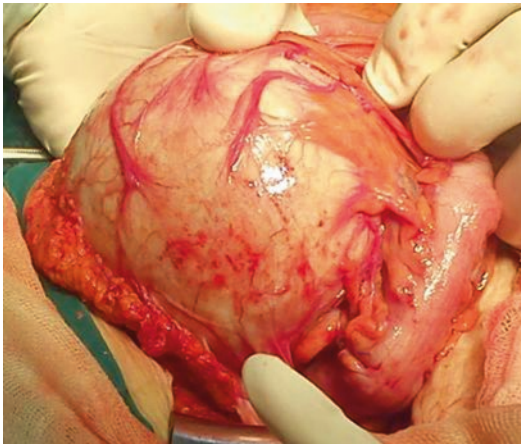


Fig. 25.6 Obstructing mesenteric dermoid cyst

Malignant tumors of the small intestine causing obstruction can be primary or metastatic [18, 19]. Gastrointestinal stromal tumors (GIST) mostly bleed; carcinoids cause obstruction; lymphomas lead to perforation [20]. Gastrointestinal stromal (GIST) tumors make 20% of small bowel neoplasms, causing acute hemorrhage and less commonly obstruction [21] (Figs. 25.7 and 25.8).

Small bowel neuroendocrine tumors (NETs) (Figs. 25.7, 25.8, and 25.9) in the USA have increased by 300–500% in the last 35 years [22, 23].

Malignant fibrous histiocytoma (MFH) usually occurring as trunk sarcomas is extremely rare in the small bowel [24], especially causing obstruction (Fig. 25.10). Primary obstructing small bowel adenocarcinoma (Fig. 25.11) is as rare as infiltrating and obstructing small bowel sigma cancer (Fig. 25.12).

Invagination can be a rare cause of MBO (Fig. 25.13).



Fig. 25.7 Obstructing GIST

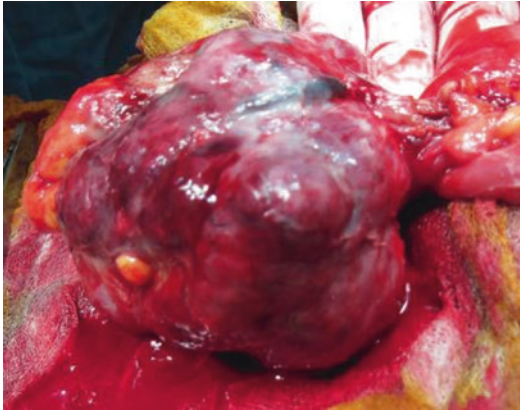


Fig. 25.8 Obstructing twisted GIST



Fig. 25.9 Giant 6 kg obstructing NET

The average rate of metastatic malignant bowel obstruction (MBO) is between 3 and 15% of cancer patients, growing to 20–50% in ovarian cancer and 10–29% in colon cancer (Figs. 25.14 and 25.15).

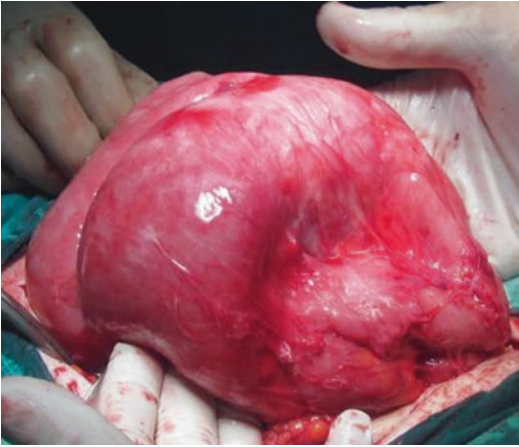


Fig. 25.10 Obstructing histiocytoma

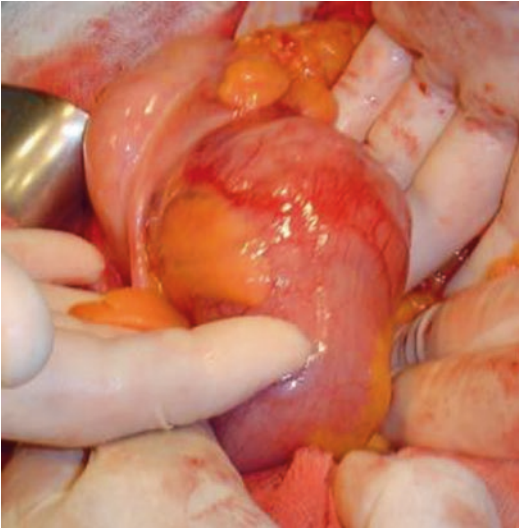


Fig. 25.11 Obstructing adenocarcinoma



Fig. 25.12 Infiltrating and ileum obstructing sigma cancer

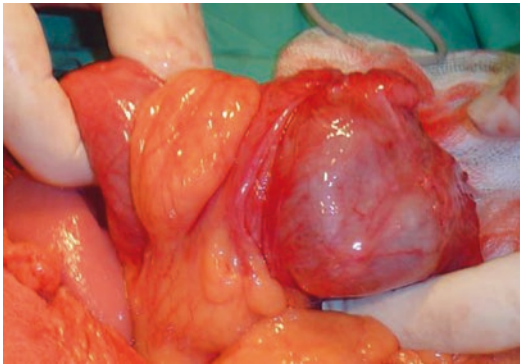


Fig. 25.13 Adult ileocecal cancer invagination

Extra-abdominal cancers most frequently leading to MBO due to peritoneal infiltration are those of the breast (2–3%), melanoma (3%) [25, 26], and the lung [27].

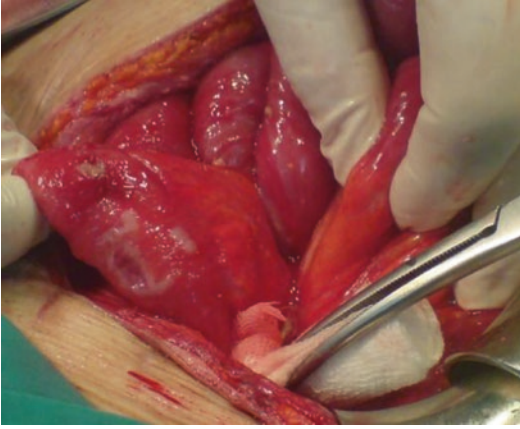


Fig. 25.14 Peritoneal carcinomatosis

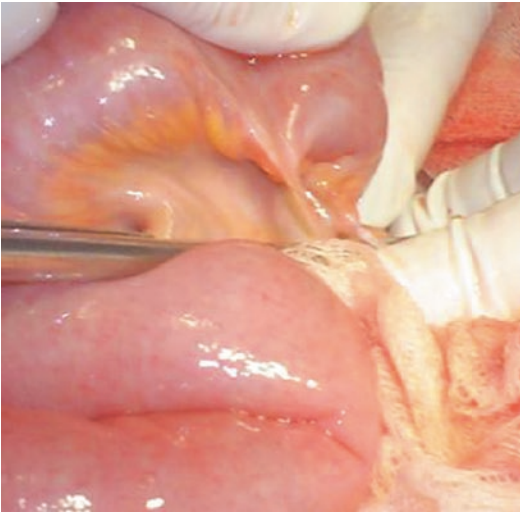


Fig. 25.15 Intestinal string

Extraluminal and extraintestinal intra-abdominal adhesions are the most common causes of SBO reaching 60–70% of all SBO cases [28].

Adhesions can be solitary, like a string (Fig. 25.14), or extensive, causing complete mechanical obstruction (Fig. 25.16), extreme proximal dilatation, and diastatic perforation (Fig. 25.17). Adynamic obstruction (paralytic ileus) (Fig. 25.18)



Fig. 25.16 Adhesive obstruction

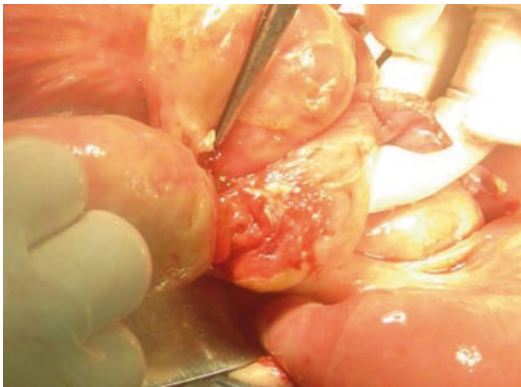


Fig. 25.17 Diastatic adhesive perforation

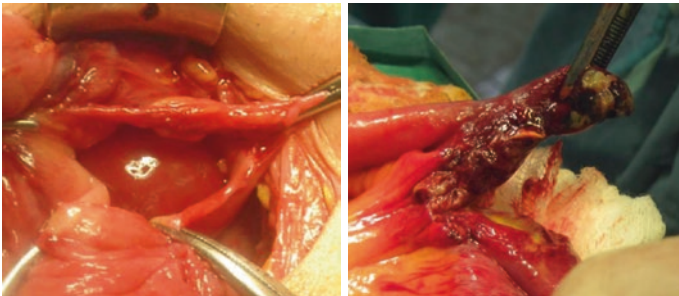
is not rarely a postoperative complication [29], sometimes even requiring re-laparotomy.

Incarcerated inguinal or femoral hernia (Fig. 25.19) is a common cause of intestinal obstruction and perforation (Fig. 25.20). In strangulation, caused mostly by volvulus (Fig. 25.21), the leading pathophysiological noxa is ischemia, leading to fast bowel segmental necrosis and perforation (Fig. 25.22).

Penetrating stab or gunshot (Fig. 25.23) injuries can cause multiple small bowel perforations, requiring many hours of restoration. Stab wounds (Fig. 25.24) are three times more often



Fig. 25.18 Paralytic ileus



Figs. 25.19 and 25.20 Femoral hernia incarceration and perforation



Fig. 25.21 Volvulus

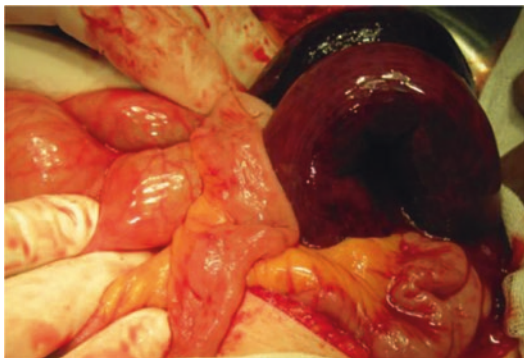


Fig. 25.22 Perforation in volvulus segmental necrosis



Fig. 25.23 Penetrating gunshot injury



Fig. 25.24 Knife stab wound

than gunshot wounds, but approximately 90% of the deaths in penetrating abdominal injury are gunshot related [30].

Non-penetrating injuries usually occur during traffic accidents in combination with other intra-abdominal injuries, such as liver and spleen rupture, which explains the rarity and late diagnosis of isolated small bowel perforation after blunt abdominal trauma [31, 32].

Although most ingested foreign bodies pass through the gastrointestinal tract uneventfully, in 1% of cases they cause bowel perforation, peritonitis, and even death [10]. Clinically unsuspected foreign bodies, causing intestinal perforation, either voluntary or nonvoluntary ingested have been evaluated in multiple series and case reports [33]. Most common foreign bodies causing small bowel perforations are long, hard, and sharp, like fish bones, chicken bones, toothpicks, dentures, and needles magnets [8, 34–36].

