Acute Care General Surgery

Workup and Management

Dale A. Dangleben
Firas G. Madbak
Editors

Springer

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To those who have taken the time to teach me along this journey, not just surgery but about life as a whole

—Dale A. Dangleben

To Tuhama and Layla for their unconditional love and sacrifice

—Firas G. Madbak

Preface

Patients who present with emergency general surgical problems often have profound physiologic derangements that require immediate correction and stabilization prior to planning a diagnostic approach. This varies from the classic approach we were taught that culminates in history taking, formulating a differential diagnosis, obtaining laboratory and imaging studies, and initiating treatment. The acute care surgeon should be aggressive, take ownership, and promptly begin treatment often without having all the information in these situations. The goal is to correct life-threatening physiology either concurrently or prior to definitive anatomic correction which can be delayed.

Despite the advent of modern imaging and state-of-the-art technology, we feel that little has changed in the approach to the emergency general surgical patient. The surgeon should always strive to learn "when to operate, how to operate, and when not to operate." From your surgical training, you already recognize that there are different ways to skin a cat. Our intention is not to reinvent the wheel but rather to provide easily digestible, quick overviews that cover essential points with the various acceptable treatment modalities. Special emphasis is placed on keeping therapeutic approaches as straightforward and simple as possible to achieve the best outcomes. We feel that what sets this book apart are the detailed photographs from our real patients' cases that complement the text. In addition, every chapter has a useful clinical pearl to serve as a catchy memory aid when dealing with difficult acute general surgical emergencies.

We are interested in what our readers think and will gladly respond to any suggestions or questions (dr.madbak@gmail.com).

We are especially indebted to our developmental editor, Connie Walsh, and our friend and colleague, Dr. Rakkiat Prasongdee, for providing some of the photographs.

Camp Hill, USA — Dale A. Dangleben
Jacksonville, USA — Firas G. Madbak
November 2016

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Appendiceal Mucocele

1

Bogdan Ionescu and Christie Hirsch-Reilly

Never forget to check the pathology report after a 'routine' appendectomy

Learning Objectives

1. The learner should appreciate the importance of preoperative identification of appendiceal mucoceles for appropriate operative planning.
2. The learner should know the treatment subtypes of mucocele and the treatments for each.
3. The learner should identify complications associated with rupture of an appendiceal mucocele.

Case Scenario

A 70-year-old woman presents to the emergency department complaining of right lower quadrant pain for 4 days. Her examination shows a palpable mass in the right lower quadrant. A CT scan shows a large distended appendix measuring 4 cm in diameter, without periappendiceal fat stranding (Fig. 1.1). The diagnosis of appendiceal mucocele is made. An open appendectomy is performed without spillage of any contents (Fig. 1.2).

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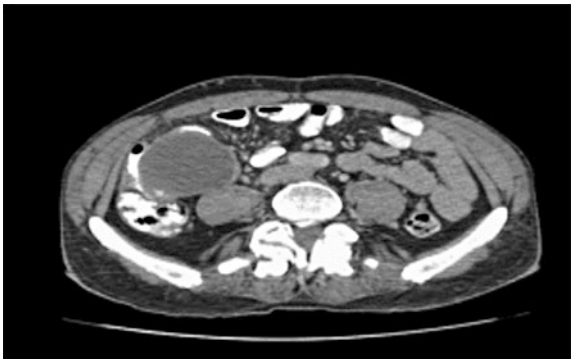

Fig. 1.1 CT scan showing very large mucocele of the appendix. © Dale Dangleben, MD

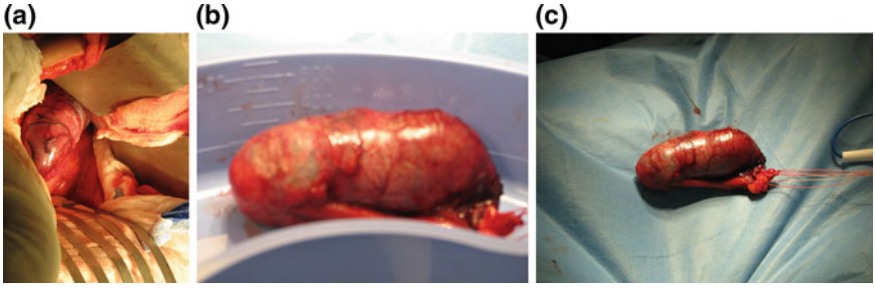

Fig. 1.2 **a–c** Open appendectomy for large appendiceal mucocele. All surgical precautions have been taken to prevent spillage. © Dale Dangleben, MD

Pathology revealed mucosal hyperplasia without signs of atypia. The patient was scheduled for routine post-appendectomy follow up.

Epidemiology/Etiology/Pathophysiology

Appendiceal mucocele is a distension of the appendix resulting from buildup of mucus secondary to obstruction of the appendiceal orifice by an appendicolith, inflammation, or mucous overproduction. It is a rare (0.2–0.7%) presentation of appendiceal pathology, more commonly found in women (4:1 ratio), after the age of 50. There are four distinct subgroups which include retention cysts that are rarely larger than 2 cm, mucosal hyperplasia which is an overgrowth of epithelial cells, mucinous cystadenoma which usually has a luminal diameter >6 cm with low-level dysplasia, mucinous adenocarcinoma where there is stromal invasion and possible intraperitoneal metastasis. Mucoceles less than 2 cm have low malignant potential. The larger the mucocele, the more likely the possibility of neoplasm [1].

Differential Diagnosis

Differential diagnosis for right lower quadrant pain includes acute appendicitis, diverticular disease, kidney stones, bladder distension, pelvic inflammatory disease, adnexal cysts, endometriosis, ectopic pregnancy, endometritis, and leiomyomas.

Diagnosis

Patients will most commonly present with right lower quadrant pain, abdominal mass, weight loss, or signs of bowel obstruction. Diagnosis is usually based on imaging findings. A distended appendix especially larger than 15 mm has a sensitivity of 83% and a specificity of 92% for appendiceal mucocele when there are no signs of acute appendicitis. The exact etiology can be determined by pathologic examination. In cases of appendiceal rupture, imaging serves a role for identifying signs of pseudomyxoma peritonei such as non-mobile ascites, multiple semi solid masses, and scalloping of the edges of the liver or spleen [2].

Complications

Large appendiceal mucoceles can result in abdominal pain and obstructive symptoms. Rupture of mucoceles can lead to spread of epithelial cells and mucoid fluid leading to pseudomyxoma peritonei (Fig. 1.3). This decreases 5-year survival rate from 91% to approximately 50% [3].

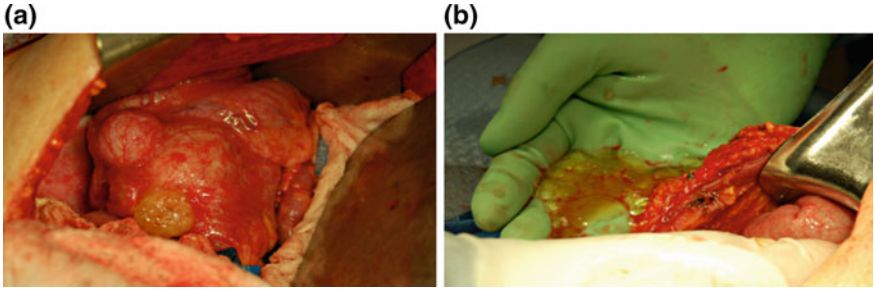

Fig. 1.3 **a**, **b** Mucinous material exuding from a perforated appendiceal mucocele. © Dale Dangleben, MD

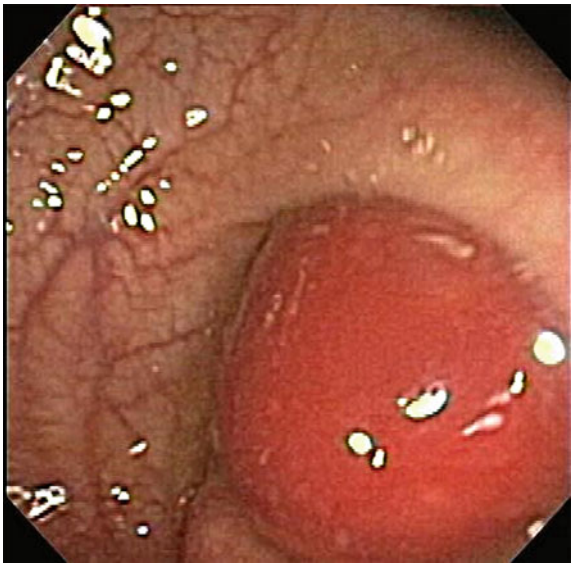

Fig. 1.4 Endoscopic view of mucocele bulging from the appendiceal orifice. © Dale Dangleben, MD

Management

The treatment for appendiceal mucocele is surgical excision. Laparotomy is preferred in most cases over laparoscopy due to the higher risk of rupture and possibility for developing pseudomyxoma peritonei which is a mucinous carcinomatosis of appendiceal origin (Fig. 1.4). The mesoappendix should be excised at the time of initial surgical management to determine lymph node involvement. In the case of tumor involvement of the base of the appendix, or tumors larger than 1 cm, a right hemicolectomy is indicated. If the patient develops pseudomyxoma peritonei treatment then involves early aggressive surgical debulking and hyperthermic intraperitoneal chemotherapy (HIPEC).

References

1. Asenov Y, Korukov B, Penkov N, Sedloev Th, Tihtchev V, Hadzhiysca V, Damianov D. Appendiceal mucocele—case report and review of the literature. Chirurgia. 2015;110(6):565–9.
2. Fishman MB, Aronson MD. Differential diagnosis of abdominal pain in adults. In: Auerbach AD, editor. Waltham, MA. Accessed on 17 Jan 2016.
3. Townsend CM, Beauchamp RD, Evers BM, Mattox KL, editors. Sabiston textbook of surgery: the biological basis of modern surgical practice. 18th ed. Philadelphia: Saunders; 2008.

Acute Appendicitis

2

Lissa C. Sakata and Lindsey Perea

Always keep it in the differential

Learning Objectives

1. The learner should be able to describe the etiology of acute appendicitis.
2. The learner should be able to formulate a differential diagnosis for right lower quadrant abdominal pain.
3. The learner should be familiar with physical "signs" specific for acute appendicitis.
4. The learner should know how to manage acute appendicitis.
5. The learner should know potential complications of acute appendicitis.

Case Scenario

A 20-year-old male with no past medical history presents to the emergency department with complaints of abdominal pain that began suddenly the night before. Initially, his pain was dull and around his umbilicus. The morning of presentation, it became sharp and located in the right lower quadrant. He complains

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of nausea and vomiting. On examination, he is afebrile with normal vital signs. He has focal tenderness to palpation in the right lower quadrant and voluntary guarding.

Epidemiology/Etiology/Pathophysiology

Acute appendicitis is the most common surgical emergency, accounting for approximately 300,000 operations annually in the USA. Appendicitis most frequently occurs during the second and third decades of life, but can occur at any age. There is a slight male predominance (1.3:1). The lifetime incidence in the USA is 8.6% for males and 6.7% for females.

Acute appendicitis occurs when there is obstruction of the appendiceal lumen. This may be secondary to a fecalith, lymphoid hyperplasia, malignancy, parasite, foreign body, or idiopathic causes. Once occluded, the appendix distends secondary to mucosal secretion which in turn limits venous return from the appendix. This leads to stasis and bacterial overgrowth in the appendiceal wall. Progressive ischemia may occur and lead to perforation of the appendiceal wall.

Differential Diagnosis

Differential diagnoses include inflammatory, gynecological, and mechanical etiologies. Inflammatory conditions include acute mesenteric adenitis, gastroenteritis, epididymitis, Meckel's diverticulitis, Crohn's disease, peptic ulcer disease, urinary tract infection, and Yersinia infection. Gynecological conditions include pelvic inflammatory disease, ruptured ovarian follicle, or ruptured ectopic pregnancy. Mechanical problems which may mimic appendicitis include testicular or ovarian torsion and intussusception.

Diagnosis

The classic presentation of acute appendicitis is dull, crampy, intermittent abdominal pain that starts in the periumbilical region. The dull intermittent pain is due to stimulation of visceral afferent nerve fibers from T8 to T10, as the appendiceal intraluminal pressure increases. Nausea, vomiting, and anorexia often accompany the abdominal pain. The pain then migrates to the right lower quadrant and becomes sharp and constant. Movement worsens the pain. The sharp, localized right lower quadrant pain is somatic and due to the inflammation of the appendiceal wall extending to the serosa and irritating the peritoneum. The classic presentation is not always present.

Physical examination is critical to the diagnosis and is used in conjunction with history and imaging. Abdominal tenderness will be at the anatomical location of the inflamed appendix.

Classically, maximal tenderness is at McBurney's point, located one-third the distance from the right anterior superior iliac spine to the umbilicus. Guarding or muscular resistance with palpation, as well as rebound tenderness when the hand is quickly removed, may be present.

Several signs exist that may help localize the location of the appendix based on examination. Rovsing's sign occurs when pain in the right lower quadrant is caused by left lower quadrant palpation. It suggests a localized peritoneal process in the right lower quadrant. The obturator sign is performed with the patient lying supine and pain is elicited with internal rotation of the flexed right hip. This suggests irritation of the obturator muscle by a low lying pelvic appendix. The psoas sign is performed with the patient lying on their left side. Pain with extension of the right thigh suggests a retrocecal appendix that is lying in contact with the iliopsoas muscle [1].

Due to differences in the location of the inflamed appendix, the clinical presentation is variable. If the appendix is retrocecal, the patient may have minimal anterior abdominal tenderness. In pregnancy, the appendix may be shifted upward, and pain elicited in the right upper quadrant. Once the appendix is ruptured, abdominal findings are more diffuse and less localized. A rectal examination should be performed, and a pelvic examination in females to rule out pelvic inflammatory disease or a pelvic mass.

Appendicitis is often associated with a low-grade fever, where a high temperature suggests a ruptured appendix or an alternative diagnosis. A mild leukocytosis may be present and associated with a left shift. A leukocytosis greater than 18,000 cells/mm^3 is suggestive of a perforated appendix. A normal white blood cell count does not rule out appendicitis. Urinalysis is performed to rule out urinary tract infection as a cause of symptoms. Pyuria and microscopic hematuria is common with acute appendicitis because of ureter or bladder irritation by the inflamed appendix. Bacteriuria is usually not present.

Imaging is generally unnecessary in young males with signs and symptoms consistent with appendicitis and a high clinical suspicion. The most common imaging modalities used are ultrasound and computed tomography (CT). Ultrasound is inexpensive, does not have ionizing radiation, and is readily available. Due to the operator-dependent nature of ultrasound, there is a variable sensitivity of 55–96% and specificity of 85–98%. Ultrasound findings may include a noncompressible appendix, diameter greater than 5 mm, wall thickening >2 mm, hyperemic wall by Doppler, presence of a fecalith, and periappendiceal fluid. Transvaginal ultrasound may help rule out gynecological disease in female patients. CT of the abdomen and pelvis with IV and oral contrast is 92–97% sensitive and 85–94% specific. It has the advantage of improved accuracy and can identify other pathology. Oral contrast may not be tolerated in patients who are vomiting and can

be performed without. Findings may include a dilated appendix >5 mm, wall thickness $\geq$2 mm often with concentric thickening of the wall, periappendiceal fat stranding, presence of a fecalith, and periappendiceal fluid. Free air or fluid, abscess or phlegmon suggests perforation. An abscess on CT is recognized as a rim-enhancing fluid collection; this is typically accompanied by a patient history of symptoms more than 48 h. Disadvantages of CT scanning are that it is expensive, exposes the patient to radiation, and is more time-consuming than ultrasound [2].

Diagnostic laparoscopy should be performed when the diagnosis of appendicitis remains unclear. This is especially useful in women when gynecological etiologies have not been ruled out. Intraoperatively, even if a normal appendix is encountered, it is usually removed, and in the future, a diagnosis of appendicitis can be ruled out. When appendicitis is not found, it is prudent to search for other etiologies of the patient's symptoms.

Complications

Abscess may occur after appendectomy, more commonly after perforation. Patients may be ill appearing and significantly tender. A fever, leukocytosis, and abdominal pain are common findings in abscesses, but one must also consider an abscess in the differential postoperatively in patients with ileus, diarrhea, or occasionally bowel obstruction.

Management

Intravenous antibiotics covering gram-negative and anaerobic flora should be initiated upon suspicion of acute appendicitis. Laparoscopic or open appendectomy is the treatment of choice for appendicitis without abscess (Figs. 2.1, 2.2 and 2.3). Periappendiceal abscesses should be treated with ultrasound- or CT-guided percutaneous drainage and antibiotics. Smaller abscesses (<3 cm) may be treated with antibiotics alone. An interval appendectomy may be planned at a later date (8 weeks); however, recurrence rates of 4% in 5 years in larger series suggest expectant management is also acceptable.

In the patient with contained perforation particularly with cecal involvement and who does not exhibit signs of peritonitis, initial nonoperative management may be advisable until inflammation subsides as urgent surgery may lead to more extensive surgery requiring colectomy [3].

Another less common scenario involving incidental intraoperative diagnosis of Crohn's disease is worth mentioning. If gross findings of Crohn's are found at surgery intended for appendectomy (mesenteric thickening, creeping fat), then

appendectomy is necessary to avoid diagnostic confusion in the future. If the appendiceal base or cecum is involved, the appendix should be left alone as the risk of fistulization is high.

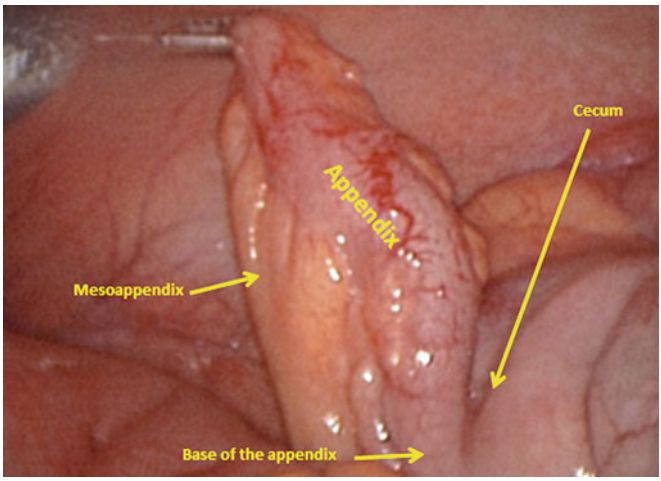

Fig. 2.1 Anatomy of the appendix. Image shows mild inflammation and congestion of the appendix. © Dale Dangleben, MD

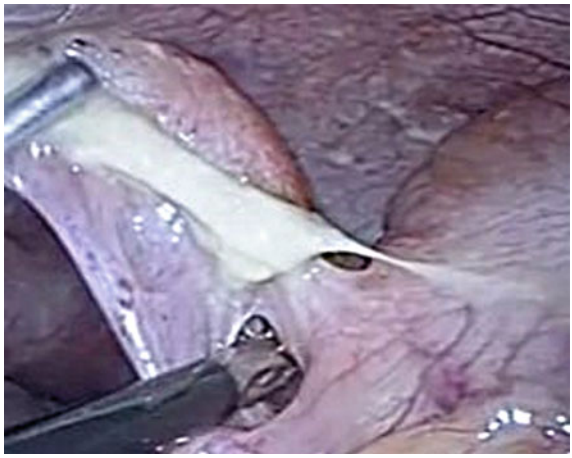

Fig. 2.2 Laparoscopic appendectomy with fibrinous exudate. AQ window is being created through the mesoappendix at the base. © Dale Dangleben, MD

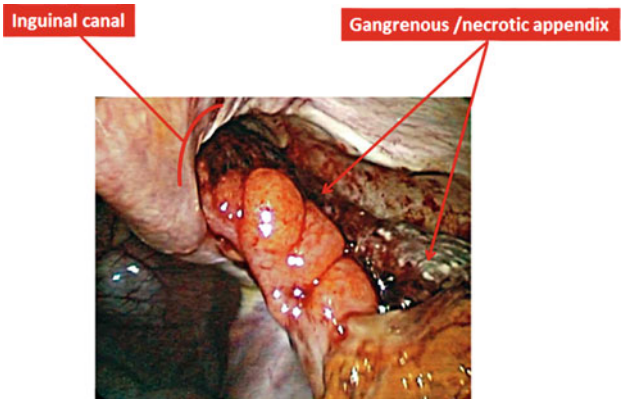

Fig. 2.3 Gangrenous appendicitis in an inguinal hernia (Amyand's hernia). © Dale Dangleben, MD

Recent large, randomized trials have questioned the traditional surgical approach and suggested a nonoperative approach for uncomplicated appendicitis. It is reasonable to individualize the decision to each patient in light of this new data, yet more studies are needed before the surgical option is no longer considered first line given the lack of long-term follow-up studies.

References

1. Bryant DP. Acute appendicitis. In: Britt LD, editor. Acute care surgery. 1st ed. Philadelphia, PA: Lipincott Williams and Wilkins; 2012. p. 538–43.
2. Flum DR. Acute appendicitis—appendectomy or the "antibiotics first" strategy. N Engl J Med. 2015;372:1937–43.
3. Liang MK, Andersson RE, Jaffe BM, Berger DH. The appendix. In: Brunicardi FC, editor. Schwartz's principles of surgery. 10th ed. New York, NY: McGraw-Hill Education; 2015.

Colonic Volvulus

3

Carlos J. Glanville Miranda and Firas G. Madbak

Often a plain film diagnosis

Learning Objectives

1. The learner should be able to formulate a differential diagnosis for large bowel obstruction.
2. The learner should recognize the more reliable diagnostic modalities for cecal and sigmoid volvulus.
3. The learner should be familiar with the available treatment options for cecal and sigmoid volvulus.

Case Scenario

A 63-year-old female presents to a rural hospital with 12 h of severe abdominal distention, obstipation, nausea, vomiting, and progressive abdominal pain. She is tender but not peritoneal on examination. She is afebrile, tachycardic with a normal blood pressure. Abdominal films show both colonic and small bowel distention with multiple air fluid levels. Advanced imaging or endoscopy are not available, so

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the decision is made to proceed with surgical exploration. Operative findings at laparotomy showed a cecal volvulus. She undergoes right hemicolectomy with a primary anastomosis (ileotransverse colostomy).

Epidemiology/Etiology/Pathophysiology

Volvulus is the term used to describe a rotation of the bowel on its own mesenteric attachment (derived from the Latin *volvere*). The incidence of volvulus is greatest in the sigmoid colon, seen at a mean age of 70 years, followed by cecal volvulus which usually occurs at an earlier mean age of 50 years. Volvulus involving the transverse colon or splenic flexure is much less common and will not be discussed here.

Sigmoid volvulus conversely typically occurs as a result of a counterclockwise twist about 15–25 cm from the anal verge (at the rectosigmoid junction). When the sigmoid colon is disproportionately long compared to its mesentery, torsion can occur at 180° or possibly involve a 360° twist where there is a higher risk of strangulation. This occurs in over half of affected individuals. It usually affects the elderly or institutionalized patients and those with psychiatric or neurologic diseases.

Cecal volvulus has two variants, the more common, occurring in 90% of cases, involves a twisting of the cecum or right colon along with the terminal ileum in a (usually) clockwise direction around the axis of its mesentery. In cases of the less common cecal bascule, the cecum folds anteromedial to the ascending colon, causing a flap-valve occlusion at the site of flexion. In order for either of these to occur, there must be a mobile or insufficiently fixed cecum or ascending colon [1].

There is a slight female predominance, and it is most commonly seen in the 6th decade of life. Patients with cecal volvulus tend to be younger than those that present with the more common sigmoid volvulus. Prior abdominal surgery and pregnancy are also considered risk factors [2].

If the volvulus does not decompress, it could rapidly progress to a closed-loop obstruction and strangulation leading to worsening ischemia, necrosis, and ultimately perforation.

Differential Diagnosis

Cecal volvulus, cecal bascule, sigmoid volvulus, small bowel obstruction (adhesive disease, internal hernia, volvulus, and neoplasm), colonic obstruction (diverticular stricture and neoplasm), transverse colon volvulus, and gastric distention.

Diagnosis

An acute presentation characteristic of bowel obstruction—namely with significant generalized abdominal pain and distention sometimes accompanied by nausea, vomiting, and constipation is typical in cecal volvulus. Feculent emesis, again similar to more proximal small bowel obstruction, can be seen in cecal volvulus (yet almost never in sigmoid volvulus).

Chronic relapsing symptoms of distention, pain, and obstipation can also be seen (especially with cecal bascule) but are more common with sigmoid volvulus.

Patients characteristically are seen late in their course. The presence of peritoneal signs and fever indicate possible strangulation. Perforation of the sigmoid is unusual, because the sigmoid colon in older patients is usually thickened.