Iatrogenic perforations of the gastrointestinal tract related to diagnostic or therapeutic endoscopy are rare but severe adverse events, associated with significant morbidity and mortality. The absolute number of iatrogenic perforations is likely to increase. Acute iatrogenic perforation during endoscopy is defined as the presence of gas or luminal contents outside the gastrointestinal tract [37]. Intraoperative laparoscopic or open surgery small bowel lesions are really considerably more often than reported, though their variety is great.

Perforations can be drain induced (Fig. 25.25), spontaneous in severe peritonitis (Fig. 25.26), persistent peritonitis in open abdomen (Fig. 25.27), severe necrotizing pancreatitis (Fig. 25.28), total postoperative anastomosis dehiscence (Fig. 25.31), and small bowel fistula in frozen abdomen (Fig. 25.32) and due to complete suture takedown (Fig. 25.29) and suture line insufficiency (Fig. 25.30).

Small bowel diverticula are quite frequent (1–5%) or seldom symptomatic, but their complications usually require emergency surgery [38, 39]. The rate of small bowel diverticula on

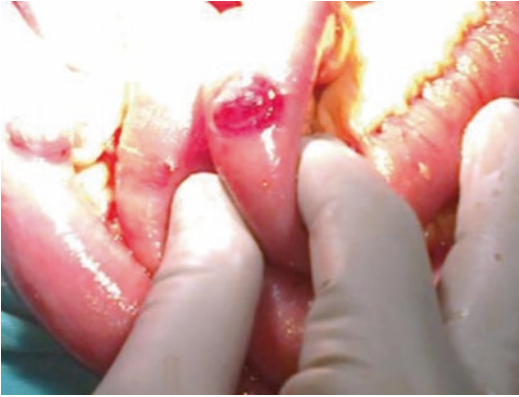
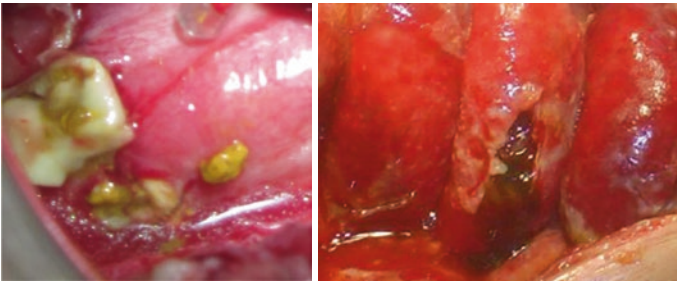


Fig. 25.25 Drain caused perforation



Figs. 25.26 and 25.27 Perforation in peritonitis and open abdomen

autopsy findings is between 0.02 and 4.5 % and on small bowel contrast studies 0.5–2.3 %, where 80 % occur in the jejunum, 15 % in the ileum, and 5 % in both [40–42]. Meckel’s diverticulum is the most common congenital abnormality of the gastrointestinal system (Fig. 25.33) – embryological remnant of the vitellointestinal duct on the antimesenteric surface of the terminal ileum [43, 44]. All other small bowel diverticula are acquired pulsion lesions, in fact false diverticula, containing

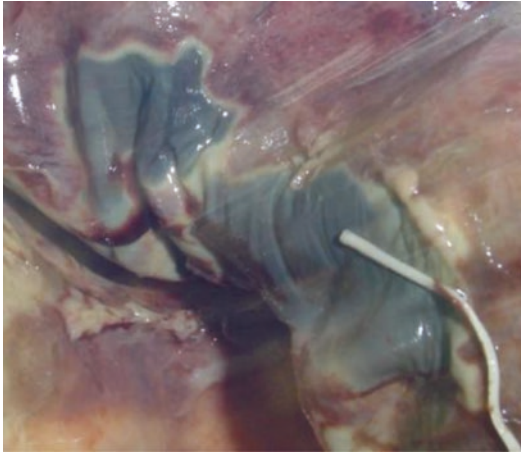


Fig. 25.28 Necrotizing pancreatitis

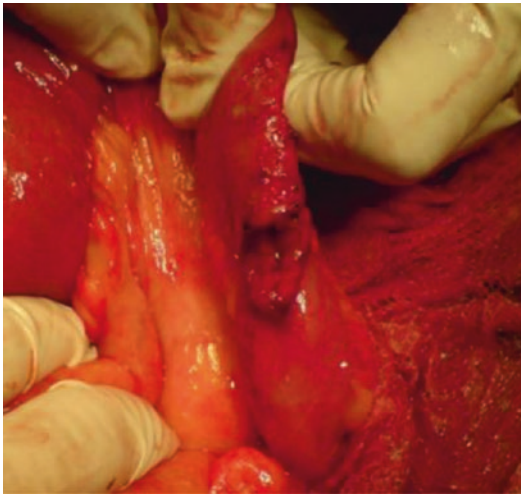


Fig. 25.29 Suture takedown

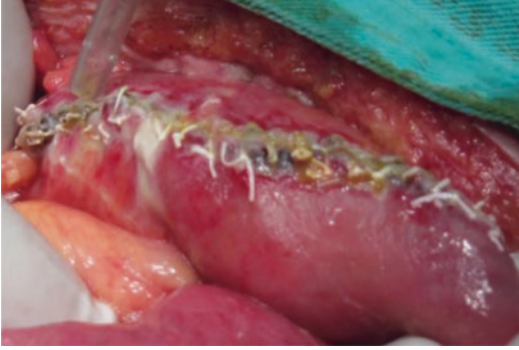


Fig. 25.30 Suture line insufficiency

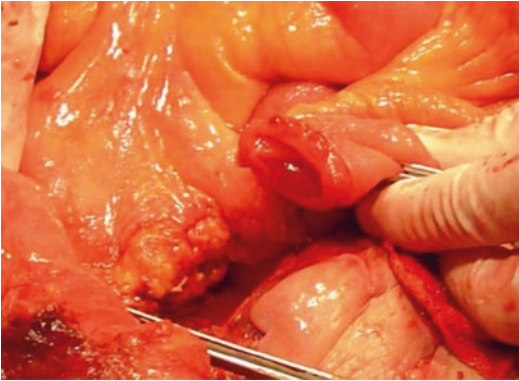


Fig. 25.31 Total anastomosis dehiscence

only mucosa and submucosa [40, 42]. Small bowel perforation rate is recently getting more common than bleeding and infection and carries out high morbidity and mortality especially in elder patients with comorbidities, requiring emergent surgical intervention [44].

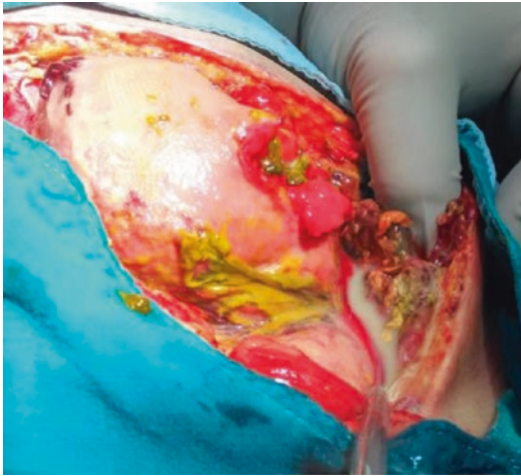


Fig. 25.32 Small bowel fistula in frozen abdomen

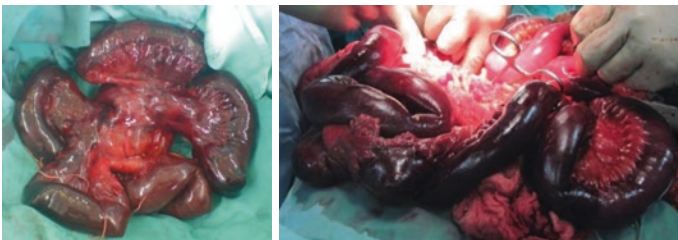


Fig. 25.33 Meckel's diverticulum with ileus

Acute mesenteric ischemia (AMI) is always a case of emergency, where mortality remains 50–80% regardless of the diagnostic and surgical developments from the beginning of this century [45, 46]. AMI occurs mainly in the age over 50, having a 0.1% prevalence of all hospital admissions. Acute mesenteric ischemia includes acute mesenteric arterial embolus and thrombus, mesenteric venous thrombus, and nonocclusive mesenteric ischemia. All these lead to impaired intestinal blood flow, bacterial translocation, and systemic inflammatory response [47]. Infarction and bowel necrosis (Figs. 25.34 and 25.35) in AMI are marked by peritoneal signs, circulatory collapse, and fever. The only most important prognostic factor that can be managed by the surgeon is the time interval between onset of symptoms and surgery [48].

Whatever the cause, the rarity of small bowel perforation combined with its propensity for nonspecific clinical presentation makes establishing the correct diagnosis challenging. Reversely, small bowel perforation is usually clinically presented by the systemic and local symptoms of either local (abscess) or diffuse peritonitis.

Plain radiography for pneumoperitoneum in different positions is usually performed as a first imaging assessment. Sonography may be the initial test chosen in patients with localized abdominal symptoms, when intestinal perforation is not a major clinical consideration [49]. Computer tomography (CT) is the method of choice, being the best radiologic tool



Figs. 25.34 and 25.35 Extensive bowel necrosis in AMI

currently available for revealing both small bowel perforation (sensitivity 92%) and the underlying condition. Localized extraluminal gas, fluid collection, and inflammatory changes adjacent to a thickened bowel segment are the image signs susceptible for small bowel perforation [49, 50]. As a gold standard in the study of small bowel perforations, CT is able to highlight pathological changes in 100% of free perforations and 60% of covered forms [50]. The gold standard for the diagnosis of acute bowel ischemia, according to the consensus statement of SICE and the latest literature articles, is the multi-detector CT angiography (CTA) with sensibility of 93.3% and specificity of 95.9% [51]. The diagnostic accuracy of laparoscopy in trauma has been reported as high as 75%, affording to avoid negative laparotomies in more than 50% [52]. It is generally indicated in both blunt and penetrating abdominal trauma, where discrepancy between clinic and imaging “unclear abdomen” is present [53].

Prompt diagnosis will facilitate correct management planning and a prompt operative procedure [48]. All cases with intestinal perforations are initially stabilized for about 2 h and transferred to the operation room, for immediate surgical intervention, which is an advantage especially for septic emergency patients [54–56]. The operative procedures performed in small bowel perforation are sewing, stapling, or resecting with primary anastomosis or rarely temporary stoma in severe peritonitis or bowel obstruction [56–58]. The first-line surgical treatment of AMI is bowel revascularization [59, 60]. The second step is the reassessment of bowel viability for 20 or 30 min after revascularization before decision making about bowel resection of obvious necrotic bowel should be performed, and after the abdomen closure, the patient should be transported to a vascular surgical center [61]. Even though sporadic reports of conservative, less-invasive, or organ-saving treatment methods for small bowel

perforations are promising, there is yet not enough evidence of their superiority [62–65].

Peritonitis from small bowel perforation is associated with prohibitive morbidity and mortality rates as high as 40% [58]. While an intestinal perforation on its own leads to a mortality of about 14%, a septic clinical progress is associated with an increase in mortality to 30% [54]. The prognosis of AMI even in the best hands is bad, because the outcome is poor. If the diagnosis is missed, the mortality rate is 90%. With treatment, the mortality rate is still 50–80%. Survivors of extensive bowel surgery face a lifetime of disability.

Postoperative complications after surgery for small bowel perforation like ileostomy evagination (Figs. 25.36 and 25.37), wound dehiscence, and evisceration (Fig. 25.38) can be easily managed, while frozen abdomen can create serious problems, as well as late complications like entero-atmospheric fistula (Fig. 25.39).



Fig. 25.36 Terminal ileostomy evagination



Fig. 25.37 Acute double-barrel ileostomy ectropion



Fig. 25.38 Wound dehiscence and evisceration



Fig. 25.39 Small bowel atmospheric fistula

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Chapter 26

Acute Mesenteric Ischemia

Miklosh Bala and Jeffrey Kashuk

26.1 Introduction

The diagnosis and management of patients with acute mesenteric ischemia (AMI) is challenging. Despite recent advancements in diagnostic accuracy and improved care of critically ill patients, the mortality rate for this entity has remained relatively unchanged over the past several decades, primarily due to delays in diagnosis leading to irreversible bowel ischemia and necrosis. Certain recent advances, however, suggest that progress may be evident, primarily via prompt early diagnosis which affords timely treatment of AMI. Endovascular therapy has become an option in patients with an established mesenteric event, especially if diagnosed early. This chapter reviews the classification, etiology, clinical presentation, diagnosis, and treatment of patients with AMI.

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Acute mesenteric arterial ischemia (AMAI) can result from arterial obstruction leading to a number of pathophysiologic conditions via varied clinical presentations [1].

1. *Embolic* occlusion of the superior mesenteric and/or celiac arteries accounts for 40–50 % of AMI [2, 3]. Emboli usually originate in the heart but may come from proximal aortic atherosclerotic lesions as well [4]. Typically, the proximal small bowel is spared, but the remainder of the small intestine and proximal colon are ischemic.
2. *Acute thrombosis* of a principal mesenteric arterial vessel (20–35 % of cases) [5]. Acute thrombosis of the SMA is most often secondary to an underlying proximal atherosclerotic lesion. In this setting, the majority of the small bowel and colon demonstrate ischemic changes, but the extent of necrosis will reflect the state of the collateral circulation.
3. *Nonocclusive* mesenteric ischemia (NOMI). NOMI is typically associated with “low-flow states” and severe mesenteric vasoconstriction. Patients at risk for NOMI include ambulatory patients taking ergot alkaloids or digitalis, the critically ill with vasopressor requirements, and those undergoing dialysis with large volume fluid removal [6]. The most common presentation to the acute care surgeon of this entity is a patient in the intensive care unit who is critically ill, requiring vasopressor support, who develops evidence of increasing acidosis, abdominal pain or distension, inability to tolerate enteral nutrition, or melena.

Acute mesenteric venous thrombosis (AMVT) is a less common event (5–15 %) and is most often related to the presence of an underlying hypercoagulable state [7]. Associated portal and splenic vein thrombosis may be part of the clinical picture.

26.2 Epidemiology and Clinical Presentation

Most patients present in the sixth or seventh decade of life and are more frequently women (60–70%) [8–12]. The classic dictum which has described this condition for over a century is still relevant today: “Severe abdominal pain out of proportion to physical examination findings.” Other patients, particularly those with delayed diagnosis, may present in extremis with acidosis and shock. Nausea and diarrhea occurred in 30–40% of patients and blood per rectum in 16% [5].

Clinical signs of peritonitis may be present early but can be difficult for the novice examiner to detect. Accordingly, one must have a high index of suspicion, because such findings almost always are predictive of intestinal ischemia and bowel infarction. Delaying intervention based upon clinical findings of obvious peritonitis will almost always lead to unacceptably high morbidity and mortality.

There are no laboratory abnormalities totally specific for AMAI. Leukocytosis, lactic acidosis, increased amylase, and liver enzymes levels are the most common findings but are noted late in the course of the acute episode [13–15].

In one study of patients with AMAI, 78% had hypertension, 71% used tobacco, 62% presented with a history of peripheral vascular disease, and 50% had concomitant coronary artery disease [9].

In our experience, patients with embolic occlusion of the SMA often had underlying atrial fibrillation or ventricular arrhythmias. Patients with SMA thrombosis are usually female and have documented chronic abdominal pain and weight loss consistent with chronic occlusion of the mesenteric arteries [16]. Although many patients may have been on chronic anticoagulation due to their known conditions, those presenting with AMAI are often not within therapeutic range or have not been taking their anticoagulants [17]. Some patients presenting with AMAI in our institution

have had concomitant simultaneous thrombosis of other regions, including limb ischemia or splenic artery thrombosis. On this basis, when a patient presents with such complications, careful consideration should be given to AMAI as well.

In the modern era, endovascular manipulation of mesenteric vessels may play a role in the pathophysiology of bowel infarction [18]. Embolic AMAI may occur when atheromatous plaques are dislodged during angiography of the coronary or cerebral circulation [5]. Aortic catheterization can induce cholesterol embolization. Accordingly, unexplained abdominal pain after any invasive procedure, particularly involving vascular manipulation, should lead to suspicion and investigation of AMAI.

Patients with AMVT typically present with abdominal pain out of proportion to physical findings, nausea and vomiting, or bloody diarrhea. The pain may be diffuse or intermittent, lasting for several days or even weeks [19, 20]. AMVT has also been described following almost every major intra-abdominal operation, including appendectomy, bariatric surgery, splenectomy, colectomy, and Nissen fundoplication [21–24]. Polycythemia vera is the most common hypercoagulable disorder associated with this entity [25].

26.3 Diagnosis

The key to early diagnosis is a high index of clinical suspicion. Although laboratory results are not definitive, they may help to corroborate clinical suspicion. More than 90% of patients will have an abnormally elevated leukocyte counts. The second most commonly encountered abnormal finding is metabolic acidosis with elevated lactate level, which occurred in 88% [26].

Perhaps the most important element contributing to early accurate diagnosis currently is a technically appropriate CT examination with accurate radiologic interpretation of images. Newer

generation CT scanners, with faster scanning speeds and higher spatial resolution, facilitate the use of biphasic CT angiography in the diagnostic workup of patients with suspected AMAI.

In this context, biphasic CT includes:

1. Pre-contrast scans to detect vascular calcification, hyperattenuating intravascular thrombus, and intramural hemorrhage.
2. Arterial and venous phases which may demonstrate thrombus in the mesenteric arteries and veins, abnormal enhancement of the bowel wall, and the presence of embolism or infarction of other organs.
3. Sagittal reconstructions are used to assess the origin of the mesenteric arteries [27].

A recent study demonstrated that in 27 of 28 patients (96.4%), MDCT correctly diagnosed AMI (specificity of 97.9%) [28]. A sensitivity of 93%, specificity of 100%, and positive and negative predictive values of 100% and 94%, respectively, were achieved for the CT findings of visceral artery occlusion, intestinal pneumatosis, portomesenteric venous gas, or bowel wall thickening [29, 30] (Fig. 26.1)

Contrast angiography has long been the gold standard for imaging the visceral vessels. This modality can visualize the aorta and the main trunks of the mesenteric vessels and can adequately assess its distal branches. Contrast angiography enables the surgeon to perform selective injection of any of the mesenteric vessels and to perform therapeutic intervention. Despite this, in the current era, these techniques are no longer used for screening due to the modern CT with 3D reconstruction. Accordingly, these techniques are most commonly reserved for use with therapeutic intervention only.

Unexplained abdominal distension or gastrointestinal bleeding may be the only presentation of acute intestinal ischemia in NOMI, and pain may be absent or undetectable in sedated patients in ICU in approximately 25% of cases [31]. Patients

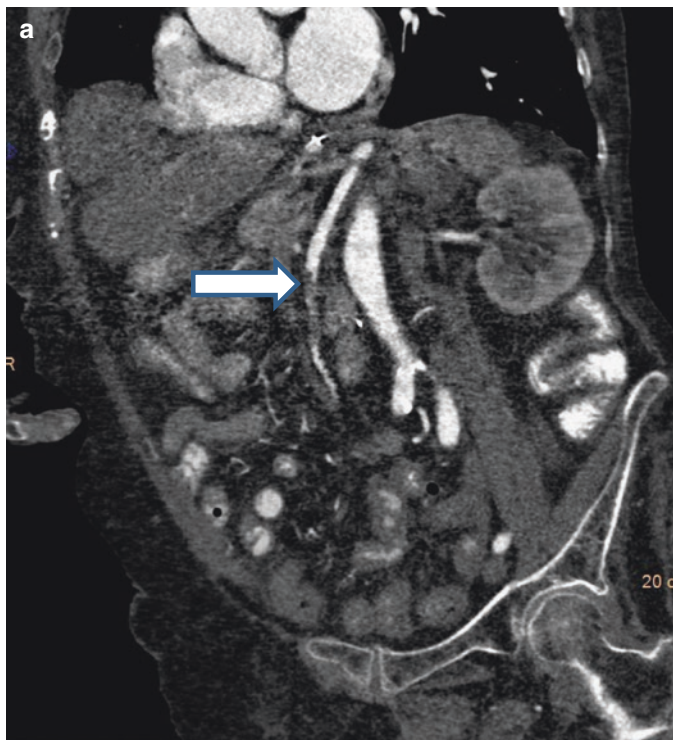


Fig. 26.1 This patient, who had atrial fibrillation and was not receiving anticoagulant therapy, had an acute onset of severe abdominal pain. These selected images are from a CT scan of a patient with acute mesenteric ischemia secondary to occluded SMA from an embolic source (*arrow, a*). Also CT showed ischemic ileum and right colon (*b*); portal venous gas was found (*c*). 3D reconstruction clearly demonstrates distal occlusion of SMA (*d*). At laparotomy, the distal ileum and ascending colon were found to be ischemic and were resected; an SMA thrombectomy (*e*) and vein patch angioplasty was performed

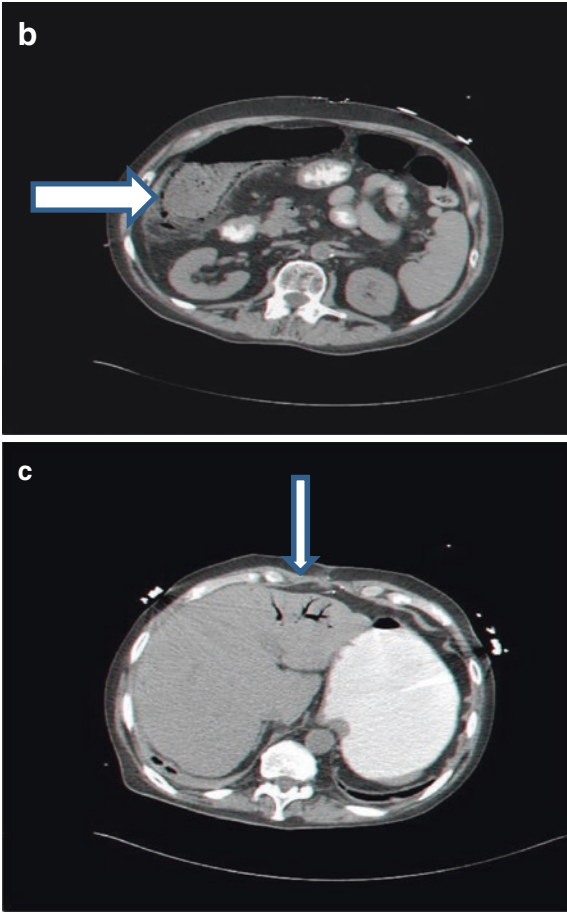


Fig. 26.1 (continued)