The diagnosis of sigmoid volvulus can often be made based on a plain abdominal radiograph (Fig. 3.1). The distended large bowel has the appearance of a bent inner tube. Barium enema was used previously for diagnosis and for possible reduction but has falled out of favor and should be avoided if strangulation is suspected, as it is likely to cause perforation. If strangulation with gangrene is suspected, CT of the abdomen may be helpful, especially if a "whirl sign" is present, though not necessary.

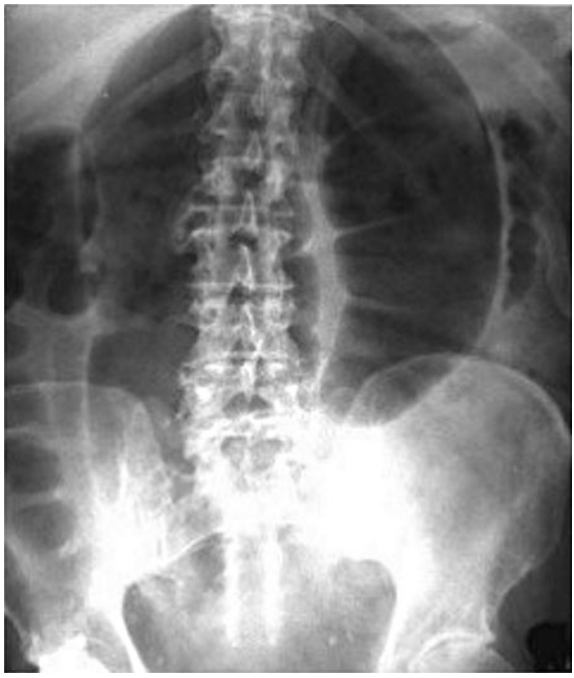

Fig. 3.1 Plain abdominal film with the classic Omega sign of sigmoid volvulus. The sigmoid dilates and twists at the root of the mesentery. © Dale Dangleben, MD

Though radiologic signs may be absent, the diagnosis of cecal volvulus can also be made on plain abdominal films (findings may include a coffee bean sign, a dilated colon segment with air–fluid level in the left upper quadrant) (Fig. 3.2), on barium enema (bird's beak sign, lack of visualization of the right colon or cecum), CT scan, and sometimes upon surgical exploration. When plain films and clinical examination are inconclusive, CT scan tends to be the most reliable imaging modality, with plain films and contrast studies correctly diagnosing cecal volvulus less than 30–40% of the time (Fig. 3.3).

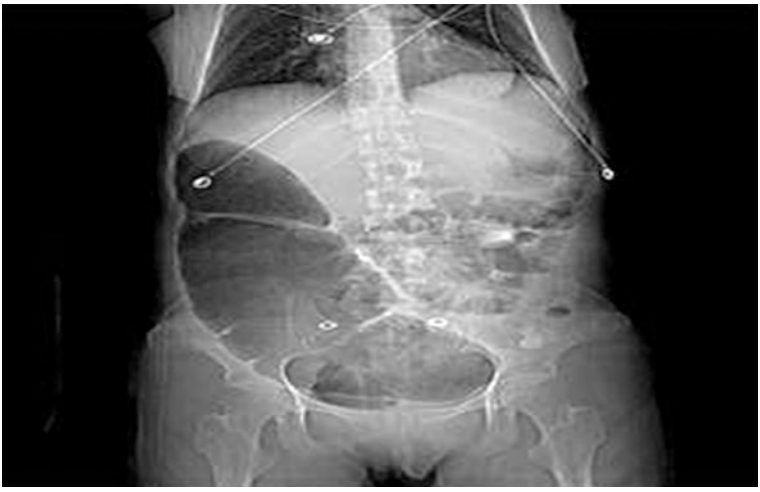

Fig. 3.2 Marked dilation of the cecum with closed-loop obstruction consistent with a volvulus. © Dale Dangleben, MD

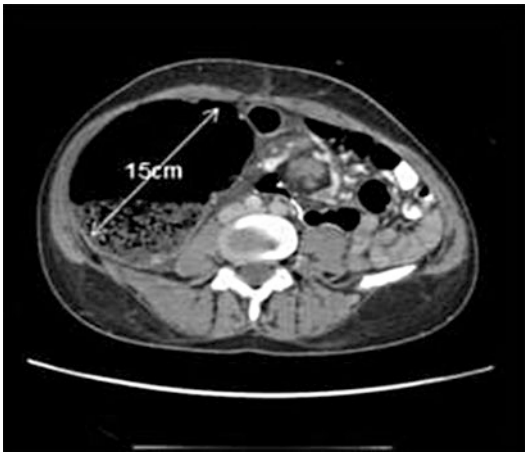

Fig. 3.3 CT scan with 15-cm cecum. © Dale Dangleben, MD

Complications

If the cycle of ischemia is allowed to progress to necrosis and perforation, the morbidity and mortality of this condition is very high, even if successful surgical management is undertaken. Mortality rates have improved however, due to advances in perioperative care and anesthesia.

Management

Sigmoid Volvulus

Nonoperative reduction with decompression is the initial treatment of choice in patients with sigmoid volvulus and no signs of bowel ischemia. At the bedside, using a rigid sigmoidoscope, the patient is placed in the left Sims position, and the scope is passed to the twist. Minor insufflation is used to enable passage of a long, 50 cm, 32–36 French flexible lubricated tube to the dilated proximal colon for decompression. If the point of torsion is more proximal, a flexible sigmoidoscope may need to be used and maneuvered into the proximal colon after which the rectal tube is passed. The tube usually remains in place for 48–72 h, and elective resection is planned during the same admission.

If there is evidence of strangulation or when nonoperative reduction is unsuccessful, then emergency surgery is required. If gangrenous bowel is identified intraoperatively, the involved area must be resected without untwisting the volvulus to avoid releasing inflammatory mediators. If the bowel is viable, resection is still considered the preferred procedure of choice given the lower recurrence rate compared to nonresection procedures. Reconstruction following resection is more contentious; options include colostomy with mucus fistula, a standard Hartmann procedure (sigmoidectomy with end colostomy and a closed rectal stump) or primary anastomosis. Some surgeons elect to leave the bowel in discontinuity after the hemodynamic derangement, and hypothermia and acidosis are corrected after which anastomosis may be more favorable at a second-look operation. The acceptable decision of constructing a primary anastomosis in unprepared left colon should be selectively individualized; if the patient is frail or critically ill, a Hartmann procedure is a much more prudent operation. The surgeon should realize that a significant number of patients never undergo reversal, a procedure that can also often be a tedious and difficult operation.

Cecal Volvulus

Given the significant risk of bowel ischemia, operative management is usually necessary despite some reports of nonoperative decompression by colonoscopy or

barium enema. Recurrence of cecal volvulus can be seen in approximately 15–25% after treatment with simple detorsion, cecopexy, and cecostomy. On the other hand, surgical resection of the mobile segment of colon with a primary anastomosis virtually eliminates this risk and is the treatment of choice unless the patient is too fragile to undergo colectomy. Cecostomy (either a tube or "matured" to the skin) is also associated with higher rates of morbidity and mortality and is no longer favored as a treatment option when compared to detorsion with cecopexy or resection [3].

While cecopexy is advocated by some particularly in the setting of viable colon, detorsion and fixation is associated with a higher recurrence rate, making resection the treatment of choice (Fig. 3.4). As in sigmoid volvulus, the bowel should not be reduced prior to resection to avoid unleashing a cascade of inflammatory cytokines that could lead to irreversible shock. In the setting of necrotic or perforated bowel, right hemicolectomy is definitively the treatment of choice (Fig. 3.5). In cases of questionable bowel viability, a planned second-look operation may be of value. When end ileostomy creation is necessary, the distal, closed colon end can be brought through the same ostomy opening in the abdominal wall ("double-barrel") to facilitate simple closure and restoration of bowel continuity using a local approach.

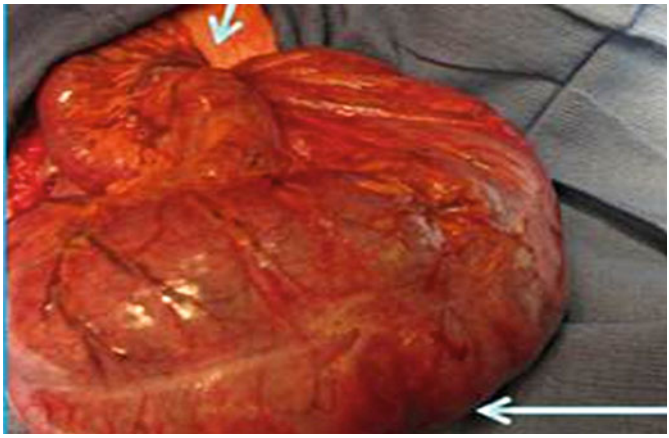

Fig. 3.4 Shows the cecum twisting at the mesentery. © Dale Dangleben, MD

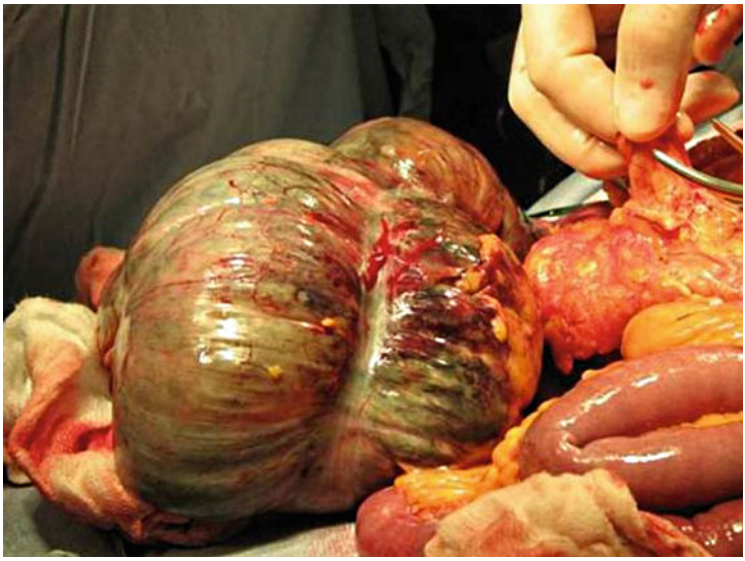

Fig. 3.5 Cecal volvulus with transmural necrosis. © Dale Dangleben, MD

References

1. Madiba TE, Thomson SR, Church JM. The management of cecal volvulus. Dis Colon Rectum. 2002;45(2):264–7.
2. Nemer FD, et al. Volvulus of the colon—a continuing surgical problem. Dis Colon Rectum. 1976;19:321–9.
3. Swenson, et al. Colonic volvulus: presentation and management in metropolitan Minnesota, United States. Dis Colon Rectum. 2012;55:444–9.

Acute Cholecystitis/Biliary Disease

4

Dale A. Dangleben

Open is not a 4 letter word

Learning Objectives

1. Discuss the etiologies of acute cholecystitis.
2. Be able to diagnose acute cholecystitis.
3. Be familiar with the diagnostic modalities used in acute cholecystitis.
4. Recognize the potential complications of acute cholecystitis.
5. Identify the risk and complications associated with cholecystectomy.

Case Scenario

A 36-year-old morbidly obese female presents to the ED with severe right upper quadrant pain. She states that the pain started 10 h prior, and she has worsening nausea and vomiting. She now has a fever of 101.8 °F and heart rate of 112. On examination, the pain is located in the epigastric and right upper quadrant. Her laboratory work shows leukocytosis with left shift and normal liver function tests, amylase, and lipase. She is transferred to radiology for an ultrasound of the gallbladder.

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Epidemiology/Etiology/Pathophysiology

Gallstones are very common, and only about 2% of patients with stones will develop a complication. The complications related to gallstones are cholecystitis, pancreatitis, cholangitis, and choledocholithiasis. Cholecystitis can be categorized into calculous and less commonly acalculous. Acute calculous cholecystitis is inflammation of the gallbladder secondary to a stone obstructing the cystic duct. This accounts for over 90% of patients presenting with acute cholecystitis. This is secondary to cystic duct obstruction by gallstones. If the obstruction persists, the gallbladder continues to distend leading to further inflammation, ischemia, and eventual necrosis.

Acalculous cholecystitis is usually due to a variety of other medical comorbidities, and as the term suggests, there is no evidence of gallstone or obstruction of the cystic duct. It presents in the severely ill such as burn and trauma patients. Cholestasis develops and increases the presence of bacteria. It is not unusual to see empyema, gangrene, or even perforation in this patient population as diagnosis can often be elusive.

Differential Diagnosis

The history and physical examination can help establish diagnosis with reasonable certainty. Elderly patients can sometimes have minimal or vague symptoms. However, based on the anatomic location of the gallbladder, the differential diagnosis is extensive and can include: Acute myocardial infarction, pneumonia, costochondritis, biliary colic, cholangitis, gallbladder cancer, hepatitis, gastritis, peptic ulcer disease, gastric cancer, acute pancreatitis, pancreatic cancer, pancreatic pseudocyst, acute mesenteric ischemia, cholangitis, colon cancer, colonic obstruction, pyelonephritis, and appendicitis.

Diagnosis

The hallmark of cholecystitis on examination is right upper quadrant tenderness. Fever and leukocytosis are frequently present. Ultrasonography remains the most important initial imaging modality in the diagnosis of gallstones. It has a specificity of 95% and sensitivity of 85%. The findings on ultrasound that suggest cholecystitis are pericholecystic fluid and gallbladder wall thickening of greater than 4 mm. Radiographic diagnosis of acute cholecystitis can also be made utilizing *hepatic iminodiacetic acid cholecystoscintigraphy* (HIDA). In fact, HIDA scan has become the procedure of choice in the diagnosis of acute cholecystitis especially when

ultrasonography is inconclusive or equivocal. The sensitivity and specificity in this case is 95% for both. Cystic duct obstruction on HIDA is pathognomonic for acute cholecystitis. During the study, if the biliary tree and the duodenum are visualized with no filling of the gallbladder, then that is suggestive of cystic duct obstruction. Moreover, if the duct is not visualized after 2 h, then obstruction is likely; however, 4–5% of patients will have visualization at about the 4 h interval. This is suggestive of chronic cholelithiasis. Opioid medications may interfere with the study. Cholecystokinin can be administered at the time of the study for the calculation of gallbladder ejection fraction, abnormal being less than 35% [1, 2].

Choledocholithiasis

Choledocholithiasis is the presence of gallstones within the common bile duct. It is estimated that about 10% of patients have choledocholithiasis at the time of cholecystectomy. Choledocholithiasis can be primary and secondary. Primary choledocholithiasis is due to bile stasis within the common bile duct resulting in formation of soft brown pigment stones. The bile stasis is also a nidus for bacteria and increases risk of infection. Secondary choledocholithiasis is due to stones from the gallbladder migrating into the common bile duct. Patients with choledocholithiasis tend to be symptomatic, they present with right upper quadrant or epigastric pain, nausea and emesis, and elevated liver enzymes. Patients may also appear jaundiced; therefore, scleral exam should be conducted. The combination of physical exam, ultrasound, and laboratory data increases the suspicion of choledocholithiasis.

Ultrasonography is the initial study of choice. However, other studies may include endoscopic retrograde cholangiopancreatography (ERCP), magnetic resonance cholangiopancreatography (MRCP), and endoscopic ultrasound (EUS). In the acute care setting, the role of EUS, ERCP, and MRCP are limited. Most patients with suspected choledocholithiasis should have an MRCP preoperatively which avoids inherent risks of routine ERCP (notably, a 5% incidence of post-procedure pancreatitis), many of which are negative, and then proceed to laparoscopic cholecystectomy and intraoperative cholangiography [3]. ERCP is not only diagnostic, but also therapeutic, so it should be considered preoperatively for stone removal in the face of a positive MRCP, followed by elective cholecystectomy. In the most acute setting when the patient must proceed to the operating room for laparoscopic cholecystectomy, an intraoperative cholangiography should be performed.

Cholangitis

Cholangitis is a bacterial infection of the biliary tract. It is characterized by right upper quadrant pain, fever, and jaundice, and it is truly a clinical diagnosis. Cholangitis tends to develop as a result of biliary stasis or obstruction with

subsequent infection in the biliary tract. Choledocholithiasis is the primary cause of duct obstruction. Other causes of obstruction are cholangiocarcinoma, benign strictures, or tumors of the ampulla of Vater. Bacteria are able to enter the biliary tract from the portal system or duodenum. The most common organism is *E. coli* followed by Klebsiella and Enterobacter species.

Mirizzi's Syndrome

Mirizzi's syndrome is the compression of the common hepatic duct from an impacted stone at the cystic gallbladder junction and Hartmann's pouch resulting in obstruction and sometimes obstructive jaundice. It increases the morbidity and biliary injury associated with laparoscopic cholecystectomy. The difficult arises from large gallstones becoming impacted in the cystic duct or in Hartmann's pouch causing a mechanical obstruction of the hepatic duct. This can also result in inflammation in that area which increases the chance of cholangitis. Other potential complications of Mirizzi syndrome are fibrosis and fistulous connection between the gallbladder infundibulum and the common hepatic duct (CHD) or common bile duct (CBD). The recurrent and prolong inflammation causes a higher frequency of gallbladder cancer.

There are two classifications for Mirizzi syndrome currently being used.

The McSherry classification is the original

- Type I—Compression of the common hepatic duct or common bile duct by a stone impacted in the cystic duct or Hartmann's pouch.
- Type II—Erosion of the calculus from the cystic duct into the common hepatic duct or common bile duct, producing a cholecystocholedochal fistula.

A more recent classification system that is used more frequently:

- Type I—External compression of the common hepatic duct due to a stone impacted at the neck of the gallbladder or at the cystic duct.
- Type II—The fistula involves less than one-third of the circumference of the common bile duct.
- Type III—Involvement of between one-third and two-thirds of the circumference of the common bile duct.
- Type IV—Destruction of the entire wall of the common bile duct.

Emphysematous Cholecystitis

Emphysematous cholecystitis is due to gas-forming bacteria, causing infection and inflammation of the gallbladder. Clostridiums are examples of the gas-forming bacteria. Emergent cholecystectomy is indicated, since this finding is characterized by gangrene and higher rates of gallbladder perforation. Mortality associated with emphysematous cholecystitis has been reported greater than 20%. Gas can be seen in the lumen, wall, or even in the pericholecystic area. The diagnosis is made by the ultrasonographic presence of air within the gallbladder wall or lumen. Plain radiographs and CT scan can also make the diagnosis. Of note, diabetic patients are vulnerable to emphysematous cholecystitis.

Porcelain Gallbladder

Porcelain gallbladder is the calcification of the gallbladder. The term "porcelain" came about due to the blue discoloration and brittle texture of the gallbladder. These patients are at increased risk for the development of gallbladder carcinoma. Recently, it has been shown that calcified gallbladder is associated with an increased risk of gallbladder cancer, but at a much lower rate than previously estimated. Gallbladder calcification can be seen on plain film. However, ultrasonography and CT scan are more accurate in distinguishing porcelain gallbladder from a large solitary calcified gallstone.

Management

Acute cholecystitis can be managed based on the severity on presentation. Definitive management is early cholecystectomy (within 24 h of admission). Delayed cholecystectomy, while the norm previously, is no longer favorable and has been shown to be associated with more complications and a more difficult operation as more scar and fibrosis develop. Conversion rates vary depending on acuity; the surgeon should always weigh the risk of persistent attempts at laparoscopy with inherent complications and consider injury to vital structures, significant hemorrhage, or difficulty in identifying critical structures mandatory for conversion to an open cholecystectomy. A subset of uncomplicated patients with biliary colic can be sent home from the emergency room, and laparoscopic cholecystectomy can be scheduled as an outpatient procedure if there are no signs of acute inflammation. Timing and persistence of symptoms can help differentiate biliary colic from acute cholecystitis. The hemodynamically unstable or frail, high operative risk patient should undergo a percutaneous cholecystostomy while being resuscitated.

The admitted patient should be made NPO, start intravenous fluid, antibiotics, and undergo laparoscopic cholecystectomy. In the case where ERCP may be indicated (evidence of choledocholithiasis denoted by hyperbilirubinemia or dilated

common bile duct on imaging), gastroenterology should be consulted preoperatively. Some studies have suggested that postoperative ERCP may reduce length of stay, although there is no strong recommendation with the available data.

Broad spectrum antibiotics should be initiated. The bacteria associated with acute cholecystitis are *Escherichia coli*, Klebsiella, Enterococcus, and Pseudomonas. Piperacillin/tazobactam 3.375 g IV Q6h or meropenem, 1 g IV q8h will provide adequate coverage for acute cholecystitis and its complications.

Choledocholithiasis

There are many modalities now available to make the diagnosis of common bile duct stones. However, in the acute care setting, the patient's hemodynamic status has to be taken into consideration when deciding what modality to use. If the patient is unstable, a percutaneous cholecystostomy is required—but note that this is not a true decompression of the common bile duct. The patient will be best served with ERCP or percutaneous transhepatic cholangiography (PTC) drainage. In the case where the patient undergoes emergent laparoscopic cholecystectomy, an intraoperative cholangiogram should be performed. If CBD stones are seen, then a transcystic or CBD exploration is done based on the stone size. The patients with stones that cannot be extracted or flushed should undergo postoperative ERCP. Open common duct exploration should be considered in the challenging laparoscopic cases should postoperative ERCP be unsuccessful in clearing the CBD. The common duct can be managed with a T tube (14–16 Fr) secured with absorbable sutures and a closed suction drain in the vicinity of the repair. The subset of patients presenting with post-cholecystectomy stones should undergo ERCP.

Cholangitis

The patient's hemodynamic status should be taken into consideration. If the patient is in septic shock they should be admitted to the ICU and fluid resuscitation initiated including any vasopressor support as needed. Antibiotics should be started immediately and cover the common biliary bacteria mentioned prior. The toxic patient should also undergo immediate biliary decompression via ERCP or percutaneous transhepatic cholangiography if amenable. If there is no means for decompression by these routes, then an open common bile duct exploration is warranted with placement of T tube.

Mirizzi Syndrome

Laparoscopic cholecystectomy is the treatment of choice for Mirizzi syndrome. The fibrosis and inflammation poses an operative challenge and care must be taken in

that anatomic location in respect to the CHD and CBD. During the exploration, be cognizant of fistulous erosion in the CBD and also the increased risk of cancer in these patients.

Emphysematous Cholecystitis

As soon as the diagnosis of emphysematous cholecystitis is made, fluid resuscitation and broad spectrum antibiotics should be started. If there is suspicion for perforation preoperative proceed with an open cholecystectomy. Laparoscopic cholecystectomy has become the procedure of choice but keep in mind that emphysematous cholecystitis has a higher rate of perforation. Laparoscopy is safe and effective but depends on operator comfort level. Use clinical judgement in the use of drain in the gallbladder fossa postoperatively. Antibiotic regimen should be continued post operatively until clinical improvement.

Porcelain Gallbladder

It has been shown recently that the risk of malignancy is not as exaggerated as it was in the past. Laparoscopic cholecystectomy is sufficient paying attention to spillage into the peritoneal cavity.

Complications

One must recognize the potential complications associated with acute cholecystitis especially in urgent cases. Also, very important is the vigilance during surgery in these patients. Attention to the critical view during the dissection and confirmation of anatomy division of structures is imperative. While some surgeons advocated routine intraoperative cholangiography, this is still controversial. A more selective approach is preferred, using the modality only when common bile duct stones are suspected preoperatively (and have not yet been treated with ERCP), or when intraoperative anatomy is anomalous or difficult to visualize. In cases of recognized iatrogenic common duct injury, it is recommended that the surgeon seek the help of more experienced surgeons with hepatobiliary experience. If unavailable, the area should be drained widely and the patient transferred to a tertiary care facility with biliary tract reconstruction capabilities.

Late complications of cholecystectomy include retained common bile duct stones (defined as within 2 years of surgery) or biliary stricture. A stone in the common duct postoperatively requires ERCP with sphincterotomy and possible stent placement. Postoperative biliary strictures are thought to result from ischemia

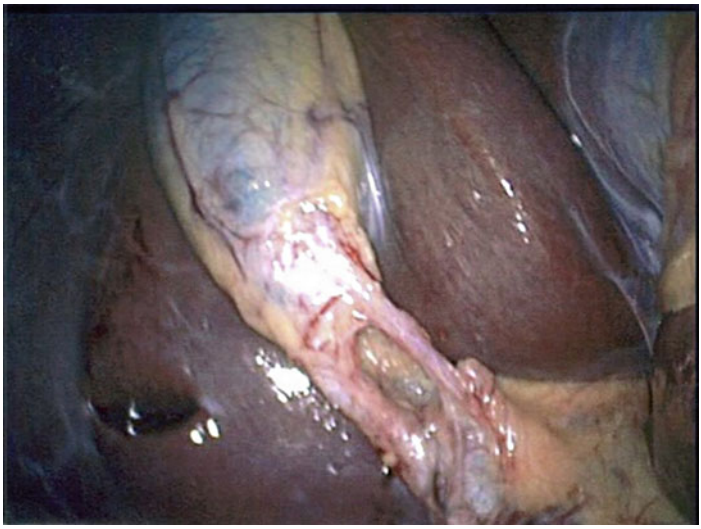

Fig. 4.1 View if the cystic duct and artery dissected during laparoscopic cholecystectomy. © Dale Dangleben, MD

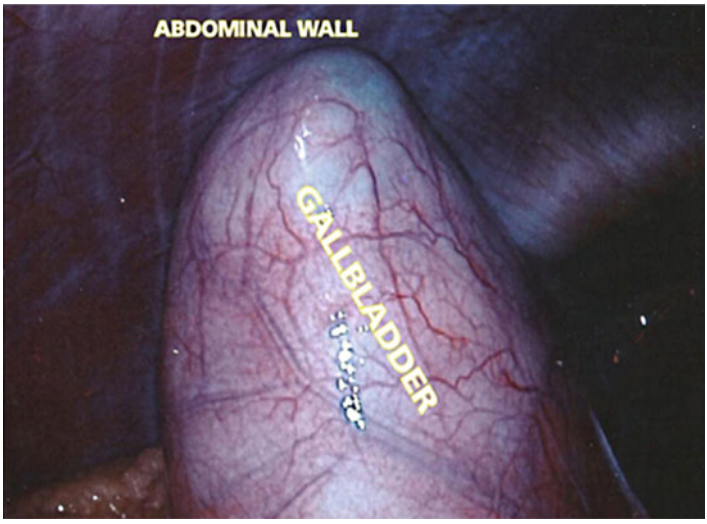

Fig. 4.2 Distended gallbladder with the tip of the funds touching the abdominal wall. This is known as Hydrops of the gallbladder. © Dale Dangleben, MD

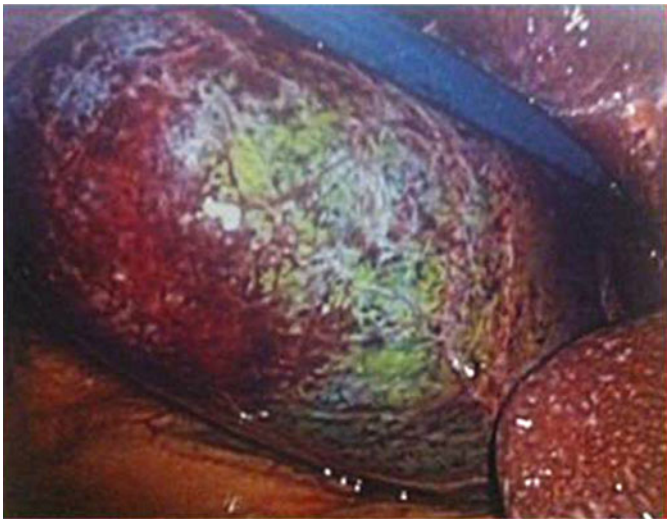

Fig. 4.3 Gangrenous cholecystitis. © Dale Dangleben, MD

of the bile duct possibly caused by surgical dissection and compromised blood supply to the bile duct during cholecystectomy (Figs. 4.1, 4.2, and 4.3).

References

1. Gurusamy KS, Samraj K. Early versus delayed laparoscopic cholecystectomy for acute cholecystitis. Cochrane Database Syst Rev. 2006;4:CD005440.
2. Kopetz ES, Magnuson TH, Duncan MD, et al. Complications of neglected cholelithiasis account for significant surgical mortality in the elderly. Surg Forum. 2000;51:489–91.
3. Magnuson TM, Bender JS, Duncan MD, et al. Utility of magnetic resonance cholangiography in the evaluation of biliary obstruction. J Am Coll Surg. 1999;189:63–72.

Diverticulitis of the Colon

5

Sergio E. Perez

Two strikes and you're out no longer stands

Learning Objectives

1. The learner will be able to formulate a differential diagnosis of left lower quadrant pain.
2. The learner will describe the spectrum and manifestations of diverticular disease.
3. The learner will be able to differentiate the management of uncomplicated and complicated diverticulitis.
4. The learner will be able to recognize, diagnose and treat potential complications of acute and chronic diverticular disease.

Case Scenario

A 45-year-old man presents to the emergency department with several days of constipation, left lower quadrant pain, fever and chills. His temperature is 38.5 °C. A plain film of the abdomen is unremarkable. A CT scan of the abdomen demonstrates sigmoid diverticula, pericolonic stranding and no evidence of abscess

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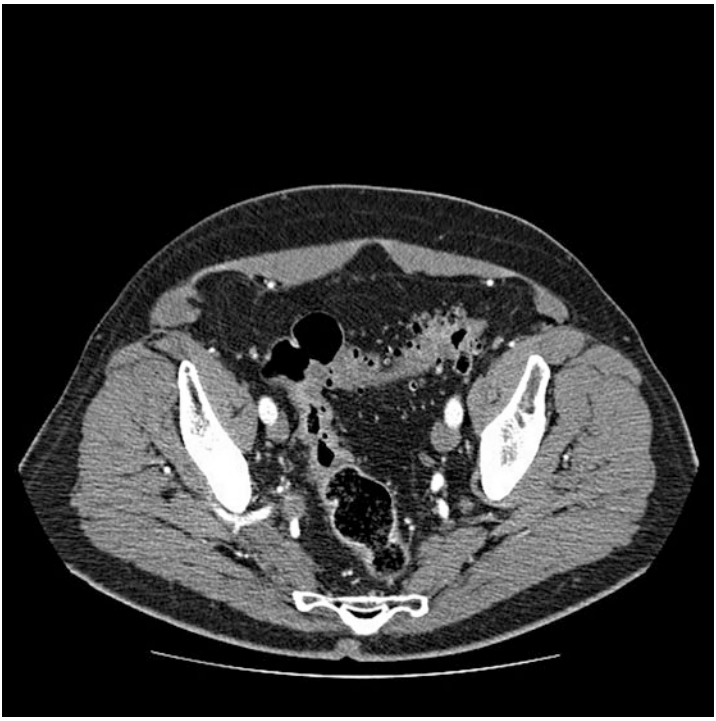

Fig. 5.1 CT scan showing extensive diverticular disease. © Dale Dangleben, MD

or perforation (Fig. 5.1). He is treated with bowel rest and intravenous antibiotics. He improves, starts tolerating diet and is discharged home after 4 days.

Seven months later, the patient returns with similar symptoms, and a CT scan shows pericolonic stranding and free air.

Epidemiology/Etiology/Pathophysiology

Diverticular disease collectively encompasses a spectrum of pathology including diverticulosis and diverticulitis. Particularly in Western populations, it is present in 30% of individuals over 60 years of age and in 60% of those over 80 years of age. Diverticulitis most commonly affects the sigmoid colon (95%) of elderly patients. It is seen equally in males and females. In 35% of patients, the proximal colon is also involved. Risk factors include low-fiber diet, tobacco use and chronic constipation. Typically, these are false (pulsion) diverticula involving herniation of mucosa and muscularis mucosa through the muscularis externa that occur at an area of weakness (near the vasa recta) [1].

Differential Diagnosis

Infectious, inflammatory and ischemic etiologies of colonic pathology need to be considered. Additionally, large bowel obstruction from a neoplastic process may occasionally present with similar symptoms. In females, gynecologic pathology (e.g., ruptured ovarian cyst or pelvic inflammatory disease) needs to be considered. In the presence of concomitant urinary complaints, renal colic from nephrolithiasis and pyelonephritis is included in the differential.

Diagnosis

Patient presentation could range from asymptomatic state to life-threatening complications of acute intraabdominal sepsis. Uncomplicated disease (occurring in 75–80% of cases) manifests as abdominal pain, fever, leukocytosis and anorexia or obstipation [2]. Complicated disease is associated (by definition) with abscess (Hinchey I and II) or perforation (Hinchey III and IV). More rarely, fistulae and stricture may develop (see below).

Diagnosis is best made using CT scanning, with findings of diverticula along with colonic thickening and inflammation or stranding within the pericolonic fat.

Complications

Complications can be categorized as acute (perforation and abscess) or chronic (stricture and fistulae). Perforated diverticulitis that presents with a few smaller pockets of extraluminal air needs to be differentiated from free intraperitoneal air over the liver on CT imaging. The latter needs to be treated emergently with fluid resuscitation, broad-spectrum antibiotics and urgent laparotomy. Classically, a Hartmann procedure (sigmoidectomy with end colostomy) is performed.

Larger abscesses (>4 cm) without free perforation in a stable patient are usually amenable to percutaneous drainage techniques and antibiotics without surgical intervention.

Chronic inflammation from diverticulitis can occasionally lead to stricture formation. Particularly prevalent in the elderly, these strictures affect short segments and may be treated with dilation. Expandable metal stents have been utilized in select cases, but remain controversial. A full colonoscopy after recovery is mandatory to rule out malignancy as in the case of acute uncomplicated diverticulitis (Fig. 5.2).

Fistulae, most commonly colovesical, will present with fecaluria or pneumaturia in the vast majority of patients. Imaging modalities for diagnosis include CT scan or barium enema; however, cystoscopy remains the most accurate [3].

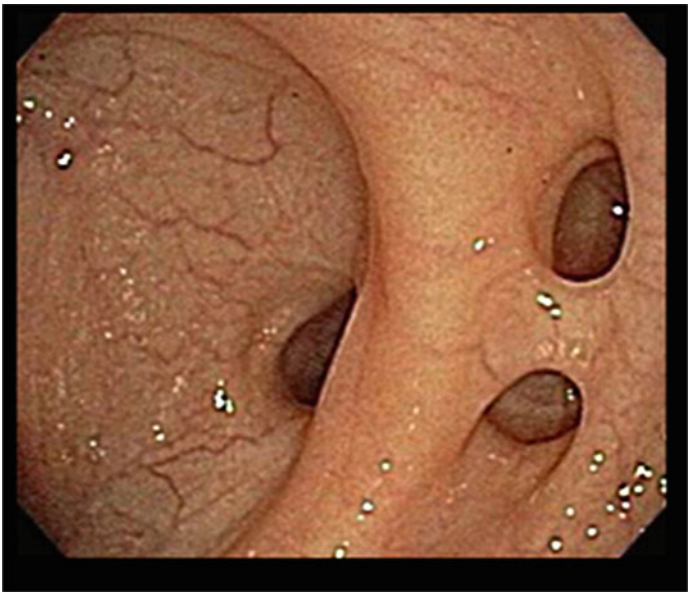

Fig. 5.2 Endoscopic view of diverticular disease. © Dale Dangleben, MD

Treatment is laparoscopic or open fistula resection. The bladder defect is usually small and is typically left alone or less frequently closed with absorbable sutures or covered with omentum. Bladder decompression with a transurethral Foley catheter continues for 5–10 days postoperatively, at which point a cystogram is done to ensure bladder healing before catheter removal.

Less commonly, colovaginal (in a patient who has undergone previous hysterectomy), coloenteric and colocutaneous fistulae may form as well.

Management

In mild, uncomplicated disease, bowel rest and antibiotics are the mainstay of therapy. Almost 75% of patients who require hospitalization will respond to non-operative management with appropriate antibiotic therapy. Recommended regimen is trimethoprim–sulfamethoxazole or a quinolone plus metronidazole. If no clinical response is observed, ampicillin or piperacillin is added for enterococcal coverage. Duration of antibiotic therapy is 7–10 days.

Abscesses that develop as a result of complicated diverticulitis are drained percutaneously while the patient is maintained on antibiotics. Colonoscopy after recovery is necessary to evaluate the colon for possible malignancy, usually after approximately 6 weeks.

In the presence of perforation, a Hartmann procedure is recommended. More recent prospective data have shown that primary anastomosis with a protective, proximal diverting loop ileostomy even in the setting of perforation with contamination may be acceptably safe. More long-term studies are needed; however, this may be used selectively given that a significant number of end colostomies created for this presentation are never reversed.

Considerable controversy exists over the question of definitive surgical treatment of recurrent uncomplicated disease. Treatment should be individualized on a case-by-case basis, taking into consideration the patient's age, comorbidities and severity of attacks. Delayed elective resection is indicated after an episode of complicated disease or after an episode of diverticulitis in young or immunocompromised patients. Historically, elective resection with primary anastomosis was recommended after two attacks; however, the trend is moving toward more conservative nonoperative management after recent studies have challenged this with series showing a very low incidence of recurrence with free perforation.

References

1. Rocco R, Baxter N, Read T, et al. Is the decline in the surgical treatment for diverticulitis associated with an increase in complicated diverticulitis? Dis Colon Rectum. 2009;52:1558–63.
2. Rocco A, Compare D, Caruso F, Nardone G. Treatment options for uncomplicated diverticular disease of the colon. J Clin Gastroenterol. 2009;43:803–8.
3. Strate L, Liu Y, Syngal S, et al. Nut, corn, and popcorn consumption and the incidence of diverticular disease. JAMA. 2008;300(8):907–14.

Enterocutaneous Fistula

6

Tyrone Galbreath and Lindsey Perea

Every fistula is different; tailor your approach.

Learning Objectives

1. The learner should be able to formulate a differential diagnosis for enterocutaneous fistula (ECF).
2. The learner should know the risk factors and associations of ECF.
3. The learner should know the diagnostic pathway for ECF.
4. The learner should have an understanding of the phases of management in a patient who presents with ECF.
5. The learner should know surgical options for management of ECF.
6. The learner should be aware of enteroatmospheric fistulas (EAF) and some of the challenges involved in management.

Case Scenario

A 56-year-old female was postoperative day eight from sigmoid colectomy with primary anastomosis performed for acute diverticulitis. An inflamed adjacent segment of small bowel was resected and primary anastomosis performed.

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Her postoperative course was complicated by prolonged ileus and intermittent fever. Her surgical wound became increasingly erythematous and fluctuant, requiring opening of the wound at bedside.

Drainage output increased, is collected in an ostomy appliance, and measured to be 750 ml per day. A CT of the abdomen and pelvis with oral and IV contrast showed a fistulous tract extending from the mid-jejunum to the anterior abdominal wall. No distal obstruction or abscess was discovered. Fluid and electrolyte resuscitation took place, a somatostatin analogue was initiated with decreased fistula output, and surrounding skin was protected. At 4 months postoperatively, the fistula output remained at 200 ml per day, despite nutrition optimization and absence of ongoing infection. At subsequent operation, the affected segment of small bowel was resected, primary bowel anastomosis was performed, and the patient's abdomen was closed without further complication.

Epidemiology/Etiology/Pathophysiology

Enterocutaneous Fistulas (ECFs)

A fistula by definition is an established connection between two epithelialized surfaces—between bowel and skin in the ECFs. The naming convention is that of naming the two epithelial surfaces such as colocutaneous, gastrocutaneous, or jejunocutaneous. The fistulas are classified as low output (<200 ml/day), moderate output (200–500 ml/day), and high output (>500 ml/day).

The etiology of fistula formation occurs most often following abdominal surgery and is related to tissue integrity and healing. The presence of inflammation, infection, or retained foreign body can all represent contributing factors. ECFs may be postoperative or spontaneous; low, moderate, or high output; or denoted by location in the bowel. Proximal ECFs tend to be high output (>500 ml/day), while distal ECFs, such as colocutaneous, tend to be low output ($\leq$ 200 ml/day).

Over 75% of ECFs are postoperative, and only 15–25% are the result of trauma, cancer, irradiation, IBD, and ischemic or infective diseases (TB) combined. Detection time is commonly at 7–12 days postoperatively, and detection is often preceded by a prodrome of a slow postoperative course with fevers and prolonged ileus, culminating in the development of wound erythema and finally breakdown and drainage.

Spontaneous ECFs occur in the setting of inflammation, irradiation, or malignancy. Inflammatory causes range from inflammatory bowel disease to pancreatitis and ischemic bowel. These types of fistulas are more likely to close non-operatively, but then also have a slightly higher risk of recurrence than postoperative ECFs. Some espouse that even with spontaneous ECFs that close non-operatively, surgical resection and reconstruction of bowel continuity are preferred to decrease recurrence risk [1].

Improved ICU care, parenteral nutrition, and increased intelligence of antibiotic use have driven mortality rates for ECF from 50% in the early twentieth century to 20% in the early twenty-first century. The three major causes of morbidity and mortality among ECF patients are fluid and electrolyte depletion, malnutrition, and sepsis. In high-output fistulas, analyzing the components and electrolytes of a fistula's output may be beneficial to maintaining early balance in the patient.

Multiple factors are considered as poor prognostic factors when predicting the likelihood of spontaneous closure, and these include high fistula output, short fistula tract (<2 cm), large fistula (diameter >1 cm), abscess, distal bowel obstruction, foreign body, malignancy, radiation, steroids, and chronic epithelization.

Preoperative factors may increase the risk of ECF and include malnutrition, infection, and emergency procedures with concomitant hypotension, anemia, hypothermia, or poor oxygen delivery. Aggravating factors should be corrected prior to operation with nutritional support, bowel preparation, and control of physiologic parameters such as cardiac output, blood glucose, and anemia. Mechanical and bowel preparation along with preoperative antibiotics will further decrease the incidence of intraabdominal and wound infection and abscess, thus further reducing the likelihood of developing an ECF. Intraoperatively, fastidious surgical technique, avoiding hematoma, and being conscientious to repair any and all enterotomies, is the primary means by which a surgeon may decrease risk of ECF or EAF [2].

Spontaneous ECFs are less likely to close without surgical intervention than are postoperative fistulas. In spontaneous fistulas that are the result of inflammation, it is not uncommon that an ECF will close with non-operative measures. There may be recurrence, however, when a diet is resumes. In the postoperative patient that has inflammatory bowel disease, distinguishing whether the fistula occurred in a healthy segment or in a diseased bowel segment may predict care. Known factors effecting whether an ECF is likely to spontaneously close include anatomic location, tract length, fistula output, surrounding bowel, etiology, nutritional status, and absence or presence of sepsis. Regarding non-operative closure of fistula, the success rate in retrospective studies is 30–65% and for combined methods, the success rate was 80%.

Enteroatmospheric Fistulae (EAF)

An enteroatmospheric fistula is a defect from the bowel directly to the outside world. They can be associated with loops of exposed bowel that gets desiccated, unrecognized enterotomy or bowel injury, or anastomotic failure. Independent risk factors according to the AAST trauma registry are large bowel resection, large-volume resuscitation, and an increased number of abdominal reexplorations. They can be categorized as superficial or deep, depending on how close to the abdominal surface the enterotomy is.

A superficial exposed fistula drains atop the granulating wound of a frozen abdomen or 'visceral block' and primarily presents a nutritional and wound management problem. There is almost no tract, or at best a very short tract. Placing an

appliance or controlling the effluent may be quite difficult as emphasized by the term 'floating stoma.' A deep fistula drains intestinal contents directly into the peritoneal cavity, causes peritonitis, and is characterized by uncontrolled infection and may require emergent surgical intervention.

In the era of increased use of open abdomen for damage control laparotomy, trauma, and emergency abdominal surgery, the number of EAFs has increased dramatically. Multiple factors may contribute: Patients may be surviving when historically they previously would have not, bowel is being exposed to external elements and not protected by abdominal wall, and EAFs previously may have developed into ECFs.

Differential Diagnosis

ECF

Differential diagnoses include anastomotic leak, unrecognized enterotomy, fascial suture through bowel, ischemic bowel segment, erosion into bowel by suction drain or mesh, perforated viscous (e.g., peptic ulcer or colonic diverticula), and local inflammation (e.g., Crohn's disease).

EAF

Differential diagnoses are essentially the same as those for ECF but more narrow as the EAF is often superficial and can be visually inspected closely.

Diagnosis

ECF

In some postoperative patients, the diagnosis may be readily apparent based on the appearance of the fistula exit site and the quality of the drainage or enteric content in the wound bed. Patients may present with abdominal pain, distension, tachycardia, sepsis, fever, drainage of enteric contents from wound, and peritonitis. Demonstrating the communication between the bowel and skin, typically with a contrast study, makes the diagnosis of enteric fistula.

Three pertinent imaging modalities include GI contrast studies (UGI series, BE), CT scan, and fistulogram. CT imaging with oral and IV contrast is the imaging modality of choice as it may demonstrate intraabdominal abscess, other fluid collections, areas of distal intestinal obstruction, or even pneumobilia. As second-line imaging, in a fistula with a well-defined cutaneous opening and absence of sepsis, a

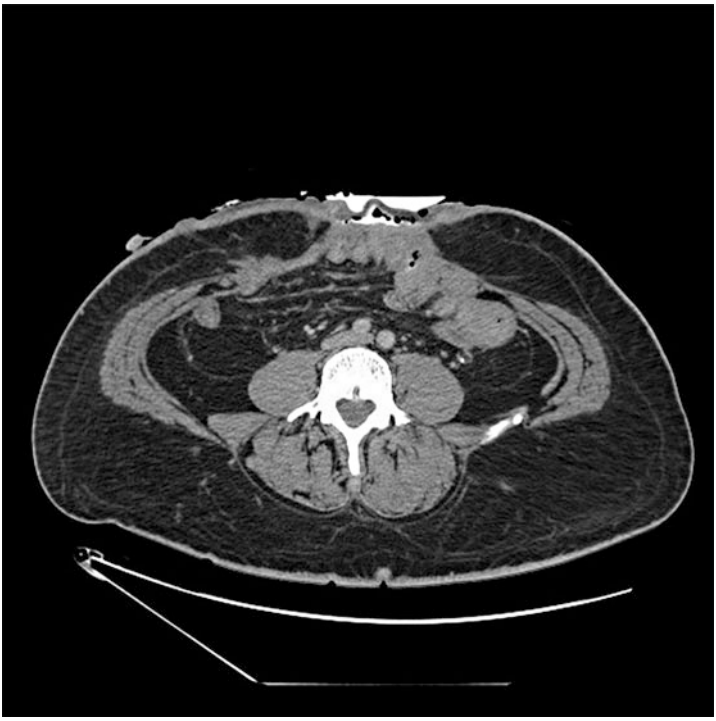

Fig. 6.1 CT scan with extravasation of oral contrast out the anterior abdominal wall consistent with an enteroatmospheric fistula. © Dale Dangleben, MD

fistulogram may be performed to document intestinal continuity and evaluate for distal obstruction. The fistulogram rarely identifies the specific origin of the tract and does not show the additional information that a CT scan would Fig. 6.1.

In cases where a fistula may be small and not apparent on contrast imaging, use of dye (e.g., indigo carmine, methylene blue, or charcoal) may be used. In fistulas arising from the rectum or stomach, endoscopic evaluation may provide additional information (e.g., neoplasm) and allow access to the origin of the fistula.

EAF

With the frequent tendency of being superficial, the diagnosis is oftentimes readily apparent. Anatomic information and output volumes remain important for prognosis and therapy choices.

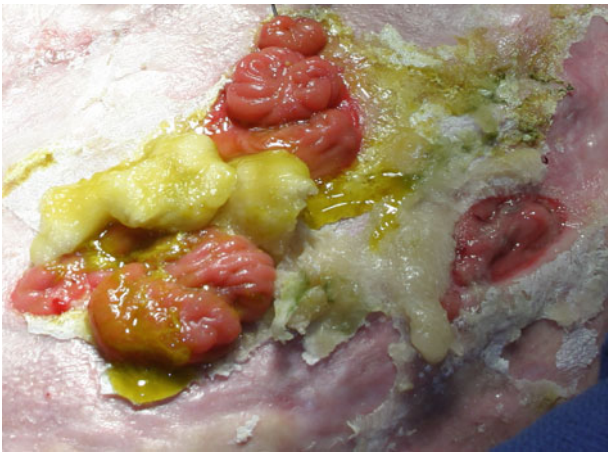

Fig. 6.2 Multiple fistulae of the lower abdomen. © Dale Dangleben, MD

Complications

ECF

Electrolyte disturbances, malnutrition, sepsis, skin breakdown and maceration, abscess formation, dehydration, protracted morbidity, and even multisystem organ failure Fig. 6.2.

EAF

In addition to above, additional EAFs may occur during the treatment process as additional bowel segments are often exposed.

Management

ECF

Management is divided into phases of: (1) stabilization—initial 24–48 h (fluid resuscitation, drainage of intraabdominal abscesses, control of fistula drainage and skin care, reduction of fistula output, and aggressive nutritional support), (2) investigation/diagnosis—7 to 10 days (imaging to determine anatomy and fistula characteristics), (3) decision—up to 4–6 weeks (decide between operative or

non-operative), (4) definitive therapy—4 to 6 months following the index operation (closure of fistula, reestablish GI continuity, and secure closure of abdomen), and (5) healing—5 to 10 days after closure onward (transition to oral intake and continue to ensure adequate nutritional support).

Determining management depends on likelihood of spontaneous closure, and decision points include fistula location and characteristics, pattern of fistula output, status of the surrounding bowel, and response to non-operative management. If fistula closure has not occurred after 4–5 week of adequate nutrition and absence of sepsis, it is 90% unlikely that a fistula will close without surgical intervention. For postoperative ECF's, morbidity and mortality have been noted to decrease by 50% in those patients that are able to wait 4–6 months from the date of original procedure to have surgical intervention. The wait allows the obliterative peritonitis and dense fibrous adhesions to subside, resulting in reduced morbidity and mortality.