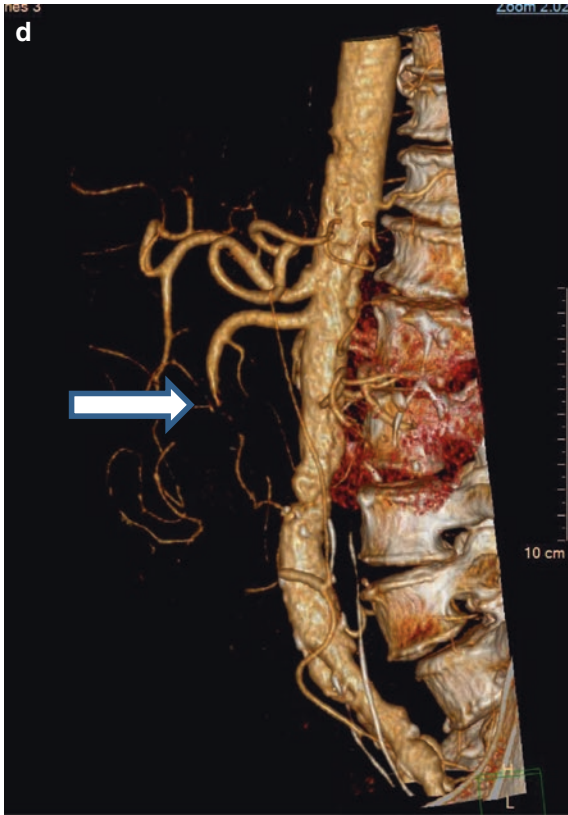


Fig. 26.1 (continued)



Fig. 26.1 (continued)

surviving cardiopulmonary resuscitation who develop bacteremia and diarrhea with or without abdominal pain should be suspected of having NOMI [19]. Right-sided abdominal pain associated with the passage of maroon or bright red blood in the stool suggests the diagnosis of NOMI. Abdominal findings develop slowly and suggest progressive loss of intestinal viability and the presence of transmural gangrene. Nausea, vomiting, hematochezia, hematemesis, massive abdominal distension, and shock are other late signs often indicating compromise of bowel viability. CT is often done and shows radiological signs of bowel ischemia, free fluid, and patent great mesenteric vessels.

In AMVT laboratory tests are not helpful in making the diagnosis—they can neither confirm nor exclude it—and should be

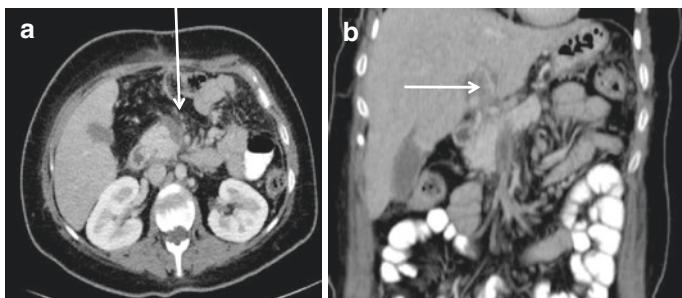


Fig. 26.2 30 Y patient with acute superior mesenteric vein (a) and portal vein thrombosis (b) due to hypercoagulable state. No sign of bowel ischemia was noted. Successfully was treated with long-term anticoagulation

used for screening purposes only. Elevated WBC count is most common. Other biochemical variables such as serum lactate and amylase may be not elevated. Measurement of coagulation factors and laboratory values associated with hypercoagulable states are of value once the diagnosis of AMVT has been confirmed on imaging.

The most common positive finding on venous phase of CTA is the demonstration of thrombus in the superior mesenteric vein (Fig. 26.2). Associated findings that may suggest AMVT include bowel wall thickening, pneumatosis, splenomegaly, and ascites. Portal or mesenteric venous gas strongly suggests the presence of bowel infarction. Duplex ultrasonography can be diagnostic only if obtained early and in chronic cases [32].

26.4 Treatment

Of note: There are no randomized controlled trials to guide treatment, and the published literature contains case reports and often small, retrospective series.

26.4.1 General Considerations

AMI is a surgical emergency. Stable patients with abdominal pain or other risk factors suggestive of AMI should undergo prompt CTA, while the presence of peritonitis or shock mandates prompt abdominal exploration. Untreated AMI leads to mesenteric infarction, intestinal necrosis, an overwhelming inflammatory response, and death.

Fluid resuscitation of the patient with suspected AMI should parallel the diagnostic workup. The main goal of resuscitation is the restoration of adequate tissue/organ perfusion. The use of vasopressors in order to improve cardiac function should be avoided whenever feasible [33]. On the other hand, after adequate volume resuscitation has been accomplished, consideration may be given to the use of drugs such as dobutamine, low-dose dopamine, and milrinone, which have been shown to cause less splanchnic vasoconstriction [34].

Broad-spectrum antibiotics (such as penicillin or a third-generation cephalosporin in combination with metronidazole) should be administered early as bowel ischemia, necrosis, and associated bacterial translocation are frequently noted [35].

26.4.2 Surgical Therapy

The goals of surgery include restoration of SMA blood flow and resection of nonviable bowel [36].

Even in situations where interventional techniques are used to improve or correct arterial occlusion, laparotomy or laparoscopy is mandatory to assess bowel viability and potential resectional therapy. Intraoperative Doppler may be a useful adjunct to assess vascular flow within the mesenteric arcades. Prompt restoration of vascular flow is required when ischemia is

evident, in order to prevent ongoing ischemia, but may not be effective when clear demarcated necrosis is present. The decision to undertake revascularization must be made after careful consideration of the physiologic state of the bowel and the overall clinical condition of the patient.

26.4.3 Damage Control Laparotomy

Damage control laparotomy (abbreviated laparotomy) was introduced for traumatic injuries over 20 years ago and may be an important option in the patient with AMI. This technique should be employed liberally in these patients. Given frequent uncertainty with regard to bowel viability, stapled off bowel ends may be left in discontinuity and reinspected after a period of continued ICU resuscitation to restore physiological balance. These patients often suffer from acidosis, hypothermia, and coagulation abnormalities, which require prompt and ongoing correction. Often, bowel which is borderline ischemic at the initial exploration will improve after physiologic stabilization. Various techniques of open abdomen have been described. The author's preferred mechanism is a simple plastic adhesive drape with underlying closed suction systems and moistened gauze over the bowel. Most often, reexploration can be accomplished within 48–72 h, and decisions regarding anastomosis, stoma, or additional resection can be made with plans for sequential abdominal closure [37].

26.4.4 Anticoagulation Therapy

Anticoagulation therapy is an important adjunct in patients with AMI. This decision must be balanced carefully with the

current hemodynamic state of the patient, degree of coagulopathy, and the needs for repeat exploration and/or bowel resection and anastomosis. In general, more aggressive anticoagulation is important in cases of mesenteric venous thrombosis as a primary treatment to the underlying hypercoagulable state. Administration of anticoagulants (heparin) to prevent further extension of thrombus in AMVT or post-revascularization in AMAI has been recommended [38] Heparin inhibits further thrombogenesis and prevents additional clot accumulation with caution to the possibility of gastrointestinal bleeding. Conversion to oral warfarin with dose adjustment is always indicated and should be continued for at least 6 months [39].

26.4.5 Endovascular Techniques

Several cases utilizing endovascular techniques in concert with thrombolytic therapy have recently been reported, although if there is any evidence clinically of bowel ischemia or infarction, thrombolytic therapy is contraindicated. Accordingly, these techniques appear indicated only in very early, almost incidental findings of AMI, and the role of such procedures still remains to be determined [40, 41]. Other contraindications to thrombolytic therapy include recent surgery, trauma, cerebrovascular or gastrointestinal bleeding, and uncontrolled hypertension.

The largest review of endovascular treatment of AMI involved 70 patients. It was considered to be successful in 87% of the patients, and in-hospital mortality was lower among those who underwent endovascular procedures than among those who underwent open surgery (36% vs. 50%) [42].

Revascularization techniques are presented in Table 26.1.

Table 26.1 Revascularisation techniques in acute arterial mesenteric ischemia

Open revascularization	Endovascular strategies
Embolectomy	Thrombectomy with
Arterial bypass	mechanical aspiration
Aorto-SMA	Angioplasty
Ileo-SMA	Stenting
Thromboembolectomy and retrograde stenting	Thrombolysis

SMA superior mesenteric artery

26.4.6 AMI Due to Embolus to the Superior Mesenteric Artery

Surgical embolectomy remains the mainstay of therapy, although there are numerous case reports of successful percutaneous treatment with comparable results to the open approach [43]. Due to technical challenges, however, even in the absence of bowel necrosis, open surgical procedures are frequently employed.

The procedure is usually performed via a midline incision approaching the SMA just below the pancreas at the mesenteric root [44]. A transverse arteriotomy is then made after proximal and distal clamping and embolectomy catheters are used to clear the artery proximally and distally. After completing the thrombectomy, the artery should be flushed gently with heparinized saline. The arteriotomy is then closed primarily or with use of a venous patch. Full anticoagulation is required with continuous heparin (PTT goal of 70–80 s) followed by LMWH 1 MG/kg twice a day corrected to renal function.

Endovascular embolectomy may be achieved by percutaneous mechanical aspiration [45] or thrombolysis [46, 47] and permits percutaneous transluminal angioplasty (PTA), if necessary, with or without stenting [48, 49] (Fig. 26.3). The applicability of

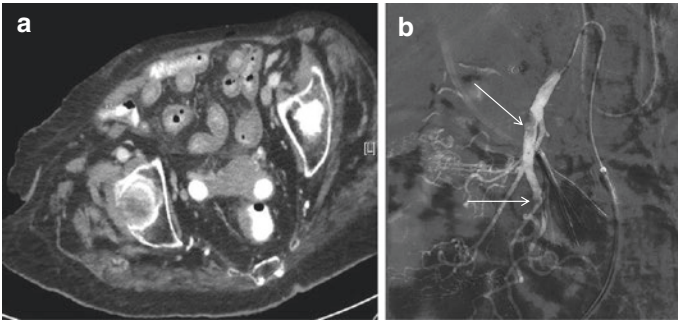


Fig. 26.3 Patient with recent history of hypercoagulable state and pulmonary emboli (after IVC filter insertion) presented with abrupt onset of abdominal pain and lactic acidosis. After initial CTA that showed superior mesenteric artery (SMA) occlusion and thickened small bowel loops (a), a selective angiography shows a nearly occluded SMA secondary to embolus in 2 levels (b, arrows). Thrombolysis was established and resulted in restoration of mesenteric blood flow

this approach is limited, since most patients present with symptoms that warrant an exploratory laparotomy for evaluation of intestinal viability.

26.4.7 SMA Thrombosis

Endovascular management is preferred for AMI thrombosis whenever possible and should be employed as expeditiously as possible in order to avoid and potentially reverse ongoing intestinal ischemia or necrosis [42, 50].

In the current era when early diagnosis of AMI may be accomplished, less invasive techniques of revascularization may be utilized, such as PTA and stenting. Other techniques described in the literature include percutaneous aspiration thrombectomy and local fibrinolysis. If surgery is required for resection of ischemic/necrotic intestine, or when percutaneous

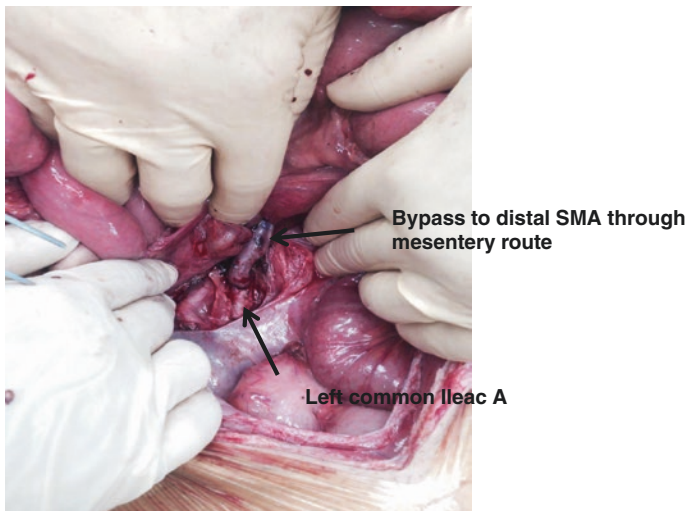


Fig. 26.4 Patient with acute thrombosis of SMA underwent left ileo-SMA bypass with common femoral vein graft

treatment has failed, conventional arterial bypass surgery remains an important option [51]. There are a variety of bypass procedures, providing either antegrade or retrograde flow, with vein (preferably) or synthetic grafts. An antegrade bypass from supraceliac aorta to superior mesenteric trunk is considered the most frequently employed open technique. However, the most practical option for proximal mesenteric atherosclerotic occlusive disease is a retrograde bypass from the common iliac artery with a vein or synthetic graft (Fig. 26.4).