In patients with inflammatory bowel disease, managing the active disease process is key and includes use of medication such as infliximab and tacrolimus. Introduction of such medications has been shown to improve spontaneous closures of fistulae in this subset of patients.

Non-operative management includes nothing per mouth and parental nutrition for proximal fistulae, optimization of nutritional status, protecting surrounding skin from the drainage, elimination of sources of infection and drainage of any abscesses, and removing foreign bodies (e.g., mesh) or sources of distal obstruction. Importantly, TPN has not been shown to decrease mortality. Instituting TPN as an adjunct after sepsis resolves may be feasible, but enteral nutrition is preferred as it preserves the gastrointestinal mucosa and supports immunologic and hormonal functions of the gut and liver. Methods for enteral nutrition include feeding by mouth, naso-enteric tubes, or even fistuloclysis (feeding tube through the fistula) [3].

Neoplasia, radiation, and inflamed segments of bowel are less modifiable risk factors and require resection to healthy bowel if that exists as an option. Somatostatin and its analogues may be used to reduce fistula output, but have not been shown to decrease healing time or improve spontaneous closure rates. However, it has been shown in fistulae producing >500 ml/day that somatostatin is effective in maintaining volume status and skin integrity by decreasing fistula output. Widespread somatostatin use for all fistulas is not indicated as benefits may be modest, but the medication is very expensive, and side effects may include cholecystitis, hyperglycemia, mucosal atrophy, and interruption of intestinal adaption. Some authors suggest a trial period of 48 h; if reduction of output occurs, then the medication can be continued.

Nasogastric drainage is useful only in cases of distal obstruction or ileus. Widespread use may result in alar necrosis, sinusitis, distal esophageal stricture, and aspiration pneumonia.

Prior to surgical repair, patients should ideally be nutritionally replete, free of infection, and have supple soft tissues adjacent to the fistula. It is advisable for postoperative fistulas that have not closed with non-operative measures, to wait 4 months for obliterative peritonitis to subside prior to surgical intervention. The surgical procedure commences with what some have labeled 'refunctionalization,'

where all abscesses are drained, all adhesions are lysed, and all areas of obstruction are corrected. Resection of the segment of bowel containing the fistula and primary anastomosis is the definitive maneuver. Wedge resections, oversewing of fistula, and bypasses or Roux-en-Y bypass in general are less preferred and have high recurrence rates. If the affected segment cannot be mobilized enough for resection, the procedure of choice is to exteriorize the distal and proximal ends of the fistula as an ostomy which can later be taken down. The final step is a secure abdominal wall closure, whether primary closure is possible or with the assistance of a plastic surgeon to assist with closure or to create flaps.

Duodenocutaneous fistulae, although the majority overwhelmingly close without intervention, operatively represent a unique challenge in that the resection strategy is not effective secondary to the anatomy. Surgical options include pancreaticoduodenectomy or the preferred gastrojejunostomy (without vagotomy) and feeding jejunostomy.

Alternative techniques of management include fibrin sealants and plugs, core matrix or porcine bladder matrix plugs, and various diversion and buttressing techniques. These techniques remain experimental with ongoing research to determine effectiveness.

Postoperative paralytic ileus is often prolonged, and a period of decompression with a nasogastric tube is helpful. Antibiotics are commonly continued for 72 h postoperatively. A minimal daily caloric intake of 1500 calories is deemed necessary prior to parenteral nutrition being discontinued.

EAFs

EAF management considerations vary from ECF management in that EAFs require more novel techniques to manage effluent, bowel is often exposed and must be protected, and EAFs lack the ability to close spontaneously as there is no overlying soft tissue coverage. Some authors recommend an early attempt at 'sealing the leak' by a patching technique where fibrin glue and Alloderm are applied as soon as an EAF is recognized. Attempt at suture closure of the fistula is generally ineffective.

The bowel that often gives rise to EAFs is often called a visceral block or frozen abdomen in that the adhesiolysis required to perform resection and anastomosis is often not feasible at the time of discovery. Remaining options in order of preference include exteriorizing the fistula, diverting proximally if able, intubating the fistula with a tube (controversial), and lastly a 'floating stoma' where the fistula effluent is controlled by plastic sheeting affixed to the periphery of fistula. Some authors recommend the use of muscle flaps such as rectus abdominis to enhance blood supply to the EAF area.

A negative pressure dressing or wound vacuum-assisted closure device is utilized to enhance granulation tissue of the remainder of the abdominal wound. Subsequently, the granulation bed is then skin-grafted, and thereby, the EAF is effectively converted to an ECF with associated hernia. The abdomen is allowed to epithelize during the ensuing 4–6 months of trialed non-operative closure. If the

EAF does not close non-operatively, the definitive surgical closure of the fistula should not be considered until the skin graft is supple and can be pinched between the thumb and index finger, which signifies the existence of a plane between the graft and underlying bowel. The definitive operation is resection of the fistula with its associated bowel and then repair of the hernia defect, frequently with a component separation technique.

References

1. Brunicardi F, Andersen D, Billiar T, Dunn D, Hunter J, Matthews J, Pollock R. Schwartz's principles of surgery. 9th ed. New York, NY: McGraw-Hill; 2009.
2. Fischer J, Evenson A. Gastrointestinal-cutaneous fistulae. In: Mastery of surgery. 6th ed. Philadelphia, PA. Lippincott Williams & Wilkins; 2011.
3. Galie K, Whitlow C. Postoperative enterocutaneous fistula: when to reoperate and how to succeed. Clin Colon Rectal Surg. Nov 2006;19(4):237–46.

Fournier's Gangrene

7

K. Michael Hughes

Think about this diagnosis in any male patient with shock and a scrotal lesion

Learning Objectives

1. Learner should understand the pathophysiology and natural history of Fournier's gangrene.
2. Describe risks for mortality from Fournier's gangrene, and understand principles for prediction of mortality.
3. Understand and describe the role of antimicrobial therapy in treating Fournier's gangrene, especially in relation to the microbiology of the disease.
4. The learner should understand priorities of management and treatment strategies to successfully treat Fournier's gangrene.

Case Scenario

A 65-year-old male patient was admitted to the hospitalist service for uncontrolled diabetes mellitus. He has been additionally known to the admitting service for previous management of his diabetes and chronic renal insufficiency, not requiring dialysis; he is obese. The patient's only physical complaint at admission was that of mild discomfort in his perineal area, mostly beneath his scrotum without appreciable physical findings.

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During the first 48 h of admission, blood sugar became increasingly difficult to control and the patient began to complain more about generalized discomfort and pain in his scrotal and perineal region. At the end of the second hospital day, the patient became febrile to 102.5 F. CBC showed a leukocytosis; blood sugar was elevated; serum sodium was 138; hematocrit was 22 (% × 100); serum creatinine was 3.7 mg/dL; CXR was unremarkable; and a CT scan of the abdomen and pelvis was obtained that revealed soft tissue gas in the perineal/pubic region. Surgical consultation was requested.

Surgical consultation showed revealed the patient with labile blood pressure, MAP in the high 50's mmHg, tachycardia, fever of 103, and complaining of pain in the perineal area. Physical examination showed pubic, scrotal, and left inguinal crease erythema with a small area of scrotal skin necrosis and scrotal edema. Mild crepitus of subcutaneous soft tissue was notable in the area of the left inguinal crease. The patient was immediately transferred to the surgical intensive care unit for resuscitation of his clinical septic shock.

In the ICU, the patient was immediately resuscitated with crystalloid infusion and blood transfusion with a target Hct of 30. This did not sufficiently correct the low MAP, and the patient was then started on pressor therapy. Blood cultures were obtained, and the patient was started on broad-spectrum antibiotics with piperacillin–tazobactam for severe soft tissue infection.

Following resuscitation from systemic sepsis, the patient was emergently taken to the OR for wide surgical debridement of devitalized scrotal and perineal skin. Operative excision was limited to the point at which subcutaneous fat was no longer easily separated from overlying skin. Scrotal skin, left inguinal skin, and bilateral spermatic cord skeletonization provided grossly adequate initial debridement. Tissue specimens were submitted to the laboratory for tissue culture. The operative site and testicles were dressed with Dakin's solution soaked gauze.

The patient was returned to the OR daily for completion debridements until all devitalized tissue was removed and infectious progress was arrested. Antibiotic coverage was tailored according to culture results which showed polymicrobial flora including *Bacteroides fragilis*, *Escherichia coli*, and *Staphylococcus aureus*. Following infection control with surgery and antibiotics, plastic surgery consultation provided definitive coverage with a rotation flap. The patient survived and was discharged from the hospital and ultimately enjoyed a full functional recovery.

Epidemiology/Etiology/Pathophysiology

Since first being described in 1764 by Baurienne, and later ascribed to Fournier in 1883, Fournier's gangrene (FG) has remained a morbid condition with a high mortality. FG is a rapidly progressive necrotizing fasciitis of the perineum. It is characterized by violation of skin integrity and infection that rapidly progresses by

way of obliterative endarteritis of subcutaneous arteries which results in a synergistic polymicrobial infection that rapidly perpetuates the condition. An inciting source can be identified in up to 90% of cases, and the microbiology of the fasciitis is often related to the cause. FG has a tenfold higher incidence in males, yet can also be seen in females. In women, Bartholin's cyst abscess, episiotomy, and other insults to the female genitourinary tract may cause this necrotizing fasciitis of the perineum. In both genders, numerous causes are described that may involve the regional perineal area, such as anorectal disease; surgical manipulations such as band ligation of hemorrhoids, fistula-in-ano; perirectal abscess; boils; and local trauma, to name a few. More remote causes include perforated diverticulitis, perforated acute appendicitis, rectosigmoid carcinoma, and bladder cancer. FG begins in the perineal region, but may spread to the inguinal areas and extend to the abdominal wall.

FG remains a disease primarily affecting adult males, usually in their 50s and 60s with approximately 1000 cases reported annually in the USA. However, FG has been reported in women as well as children and even very rarely in infants. Mortality rates are reported to be between 4 and 70% and are highly dependent on comorbidities and extent of disease at the time of treatment. As much of the literature on FG is based on limited patient volume and case reports, mortality figures vary widely. Overall, the mortality rate has remained about 20%. Inadequate treatment or treatment excluding surgery assures death.

Diabetes mellitus is the most common comorbidity associated with the development of FG. However, diabetes has not been shown to increase mortality. Immunosuppression may also play a role in the development of FG, and in areas where HIV is endemic, it may be replacing diabetes mellitus as the most common comorbid condition. Age greater than 60 and obesity are also associated with the development of FG but do not necessarily affect mortality. In studies describing and validating scoring systems for FG, renal insufficiency/failure significantly increases mortality. Various risk-for-mortality scoring systems have been described and include laboratory and clinical parameters. The Fournier's Gangrene Severity Index (FGSI) is the most widely used. During initial evaluation, an FGSI score of >9 has shown to predict a 75% likelihood of death and an index score of <9 has been associated with a 78% probability of survival. Of the 9 parameters in the FGSI, creatinine, hematocrit, and potassium may be most important for predicting mortality [1].

The microbiology of FG is that of a rapidly progressive synergistic polymicrobial necrotizing infection. Organisms can often be predicted by the underlying condition. The most common organisms are *E. coli*, Proteus, Enterococcus, and Bacteroides being the most common anaerobic organism. In diabetic patients, Streptococcus and Staphylococcus organisms are most commonly isolated in addition to others [2].

Differential Diagnosis

Early in the course of FG, the differential diagnosis describes the underlying condition such as perirectal abscess, cellulitis, incarcerated inguinal hernia, prostatitis, orchitis, Bartholin's cyst abscess, acute hemorrhoidal thrombosis, anal fissure, perirectal abscess, or most any other pelvic/perineal process where necrotizing fasciitis has not been established.

Diagnosis

FG is primarily a clinical diagnosis and should be considered in any rapidly progressing process in the perineum. Diagnosis of FG is assured by establishing the presence of necrotizing fasciitis in the perineum with or without establishing the underlying cause. Crepitus on palpation is suggestive of subcutaneous soft tissue gas, especially in the presence of skin changes suggesting infection. Definitive diagnosis is confirmed by radiographic findings of subcutaneous gas in fascial

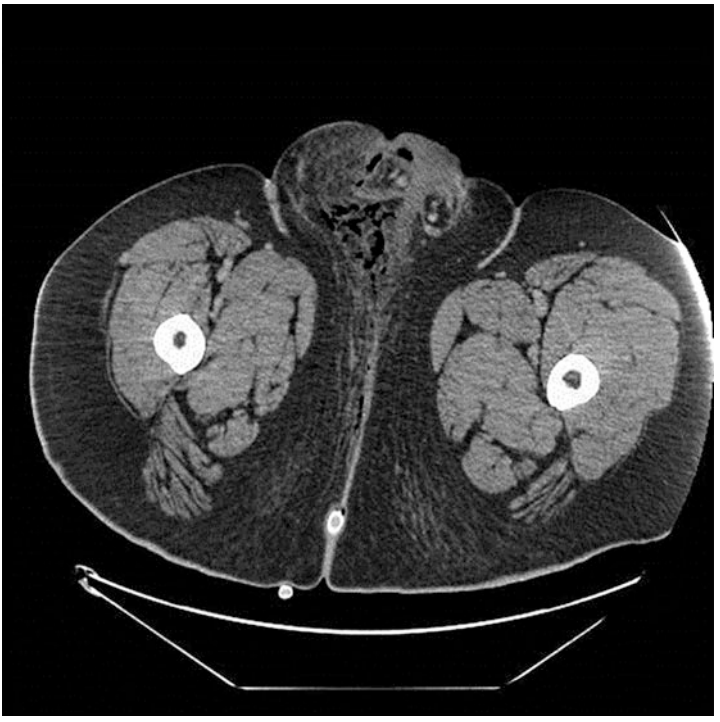

Fig. 7.1 CT scan showing gas gangrene in the scrotum. This is consistent with Fournier's gangrene. © Dale Dangleben, MD

planes. Radiographic studies may include plain X-ray, ultrasound, CT scan, and MRI. Hemodynamic collapse indicates systemic sepsis supports advanced progression of the disease (Fig. 7.1).

Complications

The most feared complication is death with failure to recognize the condition or failure to adequately treat with appropriate resuscitation, surgery, and systemic broad-spectrum antibiotics. Other complications include multiple organ failures, soft tissue defects, impotence, emotional overlay, decreased quality of life, and consequences of surgery to surrounding anatomy (sphincter dysfunction, orchidectomy, and penile excision).

Management

The mainstay of treatment of FG is that it is a rapidly progressive disease with a high mortality. Early diagnosis, broad-spectrum antibiotics to cover Gram-positive, Gram-negative, and anaerobic organisms, with antifungal therapy where indicated, coupled with surgical debridement and control of the necrotizing infection are mandatory in treating FG. Where the disease has progressed to become systemic such as in severe sepsis, surgery should be preceded by resuscitation and hemodynamic support. The disease advances rapidly with the rate of progression reported as 1–2 cm per hour, and therefore, surgery should not be otherwise delayed.

Operative management includes debridement and drainage of all necrotic skin and subcutaneous tissue. The extent of resection should be limited to the point where the necrotic skin and necrotic subcutaneous fat can be easily elevated from underlying fascia and tissue. Patients should return to the OR frequently for reevaluation and further debridement as indicated. Between surgeries, the wound can be covered with Dakin's solution soaked gauze to help topical control of infection. The perineal skin and organs are comprised of different blood supplies; the scrotal blood supply is from the pudendal branch of the femoral artery and that of the testicles and spermatic cords are from the testicular branch of the aorta. Therefore, the testicles should not be removed unless non-viability develops on the affected side (reported in 21% of patients). Only affected penile skin should be excised. Tissue cultures should be obtained at the time of the first operative debridement from which to focus antibiotic coverage (Figs. 7.2 and 7.3).

Hyperbaric oxygen therapy remains controversial with different reports providing conflicting conclusions. Hyperbaric oxygen therapy is most often used where it is available and excluded where it is not. Short of specific contraindications to hyperbaric oxygen therapy, its use does not appear to worsen FG.

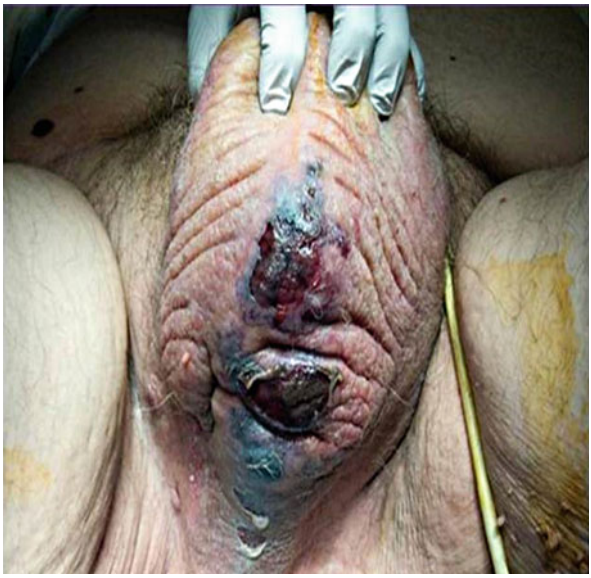

Fig. 7.2 Morbidly obese patient presenting with extensive soft tissue infection of his scrotum and perineum. © Dale Dangleben, MD

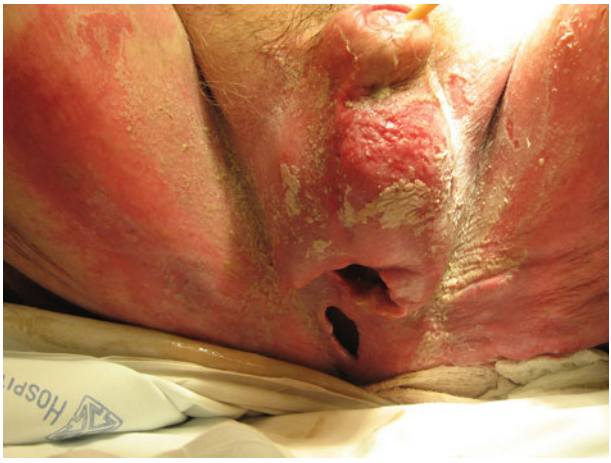

Fig. 7.3 This patient presents with severe Fournier's gangrene with extensive tissue loss and purulence. © Dale Dangleben, MD

References

1. Anaya DA, Dellinger EP. Necrotizing soft-tissue infection: diagnosis and management. Clin Infect Dis. 2007;44:705–10.
2. May AK, Stafford RE, Bulger EM, et al. Surgical infection society guidelines for the treatment of skin and soft tissue infections. Surg Infect. 2009;10(5):467–99.

Upper Gastrointestinal Bleeding

8

Firas G. Madbak

Bleeding patients belong on a surgical service

Learning Objectives

1. The learner will be able to formulate a differential diagnosis of upper gastrointestinal bleeding.
2. The learner will understand the spectrum and manifestations of upper gastrointestinal bleeding.
3. The learner will be able to describe the medical and surgical management of various causes of upper gastrointestinal bleeding.
4. The learner will be able to recognize, diagnose and treat potential complications of endoscopic modalities necessary for diagnostic workup as well as those associated with operative management.

Case Scenario

A 45-year-old female presents to the emergency department with abdominal pain and hematemesis. Her past medical history is remarkable for osteoarthritis for which she is on chronic NSAID therapy. Initial vital signs are Peripheral access is established and she is resuscitated with crystalloid and transfused with blood products. An NGT is placed and urgent upper endoscopy demonstrates a stomach full of blood and clots. Intravenous PPI infusion therapy was started. Due to

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continuing ongoing bleeding, a repeat EGD is performed and shows a bleeding gastric ulcer. Attempts at endoscopic bleeding control are unsuccessful. The patient is taken to the operating room where she undergoes laparotomy with excision and truncal vagotomy.

Epidemiology/Etiology/Pathophysiology

The incidence of acute upper gastrointestinal hemorrhage ranges from 40 to 150 cases per 100,000 per year in Western population, accounting for 200,000–300,000 hospital admissions annually. Overall mortality remains at 5–15%, which is largely unchanged over the past several decades. Despite a lack of mortality reduction, advancements in endoscopic and particularly interventional radiographic techniques have led to reduction of rebleeding rates and need for operative intervention.

Etiology can be categorized into variceal and non-variceal bleeding. Non-variceal causes include peptic ulcer disease, Mallory–Weiss lesions, tumors, reflux esophagitis, Dieulafoy's lesion, infectious cases, vascular malformations and drug-induced enteropathy. On the other hand, patients with variceal bleeding tend to have a higher mortality and should be managed in the intensive care unit.

Differential Diagnosis

A common surgical dictum states that the most common cause of lower GI bleeding (see lower GI bleeding module) is upper GI bleeding. Other etiologies that may cause chronic recurrent upper GI bleeding can be divided anatomically. Esophagitis, Barrett pathology as well as esophageal malignancy occasionally cause bleeding. In the stomach, malignant and benign tumors (e.g., gastrointestinal stromal tumor) as well as gastrinoma (ZE syndrome) can be innocuous bleeding that warrants aggressive workup in order to implement prompt treatment.

Diagnosis

In the elective setting, a complete history and physical examination eliciting tobacco and alcohol consumption, NSAID use, stigmata of liver disease as well as previous operations (e.g., AAA repair) are essential. Patients often present with hemodynamic instability, and resuscitation may have to be concurrent with a brief history.

Endoscopic diagnosis is the keystone of effective care because about 90% of bleeding sites can be identified with this modality. Chronic peptic ulcer disease, the most common etiology, is interpreted endoscopically by the mucosal defect appearance with a white fibrinous base or a deformity in the gastric contour.

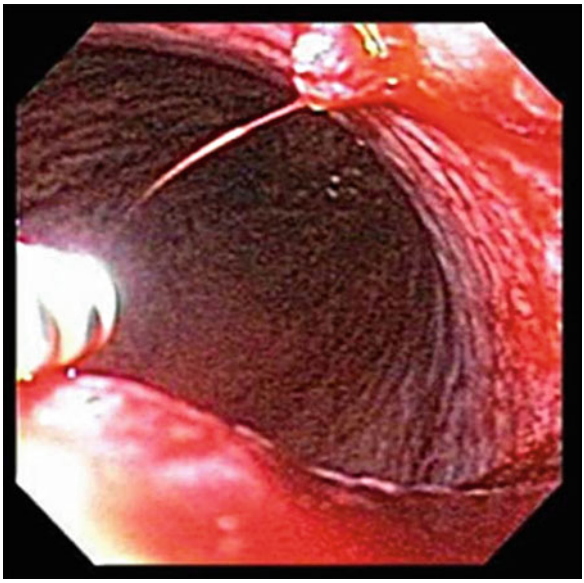

Fig. 8.1 UGI bleeding from a Dielaufoy's lesion. © Dale Dangleben, MD

Mallory–Weiss lesions are tears that occur at or near the gastroesophageal junction secondary to mechanical stress, classically induced by retching or vomiting although prior vomiting is not universal. They are usually self-limited, and they account for 4–14% of findings in EGD done for acute UGI bleeding. Dieulafoy's lesions are abnormal submucosal arteries that can cause recurrent bleeding episodes with an incidence of 0.5–14% Fig. 8.1. Most commonly in the proximal stomach, they can actually occur anywhere in the GI tract.

Complications

Estimated to occur with a frequency of 0.03%, esophageal perforation is the most troublesome complication of endoscopy. This iatrogenic injury usually occurs in the cervical esophagus (within Killian's triangle). Hemorrhage is the leading cause of death associated with peptic ulcer disease. Mortality rates for emergent operations for duodenal ulcer tend to be lower than the gastric counterpart since patients are younger and have fewer comorbid conditions. Obstruction and perforation are the other two common complications.

Management

Resuscitation with isotonic fluid is of the first priority in the acutely bleeding patient. In the setting of hemorrhagic shock, definitive airway management may be necessary secondary to diminished mental status and potential aspiration from ongoing emesis. Reversal of possible existing coagulopathy must follow. Nasogastric tube decompression should be performed. Lavage may prevent further bleeding; however, there is no clear evidence supporting the use of room temperature versus iced fluid. Laboratory studies including a type and cross and coagulation parameters along with a serologic test for *Helicobacter pylori* should be ordered. Continuous monitoring in the intensive care unit is usually required. Early endoscopy continues to be controversial, but undoubtedly helps determine the underlying cause. Intubation before endoscopy may be needed. The therapeutic options during EGD depend on findings. Various methods are available including injection with dilute epinephrine, heater probe or argon plasma coagulation as well as sclerosis and clip application Fig. 8.2 [1].

For variceal bleeding, intravenous infusion of vasopressin (0.4–0.6 units/min) and octreotide (50 μg IV bolus, then 25–50 μg/h IV) is utilized. Endoscopic band ligation and sclerotherapy are options for therapy with repeated endoscopic evaluations 1–2 weeks until varices are obliterated. Non-selective beta blockers are

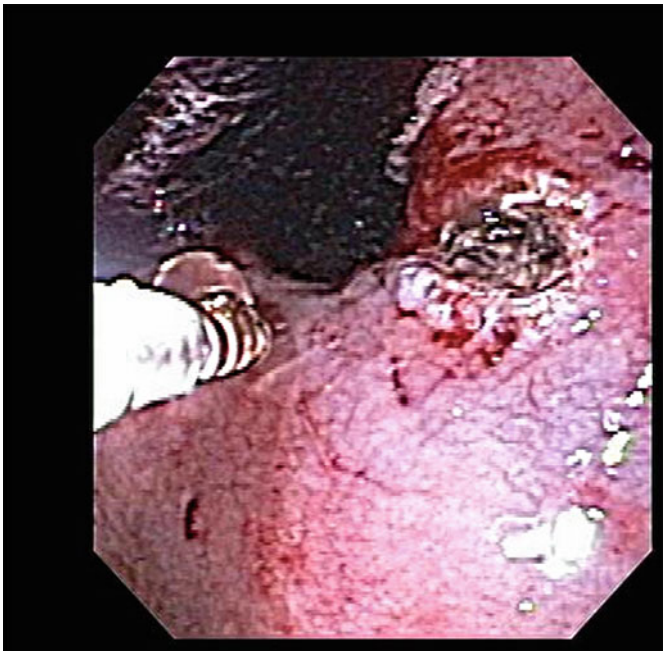

Fig. 8.2 Cauterizing a bleeding ulcer. © Dale Dangleben, MD

recommended in patients with portal hypertension to prevent recurrence, but have no role in the acutely bleeding patient [2].

Radiologic techniques such as arteriography and scintigraphy can be useful for diagnostic and therapeutic interventions. Identification of contrast extravasation localizes bleeding and can direct surgical management. Bleeding vessels may be embolized with coils or hemostatic agents in select cases [3]. Traditionally, bleeding rates as low as 1 cc/min can be detected with arteriography. Nuclear scintigraphy (bleeding scans) utilizing most commonly technetium-99m-labeled sulfur colloid or erythrocytes can be used to detect slower bleeding rates of 0.1 cc/min, and this can be advantageous; however, this modality is purely diagnostic. Intraluminal contrast studies (e.g., with barium) have no role and are not recommended.

Surgery is reserved for bleeding refractory to less invasive modalities, but can be life-saving when those alternatives fail. Bleeding gastric ulcer operative therapy depends on the ulcer type and hemodynamic stability. Type I gastric ulcers are located high on the lesser curve near the incisura. Type II ulcers are a combination of a gastric as well as duodenal ulcer. Type III are prepyloric ulcers. Type IV are juxtacardiac and located near the GE junction. Type V are those induced by NSAIDs or aspirin and independent of location in the stomach. Only Types II and III are associated with hypersecretory acid states. Stable patients can undergo the definitive antiulcer operation, which is a distal gastrectomy (antrectomy) and a reconstructive gastroduodenostomy (Billroth I, preferable because of restorative physiology but sometimes not possible secondary to duodenal scarring) or gastrojejunostomy (Billroth II, which can have complications of retained antrum, afferent loop syndrome or catastrophic duodenal stump blowout). If a Type II or Type III ulcer is encountered, then an acid-reducing truncal vagotomy procedure is added. Selective vagotomies are performed by some surgeons. Unstable patients are treated with oversewing or excision of the ulcer. Intraoperatively, biopsy of gastric ulcers to rule out malignancy is mandatory.

Acutely bleeding duodenal ulcers typically occur on the bulbar posterior wall as they erode into the gastroduodenal artery. A duodenotomy (or pyloroplasty) is used to gain access, and three-point ligation with silk sutures is used to control the gastroduodenal artery at 12 and 6 o'clock followed by a 3 o'clock stitch to control the transverse pancreatic branch medially. These can be mattress or figure-of-eight sutures. The duodenotomy is opened longitudinally and closed transversely. Some surgeons add a vagotomy depending on how stable the patient is in the operating room unless *H. pylori* status is ascertained preoperatively.

Mallory–Weiss tears usually occur near the GE junction and diagnosed by endoscopy. A midline laparotomy is used for surgical control. A longitudinal gastrostomy (near but not through the GE junction) is made, and direct suture control with absorbable monofilament is used. A locking stitch can be used. The gastrostomy can be closed with two layers. Postoperative nasogastric suction is not necessary.

Malignant lesions causing bleeding require tissue diagnosis and appropriate treatment according to oncologic principles in self-limited cases and may require operative hemorrhage control in acute situations.

References

1. Bertleff MJ, Halm JA, Bemelman WA, et al. Randomized clinical trial of laparoscopic versus open repair of the perforated peptic ulcer: the LAMA trial. World J Surg. 2009;33(7):1368–73.
2. Dell'Era A, Cubero Sotela J, Fabris FM, et al. Primary prophylaxis of variceal bleeding in cirrhotic patients: a cohort study. Dig Liver Dis. 2008;40(12):936–43.
3. Poultsides GA, Kim CJ, Vignati PV, et al. Angiographic embolization for gastroduodenal hemorrhage: safety, efficacy, and predictors of outcome. Arch Surg. 2008;143(5):457–61.

Lower Gastrointestinal Bleeding

9

Nicholas J. Madden

Location, location, location

Learning Objectives

1. The learner will be able to formulate an appropriate differential diagnosis for the causes of lower gastrointestinal (GI) bleeding based upon patient characteristics, risk factors, and examination findings.
2. The learner will understand the various presentations and degrees of severity for lower GI bleeds.
3. The learner will be able to develop an understanding for the diagnosis and management, both endoscopic and surgical, of lower GI bleeds.
4. The learner will recognize, diagnose, and treat potential complications of lower GI bleeds and the associated endoscopic and surgical treatments.

Case Scenario

A 75-year-old male presents to the emergency department with a chief complaint of bright red blood per rectum. He denies past medical and surgical history. The patient is noted to be tachycardic and hypotensive. He is admitted and resuscitated with crystalloids and transfused as needed. The patient undergoes a CT angiogram

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which identifies an area of bleeding in the sigmoid colon. Endoscopic attempts to control the bleeding were unsuccessful but revealed active bleeding secondary to diverticular disease in the sigmoid. Given his ongoing bleeding, he was taken to the operating room for laparotomy and sigmoid resection.

Epidemiology/Etiology

Acute gastrointestinal (GI) bleeding is categorized as upper or lower depending on the location in relation to the ligament of Treitz. Lower GI sources (predominantly the colon) account for 20% of all cases of acute GI bleeds. The estimated incidence is 20 cases per 100,000 individuals. The estimated mortality is 5 cases per 100,000 episodes.

Colonic diverticulosis is the leading cause of lower GI bleeds followed by colitis, neoplasms, and angiodysplasia (Figs. 9.1, 9.2, 9.3 and 9.4). Iatrogenic causes and benign rectal diseases should also be considered although they are less common. The small intestine distal to the ligament of Treitz represents the source for less than 5% of lower GI bleeds.

Certain patient characteristics are associated with an increased risk; advanced age, male gender, use of anticoagulant or antiplatelet agents, and underlying cardiac and renal disease are among the most significant risk factors.

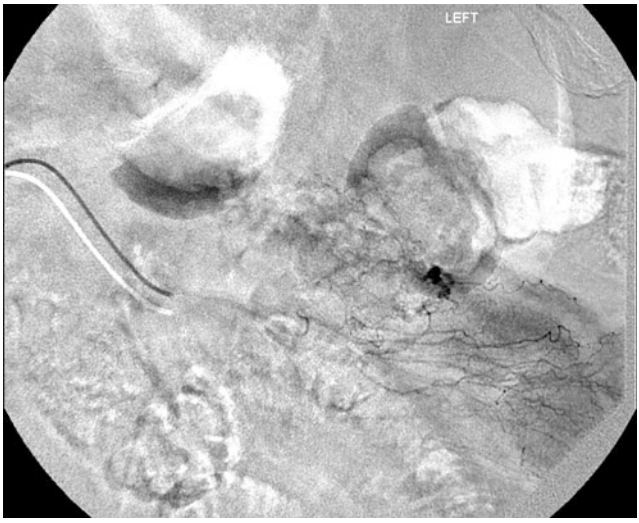

Fig. 9.1 Small bowel AVM seen on mesenteric angiography. © Dale Dangleben, MD

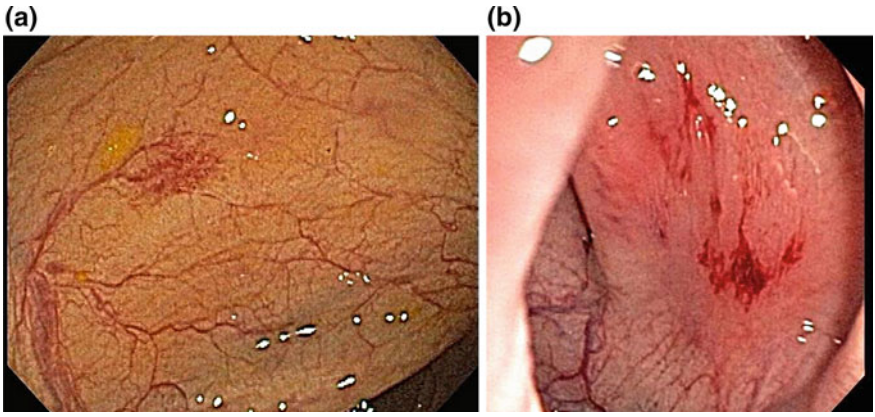

Fig. 9.2 **a**, **b** Endoscopic view of AV malformation. © Dale Dangleben, MD

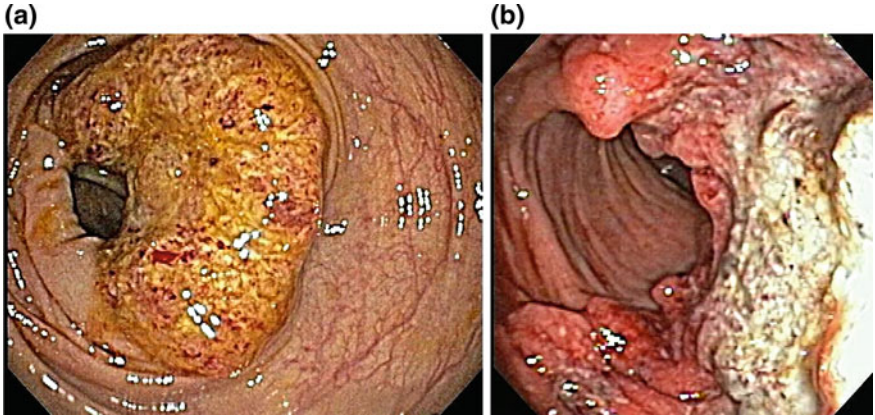

Fig. 9.3 **a**, **b** Colon adenocarcinoma. © Dale Dangleben, MD

Differential Diagnosis

When attempting to diagnose a lower GI bleed, it is always essential to rule out an upper GI source as the cause. Assessment of patient risk factors and nasogastric tube lavage can assist with this differential (see upper GI chapter). Etiologies of true lower GI bleeds can be difficult to differentiate base on history and physical examination alone. One must always consider whether the process is benign, infectious, or potentially malignant.

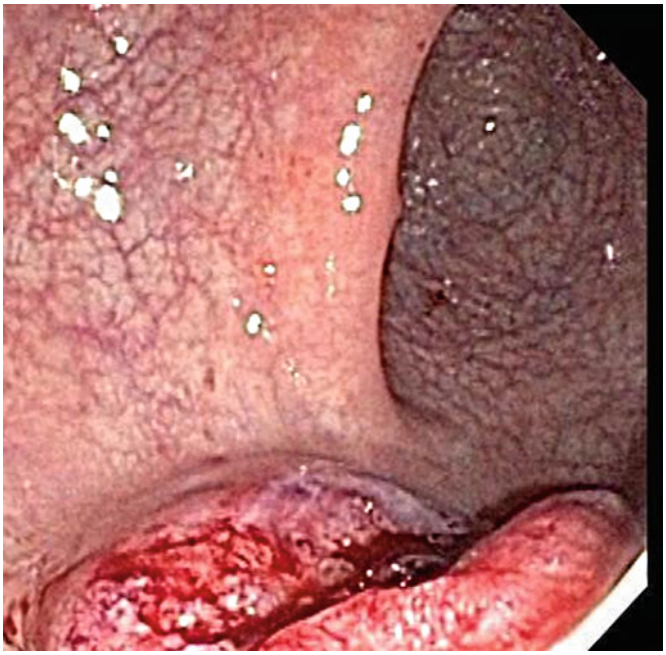

Fig. 9.4 Bleeding rectal carcinoma. © Dale Dangleben, MD

Diagnosis

As always, a comprehensive history and physical examination should be conducted if possible. In particular, attention to history of GI bleeds, diverticular disease, and inflammatory or infectious bowel disease may provide some insight. Additionally, endoscopic and abdominal surgical history should be obtained. On examination, attention should be given to the overall appearance of the patient as well as the abdominal exam. Presence of bright red blood on digital rectal exam is highly suspicious for a lower GI source unless there is a brisk upper GI source. Rectal exam also may help rule out an anorectal etiology. Nasogastric tube lavage can aid in identifying an upper GI source.

Colonoscopy remains not only invaluable for diagnosis but also has a potentially therapeutic benefit as well (Fig. 9.5). CT scan angiography, traditional angiography, and abdominal scintigraphy with radiolabeled red blood cells can also be utilized in the appropriate clinical settings if available although the latter is sensitive at lower bleeding rates but can frequently make precise localization challenging.

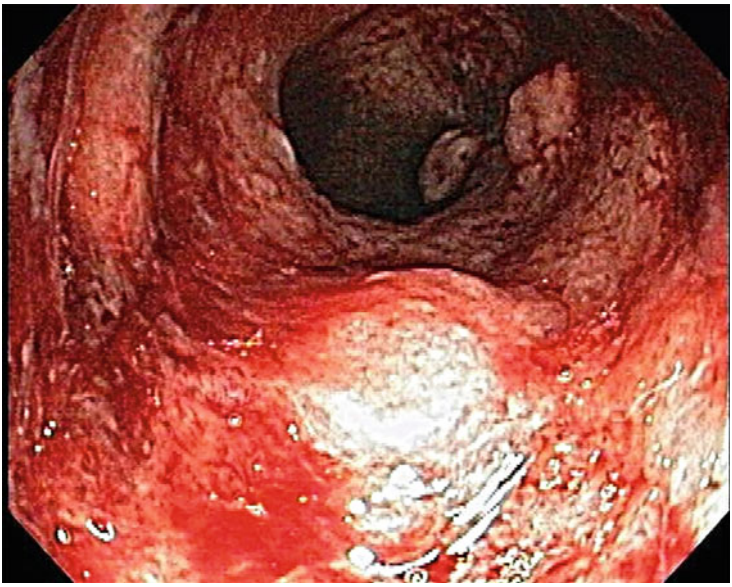

Fig. 9.5 Ulcerative Colitis. © Dale Dangleben, MD

Complications

Complications of nonoperative management and supportive care can include ongoing bleeding, re-bleeding, and hemodynamic instability. Escalation of care and endoscopic interventions are warranted in these cases.

All procedures, whether endoscopic or surgical, are associated with known risks. Colonoscopy can rarely cause significant bleeding, particularly if biopsies are taken. Perforation is another known complication associated with endoscopy that may require surgical intervention for control of bleeding and contamination.

Surgical intervention comes with the known risks of bleeding, infection and injury to surrounding structures. The perioperative morbidity and mortality are also known to be high in patients undergoing total abdominal colectomy compared to those undergoing a segmental resection. Additional complications associated with colon resections include anastomotic leaks, intra-abdominal abscesses, and complications related to ostomy creation.

Management

Initial efforts should be focused on resuscitation and stabilization as needed. Definitive airway control may be necessary in cases of hemorrhagic shock in which mental status is compromised. Isotonic fluids can be used as a first-line agent to

restore plasma volume with early packed red blood cell transfusion in the case of more significant bleeds. Laboratory studies including a complete blood count, metabolic panel, coagulation profile and type and screen/cross should be obtained and trended if ongoing bleeding is suspected. Unstable patients may require close monitoring in the intensive care unit. Although the majority of GI bleeds will ultimately cease with supportive care and noninvasive measures, some will have ongoing bleeding or re-bleeding which will mandate intervention.

Colonoscopy is a crucial initial procedure that can be performed initially to not only identify a source but also obtain hemostasis in patients with ongoing bleeding. It is especially useful for bleeds secondary to diverticular disease and angiodysplasia. Endoscopic cautery, sclerotherapy, and injection of vasoconstrictors are some of the more commonly performed techniques. Visceral angiography with selective embolization utilizing coils or hemostatic agents in areas of active contrast extravasation can also be used in those with an ongoing bleed [1–3].

Advanced endoscopic and radiographic techniques have become the mainstay of treatment. When these fail, however, surgical intervention is warranted. Ideally, the source of the bleeding will be localized preoperatively with a scintigraphy or angiography to assist with surgical decision making. Both of these studies require that the patient is actively bleeding for them to be of utility. In cases where the location of the bleeding is known a partial colectomy can usually be performed. If identifying the source is not feasible, a subtotal abdominal colectomy with end ileostomy may be necessary for definitive control. Surgical resection is also warranted for bleeding neoplasms not amendable to endoscopic therapy [4].

References

1. Farner R, Lichliter W, Kuhn J. Total colectomy versus limited colonic resection for acute lower gastrointestinal bleeding. Am J Surg. 1999;178:587–90.
2. Lewis M. Bleeding colonic diverticula. J Clin Gastroenterol. 2008;42:1156–8.
3. Miller M, Smith TP. Angiographic diagnosis and endovascular management of nonvariceal gastrointestinal hemorrhage. Gastroenterol Clin N Am. 2005;34:735–52.
4. Song LMWK, Baron TH. Endoscopic management of acute lower gastrointestinal bleeding. Am J Gastroenterol. 2008;103:1881–7.

Acute Anorectal Emergencies

10

Lindsey Perea and Dale A. Dangleben

Acute perianal pain is one of three things; a fissure, abscess or thrombosed hemorrhoid

Learning Objectives

1. The learner should be able to formulate a differential diagnosis for anorectal pain.
2. The learner should know the classifications of perirectal abscesses.
3. The learner will differentiate between internal and external hemorrhoids and know the grades of internal hemorrhoid prolapse.
4. The learner will be able to recognize some of the more common causes of anorectal pain.

Case Scenario

A 37-year-old man presents to the emergency department complaining of anorectal pain and pressure that began 4 days ago. Today, he noted some drainage, prompting his visit to the emergency room. Upon rectal examination, you note

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external hemorrhoids and an area of erythema, induration, and fluctuance. You diagnose him with a left perianal abscess and note purulent discharge. His past history is significant for a sphincterotomy three weeks ago by a colorectal surgeon for an anal fissure.

The abscess is drained in the emergency room, and the patient is discharged with pain medication and a follow-up appointment with his colorectal surgeon.

Epidemiology/Etiology/Pathophysiology

Perirectal Abscesses

Perirectal abscesses most commonly originate secondary to a cryptoglandular infection. Some other causes include Crohn's disease, radiation, malignancy, anal fissures, HIV, and trauma. They can also arise as a complication of anal operations such as hemorrhoidectomy and lateral internal sphincterotomy although that is exceedingly rare. Overall, abscesses seem to occur more commonly in men than in women.

Description of anorectal abscesses is based on location and is thus classified into perianal, intersphincteric, ischiorectal, supralevator, and submucosal. Perianal abscesses are superficial, tender masses found outside the anal verge. They constitute 60% of all anorectal abscesses. Ischiorectal abscesses present as large, erythematous, indurated, and tender masses of the buttock. However, they may not be apparent on examination and the patient's only presenting symptom may be pain. This type of abscess is seen in 20% of patients. Intersphincteric abscesses occur in the intersphincteric plane but not under the mucosa and account for 5% of abscesses. The infection of the crypt in the anal canal extends cephalad and presents as a mass within the lower part of the rectum, often after manifesting fever and pain. Supralevator abscesses are rare and constitute less than 4% of abscesses. These patients often describe vague pain due to the lack of outward manifestations. These typically result secondary to upward spread in the intersphincteric space. These patients often are febrile and have an elevated WBC count. They also often have an underlying inflammatory process: recent surgery, Crohn's disease, or a pelvic inflammatory process. Submucosal abscesses are extremely rare accounting for only 1% of all perirectal abscesses.

Hemorrhoids

Hemorrhoids are cushions of vascular submucosal tissue containing venules, arterioles, and smooth muscle fibers that are located in the anal canal. There are three cushions: left lateral, right anterior, and right posterior. They are part of the normal anorectal anatomy and are thought to aid in complete closure of the anal canal contributing to continence. They are classified into external and internal

hemorrhoids. External hemorrhoids are located distal to the dentate line. They can cause itching and difficulty with hygiene if they are large.

Internal hemorrhoids are proximal to the dentate line and may cause bleeding or prolapse. Since they are covered by insensate mucosa they only become painful in the event of thrombosis or necrosis. Internal hemorrhoid grading consists of four grades. First-degree internal hemorrhoids present with bleeding but no prolapse. Second-degree internal hemorrhoids have prolapsed but with spontaneous reduction. Third-degree internal hemorrhoids are prolapsed and require manual reduction. Fourth-degree internal hemorrhoids are prolapsed and they are not reducible.

Anal Fissures

Anal fissures are linear ulcers in the lower half of the anal canal, typically distal to the dentate line. Women are more commonly affected than men, with 40% of females with fissures occurring anteriorly. Overall, most fissures are located posteriorly in the midline. Associated findings include a sentinel pile or tag located externally or an enlarged anal papilla found internally. Fissures located laterally should raise concern for other conditions such as Crohn's disease, HIV and carcinoma.

Fissures cause pain because they involve highly sensitive squamous epithelium, and when the ulcers stretch, they can cause pain and bleeding. Anal resting pressures have been noted to be higher with decreased blood flow in the posterior midline therefore resulting in anal sphincter hypertonia and mucosal ischemia. Some of the factors that seem to contribute to the development of anal fissures include large hard stools, inappropriate diet, previous anal surgery, childbirth, and laxative abuse [1].

Differential Diagnosis

Rectal pain can be a result of perirectal abscesses, anal fissures, fistulas, thrombosed hemorrhoids, prolapsed incarcerated internal hemorrhoids, colonic or anal malignancy, constipation, fecal impaction, diarrhea, Crohn's disease, ulcerative colitis, proctitis, pruritus ani, levator ani syndrome, trauma, and foreign bodies.

Diagnosis

Perirectal Abscesses

Pain is the most common presenting symptom of perirectal abscesses. Pain is aggravated with activity or straining. Diagnoses of perirectal abscesses can be

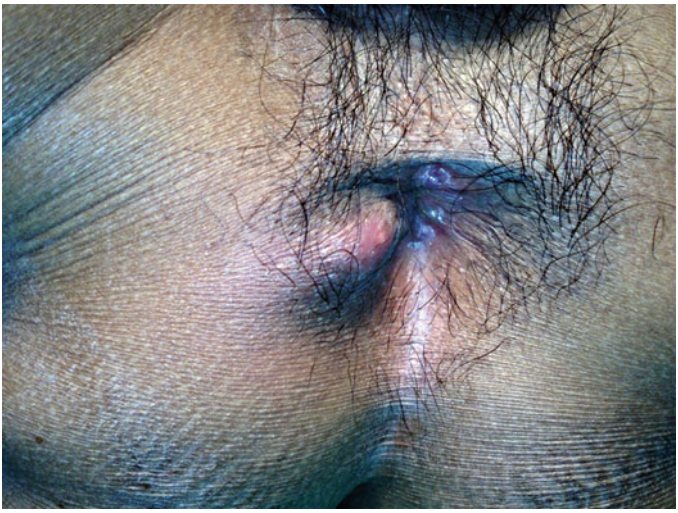

Fig. 10.1 Perianal abscess. © Dale Dangleben, MD

accomplished by inspection and a digital rectal examination (Fig. 10.1). A palpable mass is often felt, but occasionally patients may present with fever, urinary retention, and sepsis. If the patient is too uncomfortable, a rectal examination under anesthesia can be very helpful. Intra-anal ultrasounds have been a subject of interest and can aid in the evaluation of anorectal abscesses and fistulas. A limitation of ultrasonography is the inability to fully image the supralevator and ischiorectal spaces. For the more complex and atypical presentations, CT or MRI can help clarify the anatomy of the abscess.

Hemorrhoids

Hemorrhoids can be diagnosed by a thorough history and physical examination. The examination should include inspection while straining. In all patients who present with rectal bleeding, an anoscopy and proctosigmoidoscopy should be part of the initial evaluation (Fig. 10.2). A colonoscopy or a barium enema must be performed if the source of bleeding is not identified and in order to rule out malignancy (Fig. 10.3).