26.4.8 NOMI

Management of NOMI is based on treatment of the underlying precipitating cause. Fluid resuscitation, optimization of cardiac

output, and elimination of vasopressors remain important primary measures that greatly impact outcome. Additional treatment may include systemic anticoagulation and the use of catheter-directed infusion of vasodilatory and antispasmodic agents, most commonly papaverine hydrochloride [52]. The decision to intervene surgically is based on the presence of peritonitis, perforation, or overall worsening of the patient's condition [53]. Generally, these patients are often in critical condition in the intensive care unit and mortality remains very high (50–85%) [54].

26.4.9 Venous Ischemia

Mesenteric venous thrombosis (MVT) has a distinctive clinical finding on CT scan, and when noted in a patient without findings of peritonitis, nonoperative management may be considered. . The first-line treatment for mesenteric venous thrombosis is anticoagulation. Systemic thrombolytic therapy is rarely indicated. When clinical signs demand operative intervention, one should resect only obvious necrotic bowel and employ damage control techniques liberally, since anticoagulation therapy may improve the clinical picture over the ensuing 24–48 h. Early use of heparin has been associated with improved survival [55].

26.5 Clinical Course and Outcomes in AMI

Perioperative complications occur frequently in AMI. A recent review suggested that up to 70% of patients may develop pneumonia, renal insufficiency, and sepsis to varying degrees.

A small proportion of patients survived massive bowel resection and develop short-gut syndrome, requiring long-term total parenteral alimentation or small-bowel transplantation.

Most large studies examining outcomes of patients with AMI report perioperative mortalities ranging from 32 to 69% [56–58]. Mortality rates for thrombotic occlusion exceed those for embolic occlusion and lowest (~20%) for MVT [54, 59]. Multiple organ failure remains the most frequent cause of death [60].

A recent retrospective study reviewed outcomes of 1,857 patients who underwent SMA PTA with or without stenting versus 3,380 patients who had open surgical exploration during a recent 16-year time frame. In-hospital mortality was significantly lower for patients treated with PTA (15.6%) versus surgical exploration (38.6%) [59].

But in another prospective review of 257 patients treated for AMI before and after the development of endovascular techniques, there were no differences in operative morbidity, mortality, or length of stay between patients treated with open repair and endovascular techniques, and at 5-year follow-up, there continued to be no differences between the groups for primary and secondary patency rates and recurrence-free survival [61].

26.6 Summary

AMI constitutes a varied and broad clinical spectrum. The underlying principles of management, regardless of the type of ischemic injury, require a high index of clinical suspicion based upon the individual patient, their clinical history, and exam findings, complemented by early imaging to establish a diagnosis and subsequent treatment plan. Ischemic injury requires a unique management plan. Since there are no current randomized trials to guide treatment, the approach should be based upon the individual experience of a particular institution. Improved imaging techniques and critical care suggest that for the first time, improvements in survival of this group are feasible. Such

improvements are feasible based upon the following management principles:

1. Patients with AMI must be identified early in the clinical course and treated aggressively.
2. All patients presenting with abdominal pain and a paucity of physical findings should undergo emergent imaging with three-dimensional reconstruction to identify vascular pathology and facilitate prompt treatment plans.
3. Damage control laparotomy will help to facilitate prompt ICU restoration of physiological derangements and allow for reassessment of bowel viability and the needs for further vascular intervention or bowel management.

Adaption of these principles will most certainly promote improved results to this constellation of disease processes.

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Chapter 27

Open Abdomen Strategies in Acute Care Surgery: When and How

Michael Sugrue

27.1 Acute Care Surgery

Acute care surgery delivers treatment to a broad range of surgical patients in a wide range of hospitals and health systems. Emergency surgery workloads vary from institution to institution, with emergency surgical admissions accounting for 25 % of admission in most hospitals. Letterkenny University Hospital like many regional or rural hospitals has an even higher emergency admission load accounting for 80 % of all the 3000 surgical admissions.

Increasingly acute care surgical units and systems are evolving, with improved process and delivery of care. Challenges in surgical training may make hands-on experience, especially for uncommon conditions such as the open abdomen, more difficult [5]. Surgical patients presenting with acute abdominal conditions

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requiring emergency surgery have a significant mortality; on average 13% die. A key to optimizing outcomes is the early identification of sepsis, of which intra-abdominal sepsis is one of the most important [36, 37]. In the critically ill septic surgical abdomen patient with progressive sepsis, there is an important link between sepsis and intra-abdominal hypertension [43]. Understanding the critical nature of intra-abdominal hypertension and its progression to the abdominal compartment syndrome (ACS) is essential in optimizing outcomes [11, 25, 41, 42]. One has however to have a balanced approach and avoid overuse of the open abdomen in acute care general surgery [21].

This chapter will explore when to consider leaving the abdomen open and how to close. Tips and traps during patient management will be highlighted.

27.1.1 Defining the Population and Problem

The use of the open abdomen (OA), either prophylactically or therapeutically, has been a well-recognized option in trauma patients for over 20 years [22]. The indications and frequency of use of the OA in non-trauma patients is less well understood. Between 10 and 25% of trauma patients undergoing abdominal surgery have had OA [30]. The reported incidence of OA in non-trauma general surgical patients is hard to quantify [2, 6, 24, 40]. In a recent review of the open abdomen in 338 primary laparotomies (excluding reexploration of initial laparotomy) that were performed in 1 year at Maryland, 96 patients (28%) were managed with an OA ([6]). This probably reflects the tertiary referral pattern to that hospital, and a more realistic figure would probably be 3–5% of non-trauma laparotomies that need an OA. The initial indications for surgery that lead to the OA from the Maryland series are shown in Table 27.1. The usual indications for open abdomen are shown in Table 27.2. The large number of the Maryland series having an OA would be

Table 27.1 Indications for laparotomy leading to open abdomen $n=96$

Perforated viscus/extraluminal gas on imaging	20
Mesenteric ischemia	17
Peritonitis/septic abdomen	16
GI hemorrhage	12
Intestinal obstruction	9
Incarcerated hernia	7
Abdominal compartment syndrome	6
Pancreatitis	5
Other	4
Elective operation	4

Source Bruns et al. [6]

Table 27.2 General indications for open abdomen in non-trauma patients

Damage control	40
Facilitate early second look	25
Multiple	20
Excessive contamination	10
Decompression to prevent-treat ACS	5

unusual, and an OA in a general surgery patient would be the exception, with rates of 2–5% of all laparotomies reflecting usual practice.

Harvin, in a recent study, has highlighted problems with overuse of damage control patients [21]. In their retrospective series, damage control laparotomy (DCL) was associated with an 18% increase in hospital mortality, a 13% increase in ileus, a 7% increase in enteric suture line failure, an 11% increase in fascial dehiscence, and a 19% increase in superficial surgical site infection [21]. Consequently a cautious approach to open abdomen is important. Laparostomy and the OA are a life-saving procedure in the right patient but also a potential life-altering event with potential source of morbidity and mortality in the wrong patient.

Therefore to optimize outcomes in open abdomen, it should be “decision before incision.” Choosing the right indication for OA is crucial. Atema, in a recent review of open abdomen in non-trauma patients, identified 74 studies describing 78 patient series, comprising 4,358 patients of which 3,461 (79%) had peritonitis. The mean age of the included patients ranged from 45 to 66 years, mean APACHE II scores ranged from 13 to 28, and the mean Mannheim Peritonitis Index ranged from 24 to 34 points. Two recent publications found overall mortality rate between 30 and 36% [2, 16]. Atema et al. found the lowest weighted mortality were techniques using dynamic retention sutures (11.1%, 95% CI 4.5–25.0%), while the highest mortality was reported after loose packing (40.0%, 95% CI 25.5–56.5%). This is not surprising as loose packing should only be used in resource challenged health care.

27.1.2 Who Needs an Open Abdomen

The decision to leave an abdomen open is generally decided at index emergency laparotomy. Occasionally patients with critical peritonitis can be identified prior to surgery, in the emergency department or ICU, signifying that a primary fascial closure would be unlikely. In general patients with Hinchev III or IV peritonitis do not need to have their abdomen left open [14].

Specific subgroups of patients pose a particular challenge. Patients with severe acute pancreatitis are heterogeneous, displaying different sequelae of the inflammatory process, some with severe intra-abdominal necrosis and others with acute lung injury and ARDS. Due to the variable amount of tissue edema, ascites, and resultant intra-abdominal hypertension, a tailored approach to decompression is required. In general patients with pancreatitis need decompression if IAP \geq 25 mmHg with associated progressive abdominal signs and worsening respiratory status. These constellations of symptoms have the hallmarks of acute compart-

ment syndrome (ACS). Failure to open (anterior abdominal compartment) may result in failure to prevent mesenteric ischemia or if ischemia is established delayed resection of bowel [38]

Traps

Elevated lactate occurs in less than 75 % of cases of mesenteric ischemia.

Tips

When you look at the patient's monitor, don't be fooled by a systolic BP reading >100 mmHg. Anesthetic advances have ensured that they can avoid hypotension as the patient may be on large doses of inotropes such as noradrenaline or adrenaline. Anesthetists in general will not burden you with this information while you are in the middle of surgery. Trending of physiological perfusion variables is helpful. The most easily assessed is base deficit trends (lactate also, if is available as a point of care test). Ideally surgery should be less than 90 min for an emergency laparotomy. Exceptions to this will include patients with BMI in excess of 40, extensive resections such as subtotal colectomy or re-operative surgery. A consultant surgeon should be present at an emergency laparotomy and a bed booked in a high dependency or ICU given the risk of mortality in excess of 10 %, rising to >30 % in an open abdomen.

The need to leave the abdomen open increases with massive resuscitation. We are familiar with this in trauma patients and those following emergency aortic surgery. Occasionally we have to surgically decompress patients with secondary abdominal compartment syndrome. Before considering decompression in secondary abdominal compartment syndrome, ensure that any ascites fluid is percutaneously drained and that there is no gastric distension amenable to NG decompression [12]. Striking a balance in fluid resuscitation has been very challenging, and Mason and

colleagues from Sunnybrook suggest that we may have gone too far and that restrictive resuscitation predicted by the Parkland formula increases acute kidney injury without increasing infectious complications [29]. About 20% of decompressed patients with an OA will develop tertiary ACS from either persistent bleeding, sepsis, or tissue edema. Kirkpatrick has recently coined the term quaternary compartment syndrome in patients following complex abdominal wall reconstruction.

Patients with tertiary ACS need reexploration and creation of a silo unless there is severe coagulopathy, which would need to be addressed first if possible. Negative-pressure therapy will reduce tertiary abdominal compartment by effective removal of excess fluid and toxic cytokines [26].