Anal Fissures

The diagnosis of an anal fissure can be obtained by history and physical examination. A typical history includes pain and bleeding with defecation in the setting of prior constipation. The diagnosis is confirmed by gently parting the buttocks and

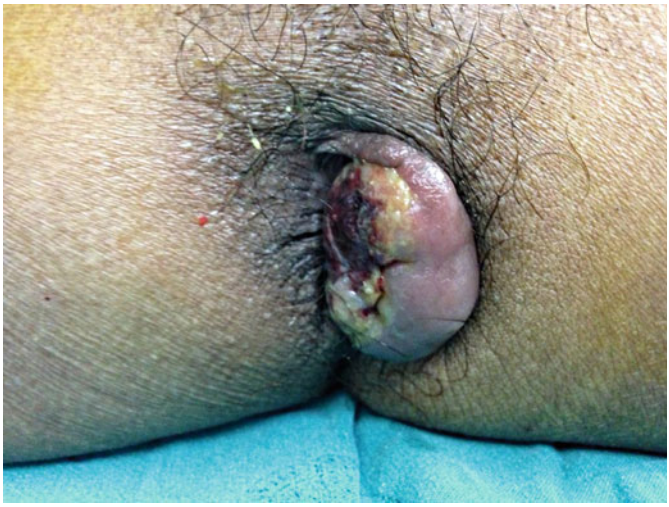

Fig. 10.2 Thrombosed hemorrhoid with necrotic area. © Dale Dangleben, MD

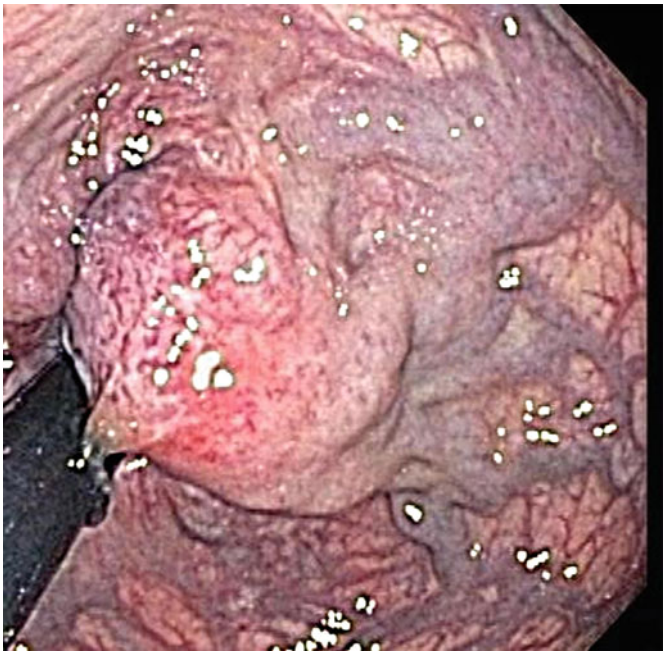

Fig. 10.3 Endoscopic view (retroflex) of internal hemorrhoid. © Dale Dangleben, MD

inspection. More invasive examination such as digital examination should be deferred as it is often not tolerated in acute cases. Endoscopic examination should be performed, but this can be delayed 4–6 weeks until the pain is resolved.

Complications

Perirectal Abscesses

The majority of complications that arise from perirectal abscesses result secondary to improper, or incomplete, drainage. One of the most common complications of perirectal abscesses is anal fistula which occurs in about 50% of drained anorectal abscesses. In cases where circumferential spread occurs, a horseshoe abscess may result.

Improperly treated perirectal abscesses can lead to severe sepsis, which may prove to be life threatening in the immunocompromised. Necrotizing soft tissue infection of the perineum is a rare but lethal condition. Recurrence is also common in cases of inadequate drainage and fistulas.

Hemorrhoids

External hemorrhoids can cause significant pain and discomfort. If thrombosed, they can cause severe pain. Internal hemorrhoids cause painless bright red bleeding. One of the complications of internal hemorrhoids is prolapse. Prolapse below the dentate line can cause fecal leakage and pruritus. Strangulated prolapsed internal hemorrhoids can cause significant distress to patients.

Anal Fissures

A complication related to anal fissures is failure to heal. Patients with previous fissures are more prone to recurrent fissures in the future. Incontinence is a major complication that may result following sphincterotomy for anal fissures.

Management

Perirectal Abscesses

Anorectal abscesses should be drained as soon as the diagnosis is established. Perianal abscesses typically drain well after a simple skin incision. Intersphincteric abscesses are drained into the anal canal via division of the internal sphincter.

Drainage of supralevator abscesses is determined based on the etiology of the spread. For those resulting from an initial ischiorectal abscess, the drainage occurs into the lower rectum or upper anal canal. In cases where the abdomen is the course, the abscesses should be drained transabdominally.

Ischiorectal abscesses should be drained through the skin after localization. Circumferential spread resulting in a horseshoe abscess requires the modified Hanley procedure that drains both the lateral aspects of the abscess as well as the deep post-anal space. The external sphincter is incised in the posterior midline which allows access to the post-anal space; the lateral incisions allow for added drainage routes [2].

If the diagnosis is uncertain, a rectal examination under anesthesia can be both diagnostic and therapeutic. Antibiotics should be considered if the patient is immunocompromised, diabetic, has a history of a systemic illness, and in patients with valvular heart disease. Antibiotics alone are not adequate for the treatment of perirectal abscesses. It is important to distinguish between the different types of abscesses in order to achieve the best outcomes after drainage.

Hemorrhoids

Management of hemorrhoids can be broken down into nonoperative and operative management. Nonoperative management includes improved hygiene, avoidance of excessive straining, increased fluid intake, and a high-fiber diet to keep stools soft, formed, and regular. First-, second-, and some third-degree internal hemorrhoids can be treated in the office with procedures such as sclerotherapy, infrared coagulation, heater probe, bipolar electrocoagulation, and rubber band ligation. Third- and fourth-degree internal hemorrhoids often require operative management. Those with failed conservative management should also consider operative management. The surgical options consist of open and closed hemorrhoidectomies as well as hemorrhoidopexy. Hemorrhoidectomy can be performed via the closed technique where both internal and external hemorrhoids are excised and then the site sutured closed. The open approach is similar but does not close the excised area. Additional methods include use of electrical or ultrasonic energy which have shown to have decreased postoperative pain. Hemorrhoidopexy is an option in which a circular stapler excises all redundant hemorrhoidal tissue. Possible complications may result from aggressive tissue resection resulting in rectovaginal fistulas, or unrelenting pain.

Patients with thrombosed external hemorrhoids should undergo excision typically during the first 3 days when the pain is the greatest. During this time, incision of the thrombosed hemorrhoid is not favored given the risk of re-thrombosis. Patients in the subacute phase (>72 h) are often best treated with supportive care.

Anal Fissures

The management of anal fissures can be medical or surgical. The first-line therapy for acute fissures consists of sitz baths and bulking agents. Medical treatment includes topical nitroglycerin, calcium channel blockers, and at times botulinum injections. Patients with chronic fissures who have failed medical management may benefit from surgery. Lateral internal sphincterotomy can be performed either via the open or closed technique. The open technique involves incising the anus, elevating the internal sphincter from the external sphincter dividing it. The closed technique involves placing a fine scalpel in the intersphincteric groove while the surgeon's index finger is in the anus. The blade is then advanced toward the anal canal incising the internal sphincter but not the mucosa. It should be noted that overall incontinence rates may be as high as 14% after the procedures.

References

1. Keighley MRB. Anorectal Disorders. In: Fischer JE, editor. Fischer's mastery of surgery. 6th ed. Philadelphia, PA: Lipincott Williams & Wilkins; 2012.
2. Merchea A, Larson DW. Anus. In: Townsend C, editor. Sabiston textbook of surgery. 20th ed. Philadelphia, PA: Elsevier; 2017. p. 1394–417.

Incarcerated Groin Hernias

11

Busayo Irojah

An obstructed patient should prompt a careful physical examination to rule out this diagnosis

Learning Objectives

1. Review the presentation of incarcerated groin hernias.
2. Discuss the appropriate management of incarcerated groin hernias.
3. Review the indications for operative intervention for acute groin hernias.

Case Scenario

A 57-year-old male presents to the Emergency Department with 4 h of sudden onset nausea, vomiting and obstipation. On examination, he has a hard tender lump in his right groin. He has a history of an asymptomatic right inguinal hernia. Abdominal plain film shows dilated loops of small bowel with air fluid levels. CT scan of the abdomen and pelvis shows a small bowel obstruction with the transition point in a right inguinal hernia.

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Epidemiology/Etiology/Pathophysiology

Groin hernias are one of the most common problems encountered by general surgeons, particularly inguinal and femoral hernias. Approximately 25% of men and 2% of women will develop a groin hernia.

Inguinal hernias are most common and may be direct or indirect. Direct hernias occur in Hesselbach's triangle which is the area bounded by the inferior epigastric laterally, the rectus medially and the inguinal ligament inferiorly. Indirect hernias occur lateral to the inferior epigastric vessels. Direct hernias are less likely to become strangulated than indirect hernia with a reported rate of strangulation 10% that of indirect hernias.

Femoral hernias, more common in women, occur in the femoral ring (more often on the right since the sigmoid colon occludes the femoral canal on the left) which is the area bounded by the femoral vein laterally, the inguinal ligament anteriorly and posteriorly by the iliopubic tract and iliopectineal ligament. Femoral hernias have a higher tendency to present with incarceration and strangulation and subsequently more patients with these hernias will require a bowel resection. On examination, femoral hernias are found inferior to the inguinal ligament just medial to the femoral pulse. Unlike inguinal hernias, they are sometimes difficult to elucidate on examination alone and may require further evaluation with imaging.

Patients with incarcerated groin hernias typically present with focal abdominal pain and an irreducible mass in the groin region. Signs of small bowel obstruction may also be present. In children, early signs include irritability and crying and precede emesis and distention. The most common incarcerated viscera from most to least common are small bowel, omentum and large bowel [1].

Differential Diagnosis

The differential includes groin abscess, inguinal lymphadenopathy or lymphadenitis, testicular torsion, neoplasm, hydrocele or varicocele.

Diagnosis

The importance of a detailed history as well as a careful and thorough physical examination of the bilateral groins cannot be overstated. Occasionally, imaging may be necessary in the morbidly obese patients where groin palpation may be difficult or equivocal (Fig. 11.1).

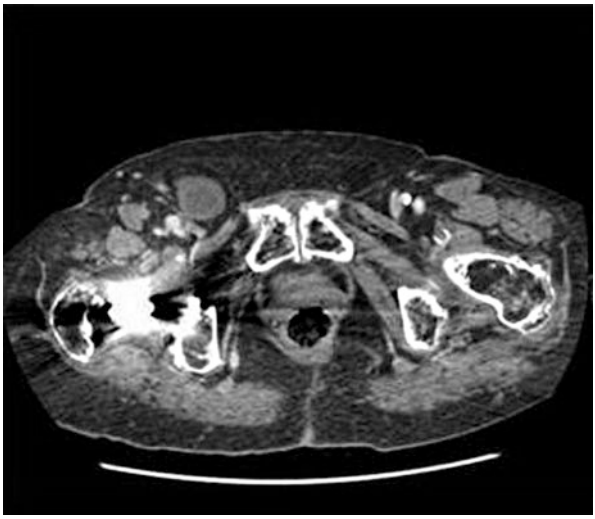

Fig. 11.1 CT scan showing a *left* groin hernia. © Dale Dangleben, MD

Complications

Delayed diagnosis and treatment of a painful, tender, inflamed hernia can lead to bowel ischemia and perforation necessitating emergency surgery and possible bowel resection.

Management

Hernia Reduction

Hernia reduction is contraindicated if there are signs of strangulation. Strangulated hernias may be tense and tender with a blue or red discoloration to the overlying skin. Unfortunately, it may not be immediately apparent on examination whether or not a hernia is incarcerated. Duration of acute incarceration has been used to determine when strangulation has occurred and reduction is no longer safe; however, there is no consistency to the time reported for strangulation to occur with reported time ranging from 4 to 96 h. Subsequently, the only true contraindication to reduction is peritonitis. However, when there is doubt about the presence of strangulation, urgent surgical intervention as opposed to reduction is a recommended.

During bedside reduction (called *taxus*), conscious sedation may sometimes be required especially for pediatric patients. The patient is placed in the Trendelenburg position, and on occasion, ice application for several minutes in adults may help reduce edema and facilitate manual reduction. The proximal neck of the hernia is

grasped with one hand and gentle pressure applied to the distal part of the hernia to guide it through the neck.

If the hernia is irreducible, emergent operative intervention is indicated. Those whose hernias are successfully reduced should undergo elective repair. In the not uncommon scenario of spontaneous reduction in a suspected strangulated hernia upon induction of anesthesia on the operating table, the surgeon should ensure bowel viability by exploring the abdominal cavity using laparotomy or preferentially using laparoscopy. One of the editors has explored the peritoneal cavity using a balloon trocar secured with a purse string from the preperitoneal space through the internal ring with good success. Compromised bowel is resected (Figs. 11.2 and 11.3).

Surgical Therapy

Incarcerated inguinal hernias may be approached with a standard groin incision. The literature has not answered the question of prophylactic preoperative antibiotics definitively for elective hernia repairs; however, they are recommended given the possibility of bowel resection. In contradistinction to the elective operation, the hernia sac should be opened to inspect its contents with care to avoid reduction in the peritoneal cavity before this step. Necrotic omentum should be resected with care to ensure hemostasis. In those cases where there is evidence of bowel ischemia or necrosis requiring resection, the use of prosthetic mesh for repair is traditionally discouraged given the higher risk of mesh infection. Cyanotic or questionable bowel is wrapped with moist gauze and observed and intervally inspected; then, a decision is made regarding the need for resection if ischemia is irretrievable. Incarcerated groin hernias consisting of viable bowel may be repaired in the usual preferred, tension-free fashion with mesh.

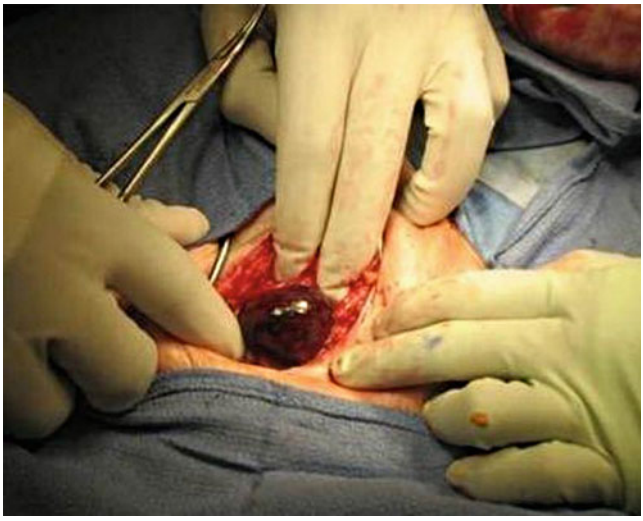

Fig. 11.2 "Knuckle" of dead bowel within the groin hernia. © Dale Dangleben, MD

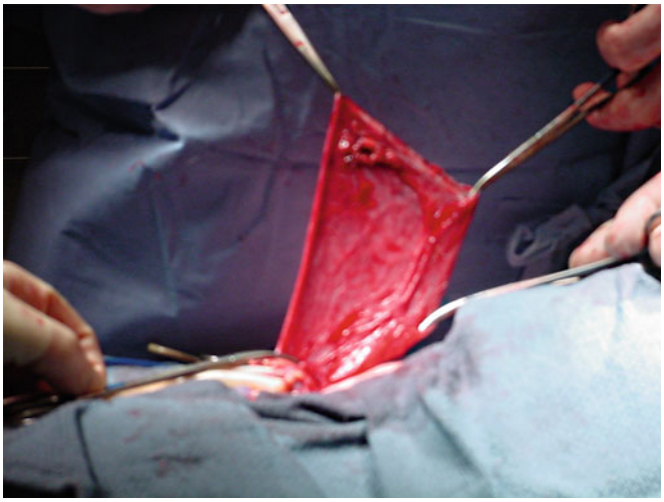

Fig. 11.3 Large inguinal hernia sac after complete dissection. © Dale Dangleben, MD

Occasionally, a chronically incarcerated hernia that causes distal bowel obstruction may need to be retrieved from the scrotum in its entirety and then carefully reduced using the same taxus maneuvers via the external ring. Rarely, this is not possible and extension of the skin incision superolaterally to enable splitting of the external oblique aponeurosis, internal oblique and transverse muscles above the internal ring is necessary. Though this incision, the peritoneal cavity is entered and the hernia contents are reduced by traction "from within."

As they are infrainguinal, it is preferable to approach an acutely incarcerated femoral hernia using a low incision over the palpated bulge. Alternatively, for incarcerated femoral hernias an infrainguinal incision, preperitoneal (McEvedy) or Lotheissen's transinguinal incision may be used. If reduction is difficult, the femoral canal's medial boundary formed by the lacunar ligament is incised. The similar principles governing bowel viability and resection apply and McVay-Cooper's tissue repairs may be necessary. Mesh repairs are typically achieved with a prosthetic polypropylene plug secured in the femoral canal [2, 3].

Laparoscopy is an acceptable approach though is reserved for elective repairs.

References

1. Anson B, Morgan E, McVay C. Surgical anatomy of the inguinal region based upon a study of 500 body-halves. Surg Gynecol Obstet. 1960;111:707–25.
2. Hair A, Paterson C, O'Dwyer PJ. Diagnosis of a femoral hernia in the elective Setting. J R Coll Surg Edinb. 2001;46:117–8.
3. Khatri VP. Femoral hernia repair (Chapter 14). In: Operative surgery manual, 1st ed. Philadelphia: Saunders Publishing Company; 2003.

Intussusception

12

Zachary Ewart and Lindsey Perea

In an adult, this diagnosis is due to a neoplasm until proven otherwise

Learning Objectives

1. The learner should be able to recognize the constellation of presenting symptoms of intussusception.
2. The learner should know the most likely causes in pediatric and adult populations.
3. The learner should be able to report the steps of diagnosis and management.

Case Scenario

A 64-year-old man presents to the emergency department complaining of intense, intermittent abdominal pain for the past 3 days; however, for the last 12 h, the pain has been progressive and constant. He complains of abdominal distension, nausea, and bilious emesis. A CT scan of his abdomen and pelvis is obtained, which shows small bowel dilation and a target sign at the transition point.

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Epidemiology/Etiology/Pathophysiology

Intussusception is defined as the telescoping of any proximal segment of the gastrointestinal tract within the lumen of an adjacent segment. Secondary to anatomy, this most commonly occurs when a free flowing segment of bowel is juxtaposed with a segment fixed by retroperitoneal attachments or adhesions. This allows the bowel to invaginate into the other during peristalsis and becomes stuck, leading to obstruction, venous stasis, edema, and pain. If intussusception fails to resolve spontaneously, this can progress to bowel ischemia, necrosis, and possible perforation.

This condition occurs due to either primary or secondary causes. In 8–20% of adult presentations, the cause is unknown or idiopathic. In the remainder of cases, the condition develops from a pathologic lesion that provides a lead point for the intussusception. These are defined as secondary causes.

Children most frequently present with the triad of symptoms including crampy abdominal pain, bloody stools, and a tender palpable mass in the abdomen. These findings are typically preceded by a viral prodrome and/or symptoms of gastroenteritis. This leads to lymphadenopathy and swelling of the Peyer's patches found in the distal ileum. Often, Peyer's patches serve as the lead point for intussusception.

In adult populations, they similarly present with the signs and symptoms of bowel obstruction coupled with intermittent crampy abdominal pain. As previously mentioned, the cause is more likely due to distinct pathologic lead points. In adults, the cause is found to be malignant in half of presenting cases [1–3].

Differential Diagnosis

The differential diagnosis of intussusception can be due to multiple causes including adhesions, incarcerated hernias, strictures, inflammatory bowel disease, volvulus, ileus, malignancy, gallstone ileus, bowel obstruction, and gastroenteritis.

Diagnosis

Patients often present with the symptoms of bowel obstruction with intermittent crampy abdominal pain. In pediatric populations, diagnosis is based on clinical suspicion and, in the absence of peritonitis, radiographic air enema which can be both diagnostic and curative. Additionally, ultrasound can demonstrate the characteristic "target sign" in children most commonly involving the ileocecal valve. In adult

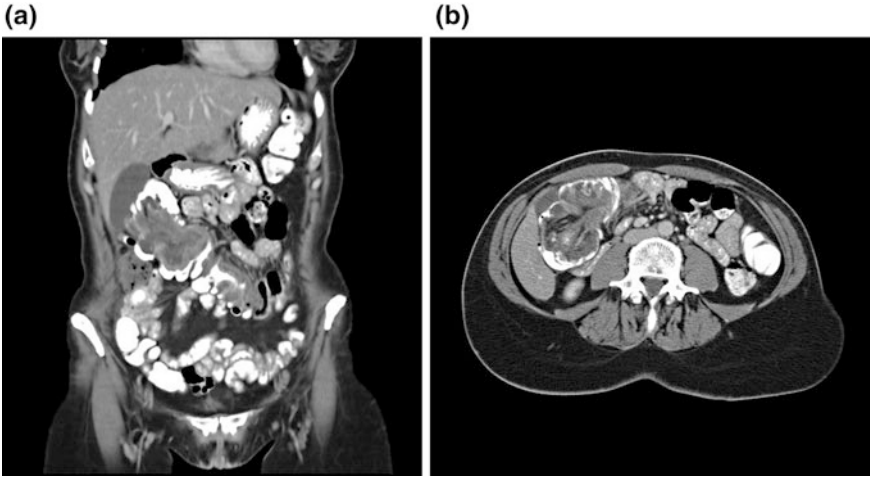

Fig. 12.1 **a, b** Intussusception on CT scan. © Dale Dangleben, MD

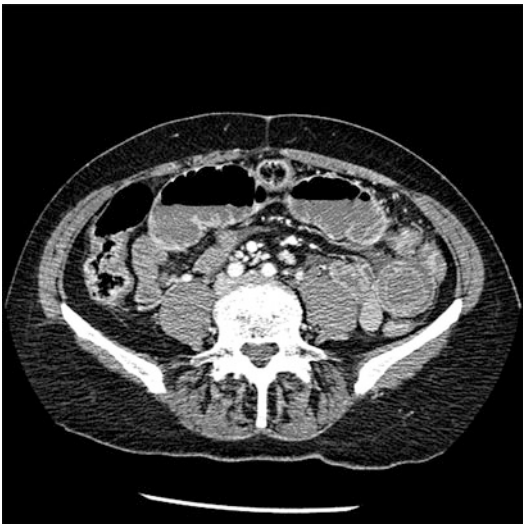

Fig. 12.2 Target sign sometimes seen with intussusception. © Dale Dangleben, MD

populations, CT imaging is the diagnostic modality of choice and will often show a "target sign," which is pathognomonic for intussusception (Figs. 12.1 and 12.2).

Complications

If the intussusception is not reduced in a timely fashion, the involved bowel can become ischemic leading to necrosis and perforation.

Management

The management strategy differs based on the presenting population. In pediatric patients, a radiographic air enema is typically performed. If the intussusception fails to reduce after three attempts, the likelihood of a pathologic lead point is high. Pneumatic decompression should not exceed 120 mm Hg. In cases where there is failure to reduce, the patient is taken to the operating room for exploration either laparoscopically or open based on surgeon familiarity. In adults, the management is operative with surgical resection of the involved segment including the pathologic lead point. The tissue is then sent for pathology to evaluate for underlying malignancy.

References

1. Ciftci F. Diagnosis and treatment of intestinal intussusception in adults: a rare experience for surgeons. Int J Clinic Exp Med. 2015;8(6):10001–5.
2. Del-Pozo G, Albillos J, Tejedor D, et al. Intussusception in children: current concepts in diagnosis and enema reduction. Radiographics. 1999;19(2).
3. Eisen L, Cunningham J, Aufses A. Intussusception in adults: institutional review. 1999; 188(4):390–5.

Meckel's Diverticulitis

13

Benjamin Palachik and Lindsey Perea

2% of the population, within 2 feet of the ileocecal valve, 2 inches in length, 2 types of heterotopic mucosa, and presentation before the age of 2

Learning Objectives

1. Understand the embryologic development of a Meckel's.
2. Review the "Rule of 2's."
3. Learn the ways a Meckel's diverticulum can present.
4. Know the imaging modality for a Meckel's presenting as GI hemorrhage.
5. Discuss surgical options when a Meckel's is encountered.

Case Scenario

A 59-year-old male with no previous surgical history presents to the Emergency Department with complaints of abdominal pain, nausea, vomiting, abdominal distension, and obstipation. He reports he has had similar episodes in the past which have resolved with non-operative management. He has also received a colonoscopy

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after the first episode with normal findings. Physical examination is only significant for mild distension and tympany. Laboratory data reveal a leukocytosis of 13, but is otherwise unremarkable. A CT of the abdomen and pelvis with oral and IV contrast shows a distal small bowel obstruction with a transition zone in the right lower quadrant involving the distal, but not terminal, ileum. There are local inflammatory changes adjacent to the ascending colon, and there is concern over a stricture, possibly from inflammatory bowel disease and a subsequent adhesion.

The patient is admitted and bowel rest initiated with nasogastric suctioning. He shows no improvement in his symptomatology or physical examination findings over the next few days. Due to his lack of previous surgery and concern over stricture of the small bowel not resolving with non-operative management, the decision was made to take him to the operating room. An exploratory laparotomy was performed, and a Meckel's inflammatory phlegmon was encountered. A short segment of small bowel proximal and distal to the lesion was resected with primary anastomosis. An appendectomy was also performed. Pathology revealed a Meckel's diverticulum with focal structuring of small bowel without ulceration of the mucosa. The patient had an uncomplicated postoperative course with return of bowel function and tolerance of oral intake and was subsequently discharged without incident.

Epidemiology/Etiology/Pathophysiology

A Meckel's diverticulum is a true diverticulum, resulting from persistence of the omphalomesenteric duct (vitelline duct). This duct connects the yolk sac to the intestinal tract and often obliterates by the seventh week of life. The diverticulum is found on the anti-mesenteric border of the small bowel and is often within two feet from the ileocecal valve. The rich blood supply is from a persistent vitelline artery, which is a direct branch from the superior mesenteric artery. Only 2% of the population has a Meckel's, and only 2% of them become symptomatic. Males are affected more commonly than females in a 2:1 ratio. Symptoms vary based on age. The pediatric population often present with lower gastrointestinal (GI) hemorrhage, often within the first two years of life. Adults, on the other hand, most commonly present with obstruction. There is also the possible presentation as diverticulitis, which can be indistinguishable from appendicitis. Half of the diverticula contain ectopic tissue, and half of those contain gastric mucosa. The gastric mucosa is metabolically active; therefore, contralateral ulceration can result causing GI hemorrhage. Pancreatic tissue may also be present, but is less common than gastric. A hernia containing a Meckel's is called a Littre hernia. Due to the Meckel's embryologic origin, there may be extensions to the base of the umbilicus possibly resulting in draining fistulas, vitelline duct cysts, and umbilical sinuses.

Differential Diagnosis

Bleeding in the pediatric patient is most commonly caused by a Meckel's, but other sources to be considered include anal fissures, inflammatory bowel disease, intestinal polyps, intestinal duplications, arteriovenous malformation, and upper GI bleeding from peptic ulcer disease.

Bowel obstructions in adults are not commonly caused by Meckel's, so the differential should include adhesive disease, hernias, obstructing masses, ileus, inflammatory bowel disease, volvulus, and intussusception.

A diverticulitis-type presentation differential should include large bowel diverticular disease, appendicitis, inflammatory bowel disease, ulcerating mass, bowel perforation, volvulus, intestinal ischemia, and stercoral ulcer.

Diagnosis

When bleeding is the presenting symptom, scintigraphy with sodium 99mTc-pertechnetate is performed to evaluate for heterotopic gastric mucosa. Due to its specificity for this disease, it is also called a Meckel's scan. To increase the sensitivity and specificity (85 and 95%), pentagastrin, glucagon, and histamine-receptor antagonists can be administered. These medications indirectly increase metabolism of mucous-secreting cells, inhibit peristalsis, and increase radionuclide concentration in the diverticula, respectively.

Patients presenting with bowel obstructive or diverticular symptoms often require imaging studies, such as a CT scan of the abdomen and pelvis, to help determine the causes of those diseases. However, due to the vague presentation and similarity to many other diseases, Meckel's are often diagnosed at time of exploratory laparotomy for the presenting symptom [1, 2].

Management

Gastrointestinal bleeding in the pediatric population related to a Meckel's is often episodic, and therefore, stabilization of the patient can be performed before proceeding with surgery. Bowel obstructions should be managed non-operatively, but if a Meckel's is found as the source of the obstruction, surgical resection should be considered. Diverticulitis should be treated with broad-spectrum antibiotics before proceeding with resection. In short, symptomatic Meckel's should be treated surgically. Open or laparoscopic procedures are acceptable options for resection. Initially, the diverticulum should be assessed, mostly to evaluate for a large mesenteric vessel that could be extending to the tip of the diverticulum. This vessel

should be ligated. Subsequently, either a diverticulectomy or segmental bowel resection with primary anastomosis is appropriate. Incidental appendectomies are often performed concomitantly. If GI hemorrhage is the presenting symptom, wedge resection of the adjacent ulceration should be performed as well. Care should be taken to minimize narrowing of the bowel. A high chance of ileal narrowing, palpable ectopic tissue, extensive inflammation, non-reducible intussusception, wide-based diverticula, significant ileal ulceration, and ischemia are all indications for segmental ileal resection. Laparoscopy can be considered in patients with a negative Meckel's scan and high clinical suspicion.

The decision to proceed with surgical excision of a Meckel's found incidentally is less straightforward (Fig. 13.1). Some authors have suggested incidental Meckel's in adult should not be resected due to complications from the surgery outweighing the low chance of developing symptoms. Mortality (0.001%) is significantly low with the development of symptomatic Meckel's. As for the pediatric population, the controversy is increased due to it being a disease of the younger population, and the possibility of developing symptoms later in life. Some argue that if palpable heterotopic tissue is present, then a resection should be performed due to higher incidence of symptoms that could result from a diverticulum containing gastric mucosa. Still, others recommend resection in all children with an incidental diverticulum. Regardless, resection should only be undertaken in the optimal condition, devoid of peritonitis and hemodynamic instability [3, 4].

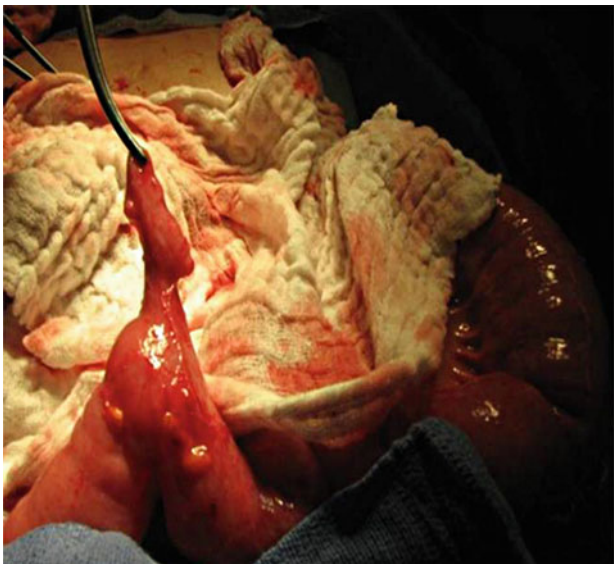

Fig. 13.1 Meckel's diverticulum found on exploratory laparotomy. © Dale Dangleben, MD

References

1. Cameron JL, Cameron AM. Management of symptomatic small bowel diverticula. In: Current surgical therapy. 11th ed. Philadelphia: Elsevier Saunders; 2014.
2. Dimick JB, Upchurch GR, Sonneday CJ. Rectal bleeding in a young child. In: Clinical scenarios in surgery: decision making and operative technique. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2012.
3. Passarge E, Stevenson RE. Meckel's diverticulum. In: Stevenson RE, Hall JE editors. Human malformations and related anomalies, 2nd ed. Oxford: Oxford University Press; 2006. p. 1111.
4. Zani A, Eaton S, Rees CM, Pierro A. Incidentally detected Meckel diverticulum: to resect or not to resect? Ann Surg. 2008;247:276.

Necrotizing Soft Tissue Infections

14

Aaron N. Sachs

Don't let the skin stand between you and the diagnosis

Learning Objectives

1. Understand how to clinically diagnose necrotizing fasciitis.
2. Differentiate different types of necrotizing soft tissue infection based on microorganism(s) involved.
3. Know that early diagnosis is critical to management.
4. Know risk factors for necrotizing fasciitis.
5. Know that early operative management is key and the basic approach.
6. Recognize that necrotizing fasciitis is a high-mortality and high-morbidity process.

Case Scenario

A 54-year-old woman with a history of poorly controlled diabetes presents to the emergency department complaining of increasing left forearm pain. A day prior she had accidentally caught her hand in a door, scratching her palm. Since then she has been having escalating pain in the hand migrating up the arm, coupled with swelling and fevers. On examination, she has a temperature of 38.4 °C, heart rate of 121, and blood pressure of 109/65. The left upper extremity is edematous up to the elbow with erythema extending to distally to the hand. There are multiple bullae

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with underlying crepitus, and the forearm is extremely tender to palpation. Laboratory studies showed a leukocytosis of 19,000, elevated creatinine, and low serum sodium.

Epidemiology/Etiology/Pathophysiology

Necrotizing fasciitis represents a wide spectrum of disease where a severe bacterial infection causes deep soft tissue necrosis. Synonymous terms include gas gangrene, synergistic necrotizing cellulitis, Fournier's gangrene (only when the male perineum is involved), Meleney's ulcers, clostridial myonecrosis and more colloquially, "flesh-eating" bacteria. Ultimately, all of these are considered necrotizing soft tissue infections caused by bacterial penetration of skin defenses, leading to tissue death. Pathologically, these microorganisms thrive in an area of low perfusion, leading to vasoconstriction, thrombosis, and local hypoxia. Toxin production can also lead to shock and multiple organ dysfunctions. Necrotizing fasciitis specifically refers to such an infection spreading in the deep fascia under subcutaneous tissue layers.

These are rare diseases affecting about 0.4 in 100,000 people. Risk factors include medical comorbidities such as diabetes, coronary disease, peripheral vascular disease, cirrhosis, advanced age, or any other condition that leads to a relatively immunosuppressed state. Prior surgery, intravenous drug abuse, or trauma can also lead to necrotizing fasciitis. However, 20% of infections can develop in the absence of any one of these factors.

Based on the microbes involved, necrotizing soft tissue infections can be divided into four types. Type I is most common—a polymicrobial infection consisting of both gram positive and negative aerobes, anaerobes, and even fungi. Overall, Type I infections are the least lethal and represent 75% of cases. Type II infections are monomicrobial and commonly associated with trauma or IV drug use. These organisms include group A streptococcus (most common), Clostridia perfringens, and Staphylococcus aureus. Type II infections are more aggressive than Type I infections. Type III infections are associated with saltwater and Vibrio vulnificus. These are uncommon but can rapidly lead to multi-organ failure and death. Finally, Type IV infections involve primarily fungal organisms [1–3].

Differential Diagnosis

Differential diagnosis of necrotizing fasciitis includes other similar necrotizing soft tissue infections such as Fournier's gangrene or clostridial myonecrosis, though ultimately one can consider them collectively. Less serious diseases to be considered include cellulitis, folliculitis, insect/spider bites, malignancy, or chronic venous disease.

Diagnosis

Necrotizing fasciitis is a clinical (not a radiographic) diagnosis where one needs to have a high index of suspicion. A thorough history and physical examination often clinches the diagnosis. Examination findings such as erythema, edema, vesicles, bullae, wound drainage, and crepitus are suspicious. Typically, pain out of proportion to exam findings or tenderness beyond an area of erythema can be a warning sign. The more drastic cutaneous manifestations, blue-black patches of tissue discoloration, sloughing, malodorous or purulent drainage may indicate a late, fulminant process. Systemic toxicity and progression of a previously diagnosed cellulitis despite appropriate antibiotics can also be seen.

Laboratory studies and imaging are nonspecific. Blood chemistries may show hypocalcemia, hyponatremia, or an elevated creatinine. Sodium levels less than 135 are associated with a higher mortality rate. Blood counts may show a leukocytosis or thrombocytopenia, and blood gas may show a metabolic acidosis. Elevated lactate may also be seen. The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) can be used as an adjunct but should not supplant clinical judgment [4]. Radiographs may show soft tissue gas, whereas MRI and CT may show fascial thickening, fluid collections, and fat stranding (Fig. 14.1).

The gold standard to diagnose necrotizing fasciitis is rapid surgical exploration. Intraoperative findings include loss of soft tissue planes (easily dissecting fascial layers), thrombosis of small vessels, and "dishwater," gray, or hemorrhagic drainage. Fascial biopsy can be confirmatory.

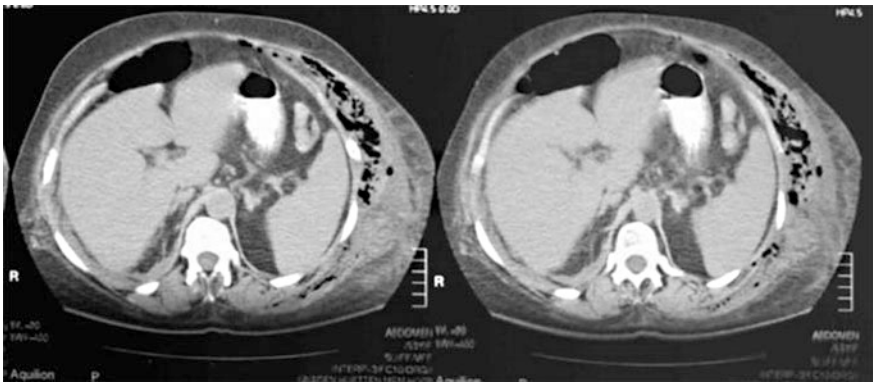

Fig. 14.1 CT scan showing extensive gas gangrene of the left flank and entire chest wall. © Dale Dangleben, MD

Complications

Complications of necrotizing fasciitis are serious, and thus early diagnosis is paramount. Delay in treatment can lead to sepsis, septic shock, multi-organ failure, limb loss, or death. The mortality rate is between 10 and 23.5%. Disability and disfigurement or limb contracture may occur in survivors.

Management

Early surgical exploration with aggressive debridement cannot be overemphasized. This should not be delayed even in the unstable patient. Fluid resuscitation, electrolyte correction, and control of hyperglycemia should be addressed concurrently. Broad spectrum antibiotics directed at polymicrobial organisms should be initiated and narrowed based on sensitivities of tissue cultures. Common combinations include penicillins with an aminoglycoside or fluoroquinolone. Clindamycin or metronidazole can be added for anaerobic coverage. Clindamycin can potentially reduce toxin formation. Vancomycin or linezolid may be added for MRSA coverage, and doxycycline should be utilized for any saltwater-associated infection.

In the operating room, the initial incision should be made as large as necessary. This is not the time to be minimally invasive. Underlying infection can be worse than appearance at the skin surface. Excision and debridement of all necrotic and infected tissues is the goal. Debridement until bleeding tissue can guide the extent of excision (Fig. 14.2). The ability to separate the fascia from surrounding tissues suggests nonviability tracking of infection. Wounds can be left packed open wet to dry or after the wound bed is cleaner possibly with negative pressure therapy. Anecdotally, some surgeons have used topical wound agents (such as mafenide acetate) between debridements. Most patients require additional surgical

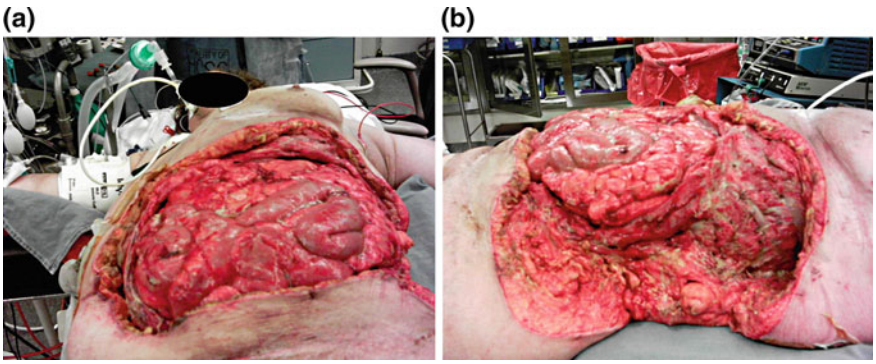

Fig. 14.2 **a**, **b** Extensive debridement of necrotizing infection with loss of entire abdominal and chest wall. © Dale Dangleben, MD

exploration within 24 h or as indicated clinically (usually up to three or four takebacks). Amputation may be required. Closure (sometimes requiring autologous flaps or grafts) can only be considered when there is no more evidence of infection. Delayed or inadequate initial debridement leads to increased mortality. Adjunct treatments such as plasmapheresis or hyperbaric oxygen are controversial, so operative intervention remains standard.

In perineal, genital or perianal infections, temporary or permanent diversion (which could be achieved with a sigmoid loop colostomy if properly constructed) may be required to avoid further fecal contamination in some patients (Fig. 14.3).

Postoperatively a brisk SIRS response can be expected, and hemodynamic monitoring in an intensive care unit is usually indicated. Antibiotics are narrowed based on intraoperative biopsy results and can be discontinued once clinical resolution of infection is seen and no more debridement is required.

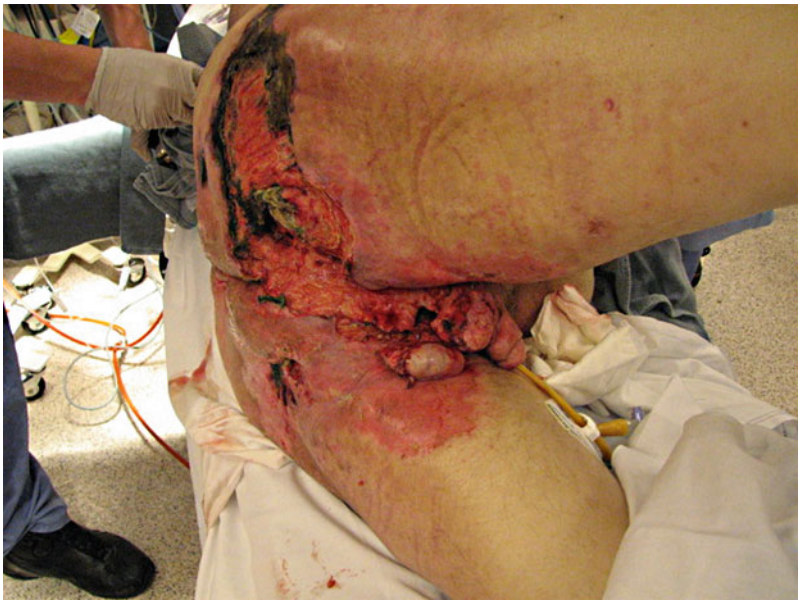

Fig. 14.3 Necrotizing infection of underlying sacral decubitus. © Dale Dangleben, MD

References

1. Cameron JL, Cameron AM. Necrotizing skin & soft tissue infections. In: Current surgical therapy. 11th ed. Philadelphia: Elsevier Saunders; 2014.
2. Dimick JB, Upchurch GR, Sonneday CJ. Necrotizing soft tissue infections. In: clinical scenarios in surgery: decision making and operative technique. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2012.
3. Greenfield LJ. Surgical infections. In: Greenfield's surgery: scientific principles and practice. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2006.
4. Wang YS, Wong CH, Tay YK. The LRINEC [Laboratory Risk Indicator for Necrotizing Fasciitis] score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections. Crit Care Med. 2004;32:1535–41.

Pancreatic Pseudocysts

15

Sheree A. Bray

Don't just do something, stand there

Learning Objectives

1. The learner should be able to formulate a differential diagnosis for cystic pancreatic lesions.
2. The learner should know the pathophysiology of pseudocyst formation.
3. The learner will learn the workup for a pancreatic cystic lesion.
4. The learner should be familiar with the complications of a pancreatic pseudocyst and how to treat them.
5. The learner should know when treatment for a pancreatic pseudocyst is indicated.
6. The learner should know the various percutaneous, endoscopic, and surgical treatments for pancreatic pseudocysts.

Case Scenario

A 36-year-old male with a strong history of alcohol abuse and a recent bout of acute pancreatitis 6 weeks ago presents to the emergency department with worsening upper abdominal pain, nausea, and early satiety. On physical examination, he is tender in his upper abdomen and has some fullness there as well. A CT shows a large cystic structure approximately 7 cm in tail of the pancreas in the lesser sac.

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Epidemiology/Etiology/Pathophysiology

Lacking a true epithelial lining, "pseudocyst" is a generic term that broadly describes chronic fluid collections associated with the pancreas. Further categorization developed by the Atlanta Classification subdivides this entity as acute peripancreatic fluid collections, pancreatic pseudocysts, walled-off necrosis, and acute necrotic collections.

Incidentally, the confusing term pancreatic abscess, previously defined as "localized collection of purulent material without significant necrotic material" is extremely uncommon and has therefore been eliminated from the most recent version of the Atlanta Classification.

Acute Peripancreatic Fluid Collection

An acute peripancreatic fluid collection can be found early (<4 weeks) in the course of acute pancreatitis or trauma. It is the result of the inflammatory process and results in a serous or exudative fluid. These lack a discrete wall of fibrinous or granulation tissue and do not contain solid material. These tend to resolve spontaneously and do not represent a ductal disruption.

Pancreatic Pseudocyst

These occur late (>4 weeks) in acute pancreatitis, chronic pancreatitis, neoplasm, or trauma. A pseudocyst is a collection of pancreatic enzymes surrounded by a wall of fibrous or granulation tissue. The wall often thickens as the pseudocyst matures. Pseudocysts account for the majority of cystic pancreatic lesions. They are caused by a disruption in the pancreatic duct and are a result of the inflammation from the leakage of pancreatic secretions. When pancreatic pseudocysts fail to resolve, it must be suspected that there is a persistent communication with the pancreatic ductal system. Based on CT scans, these occur in 10–20% of patients with acute pancreatitis and 20–40% of patients with chronic pancreatitis. It is more common in males as alcohol is a common source for the pancreatitis. Nearly half are found in the head of the pancreas with the rest being distributed through the remainder of the pancreas.

Walled-off Pancreatic Necrosis

This is a late complication of necrotizing pancreatitis or trauma. This is a collection of pancreatic or extra-pancreatic necrosis in a collection with a well-defined inflammatory wall. It usually occurs beyond 4 weeks in the setting of necrotizing pancreatitis.

Acute Necrotic Collection

Often a result of necrotizing pancreatitis, these are heterogeneous collections with variable amounts of fluid and necrosis that may involve the pancreatic parenchyma or peripancreatic tissues and they lack a well-defined wall.

Differential Diagnosis

It can be difficult to differentiate pseudocysts from other pancreatic cystic lesions. Other cystic pancreatic lesions to be considered include mucinous or serous cystic adenoma or neoplasm or congenital cysts.

Diagnosis

Acute Peripancreatic Fluid Collection

Most commonly, these patients are going to present with abdominal pain and evidence of acute pancreatitis with an elevated lipase and amylase. Imaging will show a fluid without any discrete cyst or wall.

Pancreatic Pseudocyst

Most patients present with abdominal pain or back pain late after an episode of acute pancreatitis, chronic pancreatitis, or trauma. They may have refractory pain or acute worsening of their initial pain. Pancreatic pseudocyst could be a source of increased or persistent pain in patients with chronic pancreatitis. Other common presenting symptoms include early satiety, nausea/vomiting, weight loss, jaundice, and low-grade fevers.

Serum lipase and amylase as well as liver function tests may be elevated on laboratory studies. Pseudocysts in the head of the pancreas can cause jaundice and biliary obstruction.

The initial imaging modality of choice is a contrast-enhanced CT scan. Ultrasound can demonstrate most pseudocysts and is a good tool for evaluating progression of the pseudocyst since it does not involve radiation exposure, although it is limited in the evaluation of the pancreas and retroperitoneum (Fig. 15.1). Additionally, MRI can delineate the amount of solid debris which may differentiate pseudocysts from cystic neoplasms when CT scans are equivocal. Ductal anatomy can be better defined by MRCP.

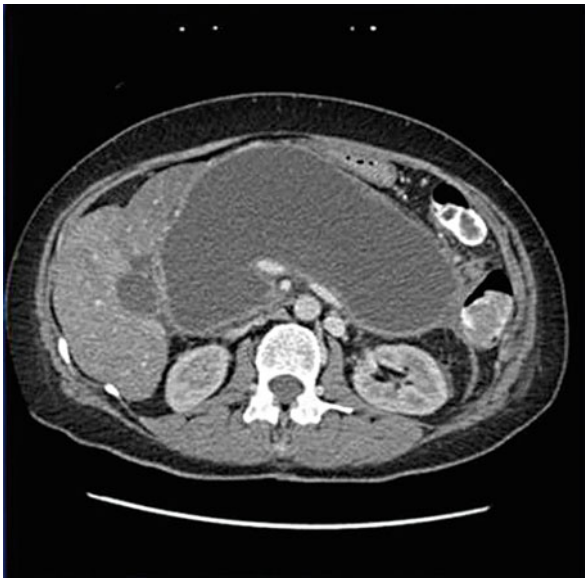

Fig. 15.1 Large pancreatic pseudocyst. © Dale Dangleben, MD

Endoscopic ultrasound (EUS) has emerged as an important adjunct, as it is useful in aspirating a sample of fluid from the pseudocyst which may be diagnostic. If fluid is sampled from the cyst, pseudocysts tend to have high amylase, low viscosity, low CEA, and low CA-125. Mucinous cystadenomas have variable amylase, high viscosity, and variable tumor markers. Serous cystadenomas have low viscosity, low CEA, but increased CA-125. Cystadenocarcinomas have high CEA, high CA-125, and positive cytology. Low CA 19-9 is strongly predictive of a non-mucinous cyst. However, percutaneous aspiration is not usually necessary for differentiation between pseudocyst and cystic neoplasms and may in fact be detrimental given the risk of seeding a sterile collection.