Tip

In patients with secondary or tertiary ACS, they are also more prone to polycompartment syndrome, involving the limbs (Fig. 27.1).



Fig. 27.1 Oedematous patient with Open abdomen prone to Limb Compartment syndrome

Trap

Never operate for an isolated elevated IAP reading as one must ensure that there is not a blocked urinary catheter or massive gastric distension. If you do perform a laparostomy, despite the expense of a NPWT dressing, use NPWT dressing from the initial laparotomy [19].

27.1.3 What Is the Best Technique for Laparostomy

“Hey diddle diddle straight down the middle”

27.1.4 A Longitudinal Midline Incision

Generally small laparostomies are rarely indicated, so the incision is long. The exception to this is small bowel ischemia, where ideally a preoperative diagnosis will allow a tailored incision; if a bowel resection/revascularization occurs, the open abdomen will facilitate a second look.

Having tailored the length of the incision to the underlying problem, one should remember additional procedures may confound laparostomy closure. These include ostomies, transperitoneal feeding jejunostomies, and placement of drains. If an anastomosis is performed in the presence of a lot of tissue edema, then a handsewn technique, rather than stapled, would be preferable. If a stapled technique is used, the stapler must be closed and locked in place for at least 20 s before firing to reduce tissue edema. Where an anastomosis has been performed, it should be kept as far away from the open abdomen as possible. Unfortunately in these cases, there is often no omentum for a variety of reasons.

A lateral stoma should be considered [1, 4] to avoid potential wound care issues, especially if negative-pressure wound therapy

(NPWT) is being used. Jejunostomy should be avoided if possible as they may leak due to the lack of adhesions from multiple repeat operations.

A transverse laparostomy, while possible, does not give the same access.

27.2 Maintaining Domain

Mesh-mediated traction system combined with negative pressure should be used from the first operation [32]. (Exception in mesenteric ischemia patients – they don't need the mesh as resection allows easier primary fascial closure.) Colonization of wounds is very common and increases the length of time the abdomen is left open. Particular attention to sterility is important when patients have an aortic graft or retroperitoneal necrosis in pancreatitis.

27.2.1 What Are the Potential Mistakes You Are Likely to Make?

We all make errors [13] and one rarely feels that you could not improve aspects of an individual's care when they have an open abdomen. Failing to have a clear plan of closure leads to ambiguity. The ICU and closure plan is essential and must be written in the index operation sheet. It provides a goal for the ICU and surgical team. This plan may need to be changed depending on sepsis profile and organ function. The common mistakes are shown in Table 27.3. The potential problems with the open abdomen patient are shown in Table 27.4.

Table 27.3 Mistakes one could make in the care of the open abdomen patient

Preoperative

Procrastination and delay

Refusing to think you need the CT now

Overcoming the demands of private practice

Coming in to see the patient

Recognizing that your patient has a problem

Failing to analyze trends in key labs such as C-reactive protein (CRP)

Intra-op

Incorrect incision

“Tram-lining” parallel incisions close to previous incisions

Not having an established open abdomen protocol

Failing to use a dynamic closure system with NPWT at the 1st laparostomy

Applying too much of the self-adhesive dressing on skin

Post-op

Not documenting a clear closure plan

Proactive recurrent sepsis identification

Not paying strict attention to fluid volume administration

Leaving the care to ICU team

27.2.2 When Do You Close? Close Early!

You close as soon as possible. At the index operation for the OA, the closure plan should in general be 24 h for post-mesenteric ischemia resection, 48 h post-trauma packing, and 7 days for pancreatitis or tertiary peritonitis patients. The abdomen needs to be washed out and sequential tightening of the mesh-mediated traction at 48–72 h. This involves a team approach which includes ICU, nursing, medical, and theater staff. Miller, in a large series, identified that if the abdomen was not closed within 7 days, the complication rate increased dramatically. In Atema’s review, when NPWT was used without fascial traction, a fistula

Table 27.4 Potential problems of the open abdomen

Loss of abdominal domain
Evisceration in ICU
Bleeding superficial/deep
Infection
Colonization (100 %)
Superficial Infection (10 %)
Fasciitis (2 %)
Deep space infection (varies on cause)
Graft infection (1 %)
Skin excoriation
Inability to close 20 %
Unplanned incisional hernia
Planned ventral hernia
Increased risk of anastomotic breakdown 5 %
Gastrointestinal fistula formation 5–10 %
Physiological/metabolic disturbance
Derangement of fluid and electrolyte balance
Hypoalbuminemia and persistent catabolic state
Psychological and family issues

rate of 14.6 % was seen. But when NPWT was combined with continuous suture or mesh-mediated fascial traction, the fistula risk dropped to 5.7 %. There will be a number of patients that you will not be able to close, and if you have not closed by day 10, you will need additional help to maintain abdominal domain [8–13, 16–18]. While NPWT can be used, coverage of the bowel is important that it is not left exposed to avoid fistulation. In this situation where there is a sizeable defect, a biological mesh is probably preferable. Other options include skin coverage, which generally will require a releasing incision or a skin graft. The advantage of grafting is that it will correct the catabolic effect of

the open abdomen quickly but will doom the patient to a delayed ventral hernia repair. A biological bridging mesh is probably most suitable. While Burlew reported up to a 100% fascial closure, a recent large series from Germany of 355 patients (even when sub-analyzed to the most recent patients examined [2011–2013]) reported a fascial closure rate of only 49% [7, 28]. If one closes too early, reexploration will be required. In a recent series, O'Meara found 14/37 patients undergoing primary fascial closure returned to the operating theater, 50% due to fascial dehiscence and the rest due to recurrent sepsis, bleeding, or abdominal compartment syndrome [31].

Tips

Aids to closure could include IV hypertonic saline, peritoneal resuscitation, and the potential to de-resuscitate with PEEP combined with albumin infusion and Lasix in the first 24 h prior to closure [20, 39].

27.2.3 *Technique in Closing*

Having made a decision to close tissue handling is crucial. The surgeon must love their tissues avoiding tension and traumatic tissue handling and reducing seroma formation. The options in closure are shown in Table 27.5. Approximately 15% of primary fascial closures will dehisce, so consideration of a supplemental prophylactic onlay mesh should be considered. Retention sutures could be used but generally result in nasty cross-hatching. The ABRA dynamic closure system is effective but may also macerate the skin.

Skin closure should be achieved unless there is significant contamination. While there will invariably be contamination, overt infection is rare, due mainly to effective NPWT, and this will allow a subcuticular stitch. The wound appearance and

Table 27.5 Technical options in abdominal closure

<i>Simple coverage</i>
Packing
Skin only
Towel clip closure
Mesh sheet
<i>Closed systems</i>
Suction drains
Negative-pressure therapy
<i>Dynamic-sequential closure</i>
ABRA
Whitman patch
Dynamic retention sutures
Mesh-mediated traction
<i>Combination therapy</i>
Dynamic and NPWT
<i>Tissue coverage</i>
Split-skin graft
Component separation
Free flaps

complication rate may be reduced by the application of a closed incision negative-pressure therapy system (Fig. 27.2).

A key to avoiding fistula formation is covering the bowel. Generally abdominal wall closure cannot occur if a fistula develops, and unless the fistula heals spontaneously, the patient will be doomed to an open abdomen for 6–9 months [44]. As Di Saverio states a fistula, it is a true surgical nightmare [15]

I would not recommend acute component separation as it may burn your bridges if you have to go back into the abdomen [23]. It cannot be performed twice and of course it requires extensive undermining of the subcutaneous tissue. Similarly a posterior component separation, as described by Petro, can be used but has similar limitations in the acute settings as anterior component separation. It does not however require extensive subcutaneous dissection [33]. The same would apply to the

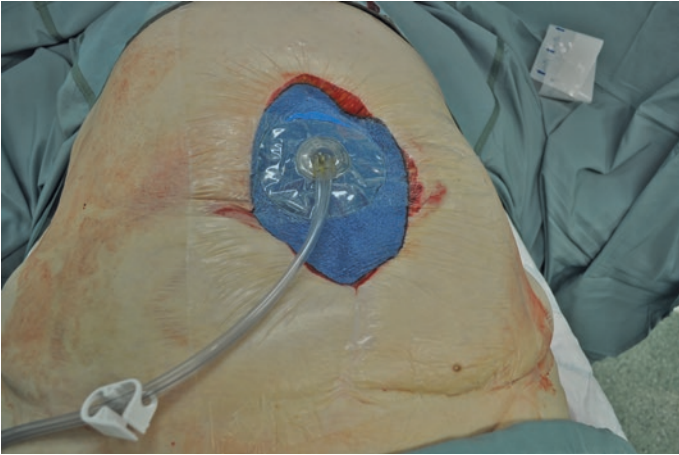


Fig. 27.2 Application of a NPWT system

transversus abdominis release. It is potentially possible to endoscopically perform aspects of the component separation; however, this is really more practical in the elective situation when undertaking large ventral hernia repair [27, 35].

Skin closure should be with subcuticular unless there is visible overt infection in the subcutaneous tissue [45]. If there is some purulent material in the wound, loose interrupted sutures can be used. If there is frank pus, a negative-pressure superficial wound system will facilitate management, and skin edges will heal by secondary intention. It is not justifiable however to keep the wound open to avoid the potential insurance company penalties for developing a surgical site infection [3]

Tips

Consider prophylactic technique to reduce risk of fascial dehiscence [34].

27.3 The Future

While outcomes in the critical surgical abdomen have improved, due in part to the Trojan research and educational activities of the World Society of the Abdominal Compartment Syndrome and the World Society of Emergency Surgery to name but a few, there remains a great opportunity to improve outcomes further. Variability in the delivery of acute surgical care needs to be addressed and the Donegal Summit on Performance and Outcome in 2016 is a step in the right direction to complement existing guidelines of many societies and introduce some measurable key performance indicators (www.wses.org.uk/congress).

Experimental work on sepsis source control combined with novel concepts such as peritoneal resuscitation is exciting. In the end, it is all about the patient and their family, and for that reason, patient-related outcomes should be expanded coupled with international registries of outcomes (www.wses.org.uk).

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Chapter 28

Entero-atmospheric Fistula: Tips and Tricks for the Management of a Surgical Nightmare

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28.1 Historic Background

The inception of open abdomen (OA) after an emergent laparotomy probably dates back in the first years of the twentieth century, when Pringle originally described the use of open abdomen in case of traumatic massive hepatic hemorrhage [1]; on the contrary, the first description of a temporary abdominal closure system for OA management is attributed to Ogilvie in 1940 [2]. These techniques remained unused for decades, because of their tremendous infective and hemorrhagic complications, and their popularity started growing in the 1990s when the widespread use of antibiotics, the progress of intensive care medicine, and the better comprehension of the physiopathology of trauma [3] and abdominal compartment syndrome (ACS) made it a cornerstone step of damage control surgery (DCS) [4, 5].

Given the indisputable advantages of OA, the exposure of the abdominal viscera to the outer environment brings itself an unavoidable load of morbidity, where the appearance of an entero-atmospheric fistula (EAF) is the most feared complication. In fact, the onset of an EAF is associated with significant morbidity and mortality, because of its extremely challenging critical care and nutritional management issues. EAF therefore requires a multidisciplinary management, and despite the continuous progresses in OA management, EAF's mortality can nowadays still be as high as up to 40% [6, 7].

Nowadays, the commonly accepted indications for OA [8, 9] are trauma, abdominal sepsis, severe acute pancreatitis, loss of abdominal wall (either traumatic or after necrotizing fasciitis), intra-abdominal hypertension or abdominal compartmental syndrome (ACS), and acute mesenteric ischemia.

28.2 EAF Definition

An entero-atmospheric fistula (EAF) is an opening in the GI wall occurring in the setting of an open abdomen, thus creating a communication between the GI lumen and the external atmosphere. This kind of fistula has specific features that make the spontaneous closure of the fistula almost impossible, such as the absence of a proper fistula tract and the lack of well-vascularized surrounding tissue. Furthermore, the location within an open abdomen results in spillage of enteric content directly into the open peritoneal cavity with all its detrimental effects.

28.3 Etiology, Incidence, and Risk Factors

EAF incidence rates are still largely unknown and extremely different in different case series; essentially, overall EAF is growing following the increased use of DCS and OA. In a recent meta-analysis, Atema et al. [10] reported rates of fistula ranging from 5.7 to 17.2% in non-trauma patients, while the incidence of EAF in a large series of OA for the management of abdominal sepsis was even higher, reaching 54.5% [11]. The pronounced variability in incidence rates could be due to the multiple possible etiologies of EAF and to the different indications for OA: noticeably, any trauma to the bowel occurring during the index operation or at the time of dressing changes can result in EAF formation, particularly when the bowel is desiccated and dehydrated due to the exposure to the atmosphere; furthermore, it became clear that the longer the abdomen is left open, the greater is the risk of EAF [12], and every attempt should be made to achieve definitive closure of the

open abdomen as soon as possible. The risk of formation of an EAF also differs between trauma patients and patients with peritonitis, with the infected abdomen more prone to fistula development [10]. Other conditions that could lead to fistula formation are anastomotic leakage, ongoing bowel ischemia, distal bowel obstruction, and adhesion of the bowel to the fascia.

Nevertheless, risk factors related to EAF development are still mainly undetermined. Recently, two interesting studies brought new light to the EAF formation process: a multicenter prospective observational study by Bradley et al. [13] demonstrated that large bowel resection, large volume resuscitation, and an increasing number of re-explorations were statistically significant predictors for the development of a fistula in trauma patients with OA; on the other hand, a retrospective dual-center analysis by Richter et al. [7] found that the only significant predisposing factor for fistula formation was the presence of diverticulitis, while bowel perforation, anastomotic leakage, and ACS showed a significant association with the occurrence of EA fistulas.

28.4 Classification

This is a fundamental step in EAF management planning, because every kind of EAF requires different approach. Traditionally, EAFs were classified only according to anatomical and clinical criteria, but in two recent works, Di Saverio et al. [14, 15] introduced a new and important criterion of classification: the number of fistula openings, dividing OA with a single fistula opening and OA with *multiple* fistula openings. This simple classification is fundamental, because the number of fistula openings and their distance from each other has substantial implications in the choice of the best management

technique for every single fistula, in order to achieve an effective control and diversion of its output.

Classical criteria are:

1. *Localization inside of the open abdomen: a superficial EAF* drains on top of a granulating abdominal wound and is easier to approach and manage, conveying mainly stoma/wound management issues. On the other hand, a *deep EAF* is a fistula arising deeply inside the OA and draining directly inside the peritoneal cavity causing an uninterrupted peritonitis; it is a surgical emergency due to the ongoing abdominal sepsis and requires immediate attention [16].
2. *Segment of GI tract involved: if the fistula originates from the stomach, duodenum, jejunum, or proximal ileus, it is classified as proximal; instead, a fistula arising from distal ileus or colon is classified as distal.*
3. *Fistula daily output: low output (<200 ml/24 h), moderate output (200–500 ml/24 h), and high output (>500 ml/24 h) [16–18].*

It is now easy to understand how distal and low output EAFs are more likely to close spontaneously, when compared to more proximal and high-output EAFs.

The arise of an EAF is also a landmark in Bjork classification of open abdomen [19], configuring the worst possible scenario for an open abdomen.

28.5 Prevention

Not surprisingly, the best way to deal with an EAF is to prevent it, and the knowledge of the causes and risk factors for fistula formation is essential for prevention. The first step is to be aware of the possibility of EAF formation, and a great effort is

required to the multidisciplinary team to prevent fistula formation. As it is for enterocutaneous fistulas (ECF), there are a bunch of conditions that are known to be involved in EAF development and maintenance and are traditionally listed in the mnemonic acronym *FRIENDS* (*F* foreign body, *R* radiation, *I* infection or inflammatory bowel disease, *E* epithelization of fistula tract, *N* neoplasm, *D* distal obstruction, *S* short tract <2 cm): these factors should be addressed promptly and prevented whenever possible.

As said above, the factors associated with the development of EAF are less clearly determined, and multiple causes can be recognized, such as anastomotic disruption, deserosalization, exposure of dehydrated and desiccated bowel to equipment and material used for temporary abdominal closure, adhesions, severe wound infections, severe trauma, bowel ischemia, visceral trauma during dressing changes, and NPWT; nevertheless, desiccation of bowel loops and unrecognized microtrauma occurring during dressing changes seems to be the most frequent trigger factor for fistula formation. Each one of these possible causes should be kept in mind and preventive measures must be put into effect:

1. Any rough and/or direct contact between the viscera and the devices used for temporary abdominal closure must be avoided or minimized, creating a sort of shield over the exposed abdominal viscera by placing the greater omentum to cover the bowel and protecting the viscera with a fenestrated plastic sheet extended from one paracolic gutter to the other.
2. Nonabsorbable prosthetic meshes are not recommended, as they can erode the abdominal viscera leading to fistula formation.
3. Whatever dressing is chosen to apply, make sure that the abdominal cavity is completely sealed and isolated from outside environment, thus preventing the exposure of bowel loops to the air and their desiccation.

4. Aggressive tissue preparation and extensive debridement should be performed only when absolutely necessary for interrupting or controlling the ongoing sepsis process.
5. Early split-thickness skin graft or cadaveric skin graft over the granulating viscera surrounding the fistula can help to protect the viscera and ease the EAF management.
6. Carefully plan dressing changes.
7. To reduce the risk of accidental bowel injury, dressing changes should be performed only or at least supervised by senior attending surgeons with specific expertise in OA management.
8. Close the abdomen as soon as possible, because the longer the abdomen open, the higher the risk of bowel exposure and trauma, invariably resulting in an increased rate of complications and EAF formation [12].
9. It has been hypothesized that negative-pressure wound therapy (NPWT) [20] may be associated to and facilitate the development of EAF, and in some animal models, performed by Lindstedt et al., higher levels of negative-pressure suction resulted in a measurable reduction in bowel blood flow that may induce ischemia and secondary necrosis in the intestinal wall, eventually leading to EAF formation [21, 22]. Even if this controversy has now largely been contradicted [23, 24] and the clinical impact of this observations is uncertain, a tendency toward caution and toward the use of lower pressures in suction [25] is advisable.

28.6 Nutritional Issues

Patients with EAF are constantly in a hypercatabolic state, due to the high amount of fluid and nutrients losses from fistula output, that almost invariably lead to fluid depletion, electrolyte alterations, and acid/base status disorders. Furthermore, the

open abdominal wound and the ongoing peritonitis constantly activate the systemic stress response. The combination of all these factors set the base for the constant malnourishment status [18, 26, 27] typical of this population of patients.

For this reason, the multidisciplinary team that is in charge for this particular population of patients must pay great attention to their nutritional status to prevent malnutrition and starvation.

The importance of a correct nutrition in critical patients was stressed for the first time in 1964, when Chapman et al. [28] noted that malnutrition was the leading cause of death in patients with ECF and mortality was considerably reduced with a daily intake of at least 1500 Kcal; since then, total parenteral nutrition (TPN) and nil per os has been the standard of care for severely injured patients.

Nowadays enteral nutrition (EN) is recognized as the best way for prolonged feeding, and the benefits of early EN compared to TPN are well known and supported by a great body of literature [29–39] and include preservation of the intestinal mucosal barrier and its immunological function [32] and a reduced rate of infectious complications; indeed, numerous studies have demonstrated the beneficial effect of EN and its feasibility even in patients with OA and EAF and showed a protective effect of EN from infectious complications without affecting fascial closure rates [31, 33]. For these reasons, EN should be started as soon as possible, and the goal of feeding EAF patients is not simply to prevent or treat malnutrition, but to manipulate the stress response to injury and infection and to improve the patients outcome [40].

Despite of its great results, the establishment and the management of EN in patients with and EAF are really challenging tasks. First of all, a precise definition of the anatomy of EAF is mandatory: the critical goal of this phase is to determine the total length of the remaining bowel and how much continuous surface or absorbing bowel is available for EN; this task can be really hard to achieve in case of multiple EAFs, and a bunch of

different radiological techniques can be combined for this purpose [37]. A correct anatomical definition is also the guide for the establishment of a correct EN access, which should be safe and easy to establish and maintain and should use as many small bowel segments for absorption as possible [34]. The establishment of a feeding access must be included into the therapeutic plan of every single patient and should be tailored upon specific patient characteristic [36, 41].

Another crucial issue is hydration and volume status monitoring that must be meticulous throughout the entire length of hospital stay; in this scenario, a correct VAC dressing could allow the precise quantification of fluid losses and may significantly reduce the evaporation across the open wound [39].

Nutritional assessment and monitoring and the creation of a nutritional plan are of pivotal importance. Different empiric nutritional estimators are currently available and can be used for a preliminary evaluation to the basal energy expenditure, and then corrections should be made in accordance to the specific characteristics of the single patient: high-output fistula will usually require 1.5–2 times the usual calories; supplementation of vitamins and trace elements is crucial, and a correction of 2 g of nitrogen per liter of abdominal fluid lost from the OA is necessary to maintain a positive nitrogen balance [38]. Furthermore, the knowledge of the electrolytes composition of the GI fluids is necessary [35].

It is necessary to highlight how EN can increase EAF output; for this reason, obtaining the complete and effective diversion of fistula effluent is mandatory. In this setting, a reduction in fistula output could be extremely helpful for a correct effluent control: H₂ receptor antagonists or proton pump inhibitors [42] can be used to reduce gastric secretion, while somatostatin and its analogues [32, 42–44] are used to reduce pancreatic and gastric secretion and gastric and gallbladder emptying. Despite their usefulness, caution must be used while employing these drugs, because they can also inhibit the secretion of insulin and glucagon and reduces splanchnic blood flow.

28.7 Principles of Management

In the worst-case scenario, all the above-cited precautions fail and a fistula arises inside of the open abdomen, configuring a real surgical nightmare. The obvious best therapeutic option, i.e., a proximal bowel diversion, is almost always impossible for several reasons, such as mesentery retraction and edema, “frozen abdomen,” and abdominal wall tissue loss or retraction (Fig. 28.1). EAF has unique features making its spontaneous closure almost impossible to achieve; for this reason, the main goal is promoting the EAF to become a chronic but well-controlled fistula. This result is really hard to achieve, because

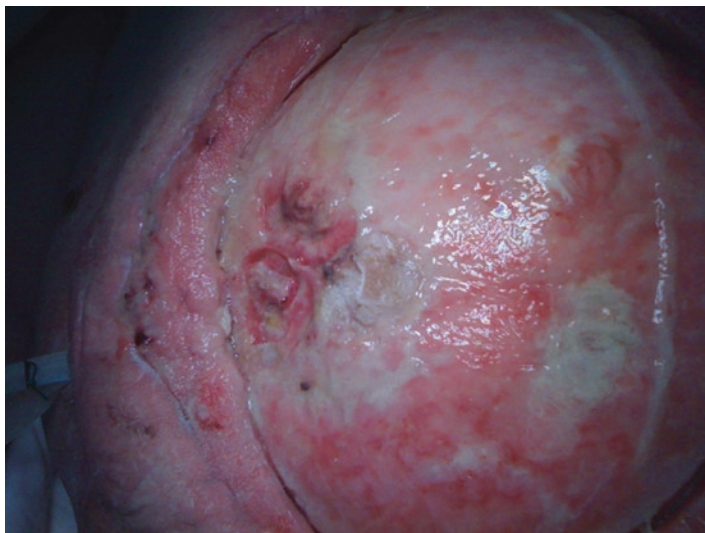


Fig. 28.1 Example of frozen abdomen with two adjacent fistula openings; in this case, it is absolutely impossible to perform any surgical maneuver, and the only option available is to promote fistula chronicization

of the extreme frailty of the tissues surrounding the fistula, the presence of a “frozen abdomen” that prohibits any surgical maneuver, and the relevant systemic derangements of the patient, driven by severe dehydration, hypercatabolic status, and ongoing sepsis caused by the spillage of enteric content directly into the peritoneal cavity.

At this point, the principles of management are similar to that guiding enterocutaneous fistula (ECF) management and are:

- Control of fistula effluent; a complete isolation of the fistula from the open abdominal cavity is mandatory and requires, especially in case of deep fistulas, the exposure and exteriorization of the fistula.
- Stop the ongoing sepsis and correct every physiological derangement.
- Protect the surrounding viscera and allow the clean granulation of the exposed bowel; obviously, this is strictly related to a correct fistula output diversion and the application of biological dressing (i.e., human acellular dermal matrix, cadaveric split-thickness skin graft, or autologous skin graft) [45–47] can create a natural protection over the exposed bowel and can avoid the creation of additional holes.

When an EAF is discovered, a careful and complete exploration of the peritoneal cavity must be performed (Fig. 28.2): it is of vital importance to exclude the presence of other hidden undiagnosed fistulas that can maintain the sepsis and to rule out any condition that could preclude the closure of the fistulas (i.e., F.R.I.E.N.D.S.); these underlying conditions must be treated promptly; otherwise, every attempt to promote fistula closure would be vain and fistula effluent control could be really challenging. Subsequently, prior to placing any dressing over the open abdomen, a meticulous irrigation of the abdominal cavity with saline solution should be performed, to dilute peritoneal contamination and limit the ongoing sepsis.

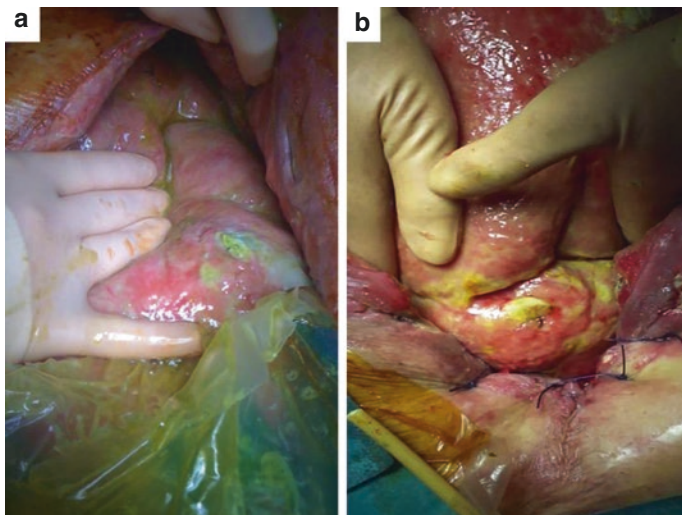


Fig. 28.2 (a) Arise of an entero-atmospheric fistula in the midst of an open abdomen. (b) The first fundamental surgical maneuver is a meticulous and delicate exploration of the abdominal cavity, followed by an accurate cleansing of the abdominal cavity and exteriorization of the fistula

A myriad of different surgical techniques is described so far in literature [24, 45–71], and none of them would prove to be ideal in every circumstance. Theoretically, the best dressing for EAF management is the one that:

- Completely isolates fistula from the remaining open wound
- Is completely atraumatic on the fistula itself and on the frail exposed viscera
- Allows fistula output collection and quantification
- Is quick and easy to apply
- Easily allows nursing maneuvers
- Prevents fascial retraction and protects the fascia for an eventual primary closure
- Possibly, is cheap and made of easy-to-find materials

Every surgeon usually develops his own technique that fits as much of these criteria as possible (Figs. 28.3 and 28.4). In an attempt to rationalize the approach to EAF, Di Saverio et al. recently proposed a clinical algorithm based on an extensive review of the literature [14, 15]. This algorithm could be extremely helpful since it reassumes the most commonly used techniques into an easy-to-read diagram, thus facilitating the choice of the most appropriate dressing in every single case. We refer to these papers for a complete description of all the available surgical options for fistula diversion.

Clinical evolution of EAF depends directly on the fistula specific characteristics: usually small, distal, superficial, and

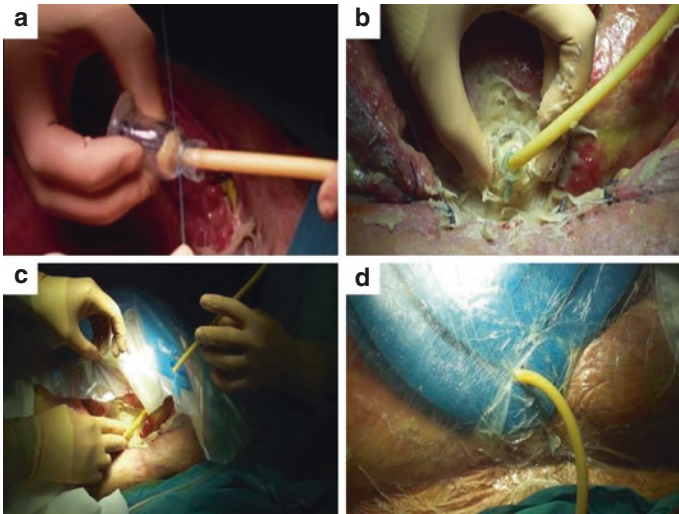


Fig. 28.3 Application of the baby bottle nipple diversion technique. (a) Preparation of the baby bottle nipple with the Petzer tube. (b) Application of the baby bottle nipple over the fistula, utilizing colostomy paste for a better sealing. (c) Passing the Petzer tube through the VAC dressing. (d) Complete dressing

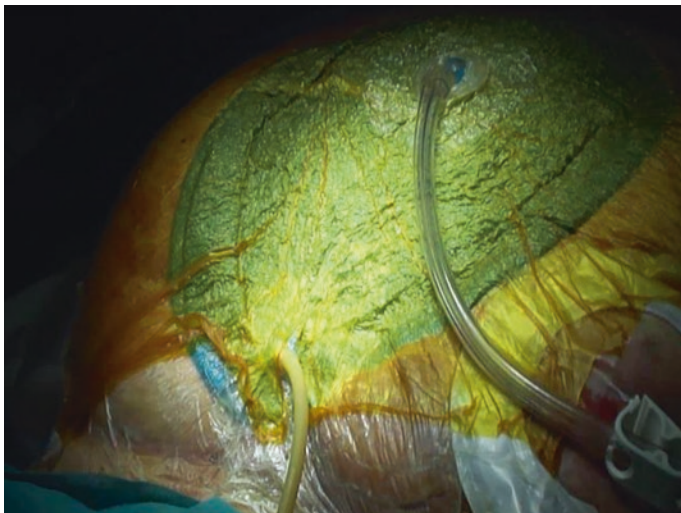


Fig. 28.4 Appearance of the baby bottle nipple diversion technique after the VAC dressing placement and the activation of negative pressure

low-output, single EAFs are more likely to close spontaneously, while large, deep, proximal, and high-output fistulas, or multiple openings, are extremely unlikely to heal spontaneously. In the first group of fistulas, it may be worth a try of primary closure with sutures and different kinds of sealants (fibrin glue, cyanoacrylates) [45, 47]. In contrast, in the latter group, the primary goal is to promote fistula control and chronicization: thus, the first step is the exteriorization of the fistula that creates a plane surface where a diversion device can be applied more easily and stops the spillage of enteric fluids deep inside of the open abdomen. Additionally, the potential presence of multiple fistulas makes the situation even more challenging to manage and requires a technique that should be easy and quick to apply and occupies only a little space in the OA, like the *nipple VAC* or the *baby bottle nipple diversion* technique.

Except for very favorable situations, where a spontaneous closure is likely to happen, every management technique is intended to be a bridge from the acute phase to a chronic phase; when sepsis and peritonitis are resolved, the surrounding bowel has granulated enough to allow a skin grafting and the patient has recovered from the insult and is well nourished and in a physiological balance. This situation usually requires months to completely realize. For this reason, most of the authors suggest to delay definitive surgery for fistula takedown and abdominal wall reconstruction for at least 8–12 months; in this lapse, visceral adhesions are supposed to get loose enough to allow a correct and safe surgery. Multiple surgical approaches for definitive fistula takedown and abdominal bowel reconstruction may be required in a step-by-step fashion, and several are described in literature [16, 66, 72–75].

Tips and Tricks

A useful acronym to be kept in mind when dealing with EAF is *SNAP* (*S sepsis and skin care, N nutrition, A anatomy, P plan*) [76]. This acronym effectively summarize all the management issues that come together with the arise of an EAF: first of all, the goal in the acute phase is sepsis control, while skin care (and wound care) is mandatory to allow a good granulation of the exposed viscera and to create the optimal conditions for skin grafting. The knowledge of the exact *anatomy* of the EAF is essential for a correct patient management: the critical goal is either to determine the total length of the remaining bowel and how much continuous surface of bowel is available for absorption and the exact location of the fistula to allow a correct quantification of fluid and electrolyte losses (knowledge of the electrolyte composition of the GI fluids is necessary [35]). The anatomical studying can be extremely challenging in case of multiple fistulas and can be achieved with a combination of computed tomography, magnetic resonance enterography, fistu-

lography, ingestion of dye or charcoal, and passage of enteric tubes used as landmarks during subsequent reimaging [37].

Dealing with an EAF is like looking into a kaleidoscope: a myriad of problems flock together, and the only way not to get lost is to prepare a precise plan with a multidisciplinary team. Surgeons, anesthetists, nutritionists, nurses, wound experts, and stoma experts should gather and formulate a plan that fits the requirements of every single patient. A well-conceived plan directly originates from a precise knowledge of the fistula anatomy and of the patient specific clinical, nutritional, and physiological condition.

28.8 Conclusion

The appearance of an entero-atmospheric fistula is an authentic surgical nightmare that offers an awful critical care problem set that includes surgical, metabolic, nutritional, and nursing issues. It is easy to understand how important prevention is: it is mandatory to be aware of the possibility of EAF formation. A multidisciplinary team formed by surgeons, anesthetists and ICU attending, nutritionists, psychologists, and specialized nurses should be in charge of the patient, and a great degree of expertise is required to correctly manage these patients. EAF management is complex and demanding and should be tailored upon the specific characteristic of every single case; from the surgeon point of view, the cornerstone of the treatment is the complete diversion of fistula effluent, thus blocking the contamination of the peritoneal cavity and the associated sepsis. Prevention of malnutrition, dehydration, and electrolyte imbalance with a proper parenteral/enteral nutrition is of vital importance.

Despite of the booming of papers about EAF management [16, 77] and the myriad of techniques proposed in literature, a great degree of confusion is still present and an optimal strategy

that could properly fit every single patient has not been found yet. The choice of the proper technique for every patient must be guided by a meticulous determination of fistula anatomy and by a careful assessment of patient's clinical and nutritional conditions. A well-designed plan is essential to integrate all the different and complex management issues.

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