If advanced endoscopic capabilities are available, an endoscopic retrograde cholangiopancreatogram (ERCP) is useful for diagnosis and treatment planning is useful since nearly all patients with pseudocysts have ductal abnormalities and sphincterotomy, stenting, or stricture dilation can be performed at the same time if indicated.

Walled-off Pancreatic Necrosis

Imaging will show a well encapsulated collection with debris. It may have loculations. Fluid sampling can be done and may have a high amylase if there is a connection with the pancreatic duct. Fluid sampling can also determine if the collection is infected.

Acute Necrotic Collection

Extraluminal gas seen in a collection in acute necrotic collections (or walled-off necrosis) suggests infection. In equivocal cases, fine needle aspiration (FNA) for culture may clarify presence or absence of infection, although most cases could be managed without FNA particularly if percutaneous drainage is pursued as part of management.

Complications

Generally, pseudocysts can cause compressive complications as a result of biliary obstruction, small bowel obstruction, or mesenteric venous thrombosis. They can also erode into nearby structures such as the small bowel, colon, or vasculature causing a fistula, pseudoaneurysm, or even significant bleeding. The pseudocyst can potentially rupture and cause pancreatic ascites, peritonitis, or even a pleuropancreatic fistula.

Management

Acute peripancreatic fluid collection typically resolves spontaneously without any intervention. In asymptomatic patients, follow-up imaging is appropriate in several months, although there is no consensus on a suitable time interval.

Symptomatic pseudocysts that cause the above complications merit treatment. Historically pseudocysts >6 cm and those that persist for more than six weeks required intervention, however, it is now appropriate to continue to observe especially for small asymptomatic pseudocysts, and this previously considered "rule" is no longer the standard.

An ERCP should be performed to delineate the ductal anatomy, see if there is any stricture or stone, and determine if there is any communication. A pseudocyst that has communication with the pancreatic duct can often be treated with ERCP and sphincterotomy, stricture dilation, or transpapillary stent placement. If this fails or there is no communication with the pancreatic duct, then one should consider drainage procedures. Percutaneous drainage can be an option if there is a safe access route, little to no debris, and there is no evidence of complete pancreatic duct obstruction. Endoscopic drainage with cystogastrostomy, cystoduodenostomy, or cystojejunostomy can be attempted. The preferred site for drainage depends on where the pseudocyst is and what is in close proximity. Operative intervention is planned if these attempts are unsuccessful. Failure rates of percutaneous or endoscopic management are higher in patients with pancreatic duct disruption or stricture. Similar open and laparoscopic options exist—cystogastrostomy, cystoduodenostomy, cystojejunostomy, with the addition of Roux-en-Y cystoenterostomy. Operative external drainage

is an option when there is infection found or if the pseudocyst wall is still immature or too thin [1, 2].

For pancreatic ascites or pleuroperitoneal fistula, non-operative management should be performed initially with bowel rest and total parenteral nutrition. Paracentesis and thoracentesis are sometimes needed intermittently to resolve symptoms. If this persists beyond three weeks, then ERCP or MRCP should be done to delineate ductal anatomy. Distal pancreatic duct leaks or complete disruption requires distal pancreatectomy. Proximal duct leaks can be internally drained with a Roux-en-Y pancreaticojejunostomy.

For sterile, asymptomatic walled-off pancreatic necrosis expectant management is indicated. If any evidence of infection or if it becomes symptomatic, treatment options are similar to that of a pseudocyst in terms of drainage procedures. Pancreatic debridement is also a consideration depending on the degree of necrosis and infection [3].

Infected acute necrotic collections are managed with broad spectrum intravenous antibiotics and external drainage. Operative drainage can also be done especially if contents are particularly thick or if there are multi-loculated collections.

References

1. Howell DA, Shah RJ. Walled-off pancreatic fluid collections (including pseudocysts). 2014 Retrieved on 25 Feb 2016, from http://www.uptodate.com/contents/walled-off-pancreatic-fluid-collections-including-pseudocysts.
2. Morgan KA, Adams DA. The management of pancreatic pseudocyst. In: Current surgical therapy. 11th ed. Philadelphia, PA: Elsevier; 2014.
3. Rosato EL, Sonnenday CJ, Lillemoe KD, Yeo CJ. Pseuodocysts and other complications of pancreatitis. In: Shackelford's surgery of the alimentary tract. 7th ed. Vol. 2. Philadelphia, PA: Elsevier; 2013.

Paraesophageal Hernia

16

Dale A. Dangleben and Christine Du

Truly asymptomatic paraesophageal hernias can be managed expectantly

Learning Objectives

1. To define the different types of hiatal hernia.
2. To diagnosis a paraesophageal hernia.
3. To describe the repair of a paraesophageal hernia.
4. Recognize possible complications after a paraesophageal hernia repair.

Case Scenario

Fifty-year-old male presents with a 2-month history of difficulty swallowing with associated chest pain. He also complains of early satiety. The remainder of his history and physical are unremarkable.

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Epidemiology/Etiology/Pathophysiology

Hiatal hernias define all defects involving the diaphragmatic esophageal hiatus. The exact etiology of the development of these defects is unknown but is more common in older and obese patients. There are four types of hiatal hernia defined by the position of the gastroesophageal junction and the extent of the stomach and/or other organs herniated through the esophageal hiatus. Specifically, a paraesophageal hernia is a type II hiatal hernia. A paraesophageal hernia is when a portion of the stomach has herniated into the chest into a well-defined hernia sac with the gastroesophageal junction (GEJ) fixed in its normal position. It accounts for <3–5% of all hiatal hernias. The remaining hiatal hernias are type I, and the most common hiatal hernia is a sliding hernia where the GEJ slides into the chest; type III is a combination of types I and II; and type IV—the entire stomach and some other organ—is intrathoracic.

Many type I, II and III hiatal hernias are found incidentally during X-ray imaging studies or an upper endoscopy (Fig. 16.1). With type I hiatal hernias, the patients usually have symptoms of gastroesophageal reflux. Type II and III hiatal hernias can present with mechanical obstructive symptoms . Acute symptoms may be present if there is complete obstruction or strangulation of the stomach, for which

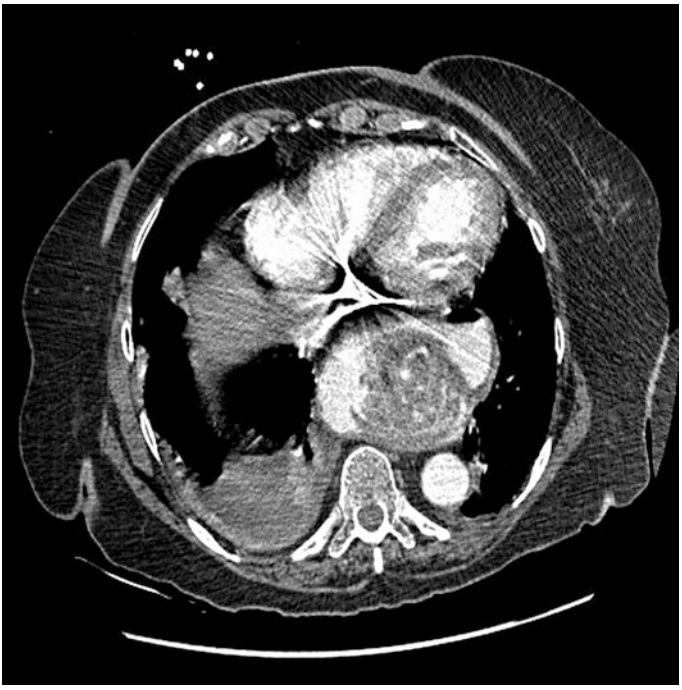

Fig. 16.1 This CT scan illustrates a gastric volvulus of a paraesophageal hernia. The "swirl" is seen within the stomach in the chest. © Dale Dangleben, MD

paraesophageal hernias are at higher risk of incarceration or strangulation of the herniated stomach. If a patient presents acutely and emergently, one should attempt to place a nasogastric tube for gastric decompression. Whether this is successful or not, the patient will undergo surgical repair. Borchardt's triad indicating an incarcerated hernia is chest pain, inability to vomit and inability to pass a nasogastric tube. If the nasogastric tube is unable to passed, this is a surgical emergency [1–5].

Differential Diagnosis

Differential includes, Angina, GERD, esophageal spasm, pneumonia, myocardial infarction, nutcracker esophagus.

Diagnosis

As stated prior, many paraesophageal hernias are found incidentally, most by X-ray or contrast studies (Figs. 16.2 and 16.3). Upper GI studies can be obtained to confirm diagnosis made on chest X-rays. An esophagogastroduodenscopy

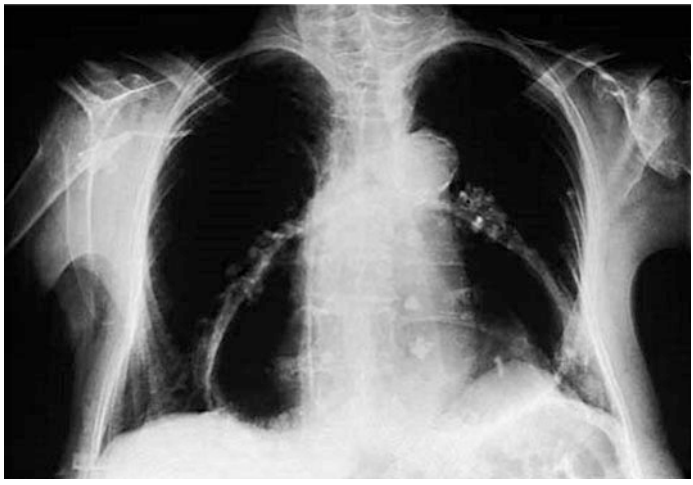

Fig. 16.2 Large paraesophageal hernia is seen here on chest X-ray overlying the cardiac silhouette. © Dale Dangleben, MD

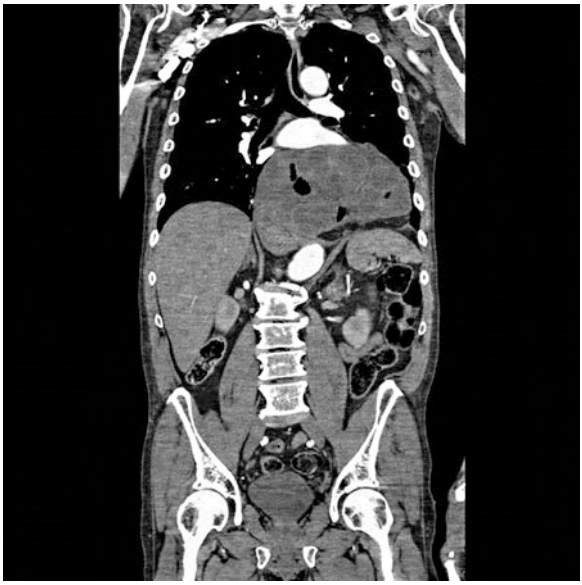

Fig. 16.3 Large paraesophageal hernia is seen here on CT scan. Most of the stomach is within the thoracic cavity. © Dale Dangleben, MD

(EGD) should be performed to evaluate all patients with paraesophageal hernias to assess any other possible pathology, such as Barrett's esophagitis. Manometry can be used to determine esophageal motility abnormalities, but in the setting of distorted anatomy, results are either unobtainable or difficult to interpret. The same is true for 24-h pH monitoring.

Complications

Gastric volvulus is a known complication of paraesophageal hernia. There are two types of gastric volvulus: mesenteroaxial and organo axial. Organo axial is the most common when there is twisting of the stomach along its long axis. Mesenteroaxial is along the short axis of the stomach and is less common. The inability to pass a nasogastric tube, retching without vomiting and epigastric pain is known as Borchardt's triad and associated with paraesophageal hernia volvulus.

There must be vigilance when it comes to operative and postoperative complications. The operative complications are vagus nerve injury, esophageal or gastric perforation. Pay attention to the spleen and the division of the short gastric to avoid bleeding. Postoperatively, the complication can be the same such as missed perforation, bleeding and also recurrence and dysphagia. If the pleura was violated, there is risk of pneumothorax or effusion.

Management

Whether you perform your repair laparascopically or open, the operation involves the need for reducing the hernia and the sac, excision of the sac, mobilization of the fundus by taking the short gastric vessels, esophageal mobilization and dissection of the crura, closure of the diaphragmatic defect, and anchoring of the stomach. One can anchor the stomach with a gastrostomy tube or a fundoplication. A Nissen (posterior 360°) fundoplication is applicable provided that the patient has no underlying esophageal dysmotility (e.g., scleroderma, achalasia). If there is an underlying motility disorder, a Dor or Toupet (partial) fundoplication can be performed (Fig. 16.4) [6–8].

Postoperatively, there is no need for routine follow-up radiographic examinations. Patients are started on clear liquid diet and slowly advanced with instruction to avoid carbonated beverages. They may have still experience early satiety and/or dysphagia in the immediate postoperative period likely either due to edema, which then the symptoms should improve, or the wrap from the Nissen fundoplication being too tight. These types of symptoms should resolve postoperatively in approximately a month's time. If these symptoms do not improve and ultimately do

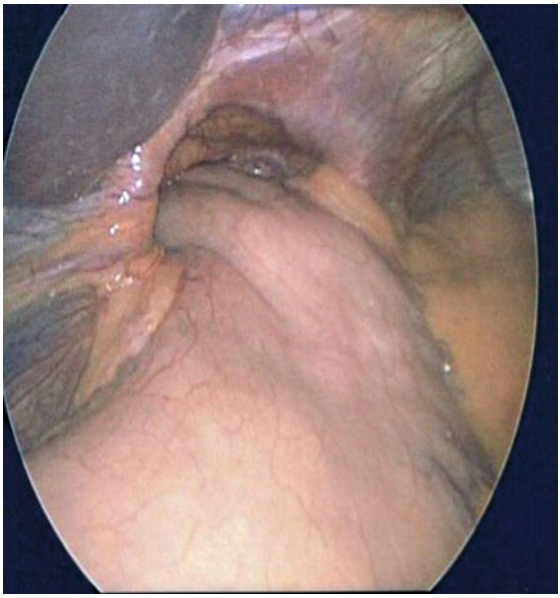

Fig. 16.4 Laparoscopic view of a large paraesophageal hernia. © Dale Dangleben, MD

not subside, then initially the patient should be evaluated with an upper GI contrast study and possibly need upper endoscopy.

References

1. Bhayani NH, Kurian AA, Sharata AM, Reavis KM, Dunst CM, Swanstrom LL. Wait only to resuscitate: early surgery for acutely presenting paraesophageal hernias yields better outcomes. Surg Endosc. 2013 Jan;27(1):267–71.
2. Conway N, Swanstrom LL. Paraesophageal hernia: indications and techniques. In: Swanstrom LL, Dunst CM, editors. Antireflux surgery. New York: Springer; 2015. p. 105–15.
3. Gastal OL, Hagen JA, Peters JH, et al. Short esophagus: analysis of predictors and clinical implications. Arch Surg. 1999;134:633–6.
4. Hashemi M, Peters JH, DeMeester TR, et al. Laparoscopic repair of large para-esophageal hernia: objective follow-up reveals high recurrence rate. J Am Coll Surg. 2000;190(5):553–60.
5. Oelschlager BK, Pellegrini CA, Hunter J, et al. Biologic prosthesis reduces recurrence after laparoscopic paraesophageal hernia repair: a multicenter, prospective, randomized trial. Ann Surg. 2006;244(4):481–90.
6. Oleynikov D. Paraesphageal hernia. In: Surgical approaches to esophageal disease, an issue of surgical clinics. Surgical clinics of North America. Elsevier; June 2015.
7. Petersen RP, Pellegrini CA, Oelschlager BK. Hiatal hernia and gastroesophageal reflux disease. In: Townsend CM, Beauchamp RD, Evers BM, Mattox ML, editors. Townsend: sabiston textbook of surgery (Chapter 42). 20th ed. Philadelphia, PA: Elsevier Saunders; 2016.
8. Pierre AF, Luketich JD, Fernando HC, et al. Results of laparoscopic repair of giant paraesophageal hernias: 200 consecutive patients. Ann Thorac Surg. 2002;74:1909–16.

Pneumobilia

17

Laura Lyn Rivera and Eric Melchior

Until proven otherwise, small bowel obstruction and pneumobilia on a plain film is diagnostic of gallstone ileus

Learning Objectives

1. Establish differential diagnosis of pneumobilia, with most common cause being spontaneous biliary-enteric fistula.
2. Review the surgical and procedural causes of pneumobilia.
3. Review the diagnosis, evaluation and management of gallstone ileus.

Case Scenario

A 67-year-old female presented to the emergency department with fever, diffuse abdominal pain and vomiting for three days. Her last bowel movement was five days before. On initial evaluation, she was tachycardic at 102 with a blood pressure of 136/78, with a soft, moderately distended, tympanic and diffusely tender abdomen, without any signs of peritoneal inflammation. Her history was otherwise

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unremarkable, and she has no past surgical history. Laboratory tests showed a white blood cell count of 14,000/mL and C-reactive protein 26; basic metabolic panel, red blood cell count, hemoglobin, hematocrit, amylase and lipase were within normal range. Plain abdominal X-ray showed pneumobilia. Upper gastrointestinal series showed communication between the duodenum and the gallbladder that was filled with air and contrast marking the biliary tree. Computed tomography scan showed a bilioenteric fistula between the gallbladder and the duodenum and identified a 3.5-cm gallstone in the distal ileum.

On the following day, cholecystectomy, extraction of gallstone and closure of the bilioenteric fistula were performed via midline laparotomy where a longitudinal enterolithotomy was made along the antimesenteric border proximal to the point of impaction. The stone was milked proximally and extracted. The enterotomy was closed transversely to avoid bowel stenosis. The patient made a complete full recovery.

Epidemiology/Etiology/Pathophysiology

Pneumobilia, or air within the biliary tree, usually suggests an abnormal communication between the biliary tract and the intestine or an infection caused by gas-forming bacteria. The three most common causes are a spontaneous biliary-enteric fistula, incompetent sphincter of Oddi and biliary-enteric surgical anastomosis. Sphincterotomy during ERCP is another fairly common cause given the current ubiquity of that procedure. The incidence of pneumobilia is much higher after surgical intervention within or near the biliary tree.

A spontaneous biliary-enteric fistula is the most common non-iatrogenic cause of pneumobilia and is seen in 90% of cases. This is associated with gallstones as seen in gallstone ileus. Ischemic necrosis of the gallbladder wall is created by the repetitive obstruction of stones on the cystic duct and chronic inflammation. Eventually, the fistula tract forms and the stone, typically measuring greater than 2.5 cm in diameter, can pass and become dislodged in the right iliac fossa about 60 cm proximal to ileocecal valve. Both an ulcer and a tumor can also erode through the tissues and form a fistula between the biliary system and an adjacent organ. The different types of fistulae can be cholecystoduodenal (70%), cholecystocolic (14%), cholecystogastric causing gastric outlet obstruction as seen in Bouveret's syndrome (6%), and choledochoduodenal (4%). Biliobiliary (cystic to common duct) fistulae tend to be rare.

Patients typically present with acute mechanical intestinal obstruction caused by a large stone lodged in the bowel lumen. Gallstone ileus causes 1% of all small bowel obstructions and has a female predilection. It is an uncommon complication of gallstone disease, occurring in 0.5% of cases. Morbidity and mortality are reported at about 20%. Patients may have a remote history of gallbladder disease without any history of prior surgeries or hernias on physical examination.

Differential Diagnosis

Pneumobilia and portal vein gas may appear similar on abdominal X-ray but can be distinguished by its appearance on CT scan. Differential diagnosis of these two is essential because patients with portal venous gas (Chap. 19) usually have acute mesenteric ischemia which is a surgical emergency. Mesenteric ischemia is associated with more than 50% of cases of portal venous gas but is not associated with pneumobilia, and mortality is as high as 70%. Other causes of portal venous gas include diverticulitis, ulcerative colitis, pancreatitis, necrotizing enterocolitis or after a liver biopsy. Pneumobilia is found in a more *central* location of the liver with fewer branches than portal venous gas, the latter usually seen more *peripherally*.

Pneumobilia can be secondary to a spontaneous internal biliary fistula causing gallstone ileus, incompetent sphincter of Oddi, biliary-enteric surgical anastomosis, emphysematous cholecystitis, cholangitis, liver abscess, blunt trauma, iatrogenic, post-ERCP with sphincterotomy, PUD or rarely gallbladder cancer.

Diagnosis

Pneumobilia in patients with a history of choledochoduodenostomy is considered benign and incidental if in the absence of clinical symptoms. An incompetent sphincter of Oddi causes duodenobiliary reflux, bacterial contamination and chronic inflammation. This is mostly due to iatrogenic causes (e.g., sphincterotomy) and usually causes pneumobilia in 40% after 1-year follow-up. Non-iatrogenic causes of incompetent sphincter of Oddi include tumors, calculi, adhesions and fibrosis secondary to pancreatitis.

Emphysematous cholecystitis is differentiated by also presenting with gas in the gallbladder wall, lumen and adjacent soft tissues. Acute ascending cholangitis presents with pneumobilia in 22% of cases identified on CT scan. Pneumobilia can be a benign and expected finding with most iatrogenic manipulation of the biliary tract such as after gallbladder surgery, in 80–90% of patients after a Whipple procedure, and after percutaneous transhepatic cholangiography.

Patients with pneumobilia secondary to gallstone ileus can present with non-specific symptoms of small bowel obstruction with episodic pain, nausea, vomiting and abdominal distention over several days. Only 20–50% is correctly diagnosed at preoperative diagnosis. Abdominal radiography demonstrates the findings of Rigler's triad such as dilated small bowel suggesting of small bowel obstruction, an impacted gallstone in the distal ileum in addition to the classic pneumobilia (Fig. 17.1). This triad is seen in less than 35% of plain films. CT scan is the diagnostic study of choice because of the high sensitivity and specificity. It can adequately measure the stone and localize the anatomical site of the obstruction and the fistula [1, 2].

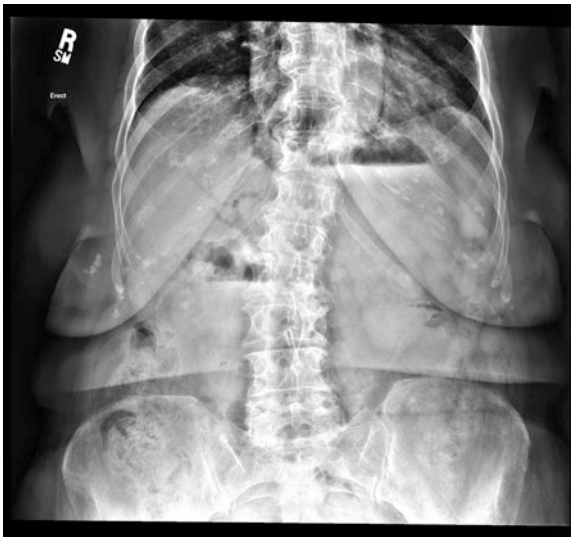

Fig. 17.1 Plain abdominal film showing the outline of the biliary tree with air (Pneumobilia). © Dale Dangleben, MD

Complications

The most important complication that should be considered is the higher rate of mortality when performing a two-stage procedure and performing a cholecystectomy as well as closing the fistula in the presence of acute inflammation associated with gallstone ileus. The rate of recurrence of gallstone ileus from leaving the gallbladder intact is less than 5%. Therefore, advocating for a single enterolithotomy is favored, particularly in frail or elderly patients.

Failure to palpate for and extract any additional stones is the most likely cause of recurrence. Attempting to extract the stone through an antegrade enterotomy can result in significant mucosal injury and is therefore not recommended. The impacted stone can be large enough to cause complete obstruction and cause pressure ischemia of the bowel wall at the site of impaction. Therefore, the bowel should be carefully inspected for any signs of ischemia prompting bowel resection. The enterotomy is made proximally for this same reason and to avoid dehiscence from a suture line at that site that can become compromised. The purposeful enterotomy is then closed in a transverse fashion to avoid narrowing.

Enteric leak is another complication to be aware of as most patients are elderly, frail and malnourished. Leakage can be from a technical complication at the site of stone extraction or from a cystic duct or duodenal repair if the patient underwent a two-stage procedure.

Management

After ruling out benign etiologies and identifying the cause of pneumobilia as being from gallstone ileus, the initial management consists of fluid resuscitation, correction of electrolyte imbalance, NGT decompression and relieving the bowel obstruction through a small proximal longitudinal enterotomy to extract the stone in retrograde fashion. A midline laparotomy is done, and the bowel is inspected for areas of ischemia or other intraluminal gallstones. Common locations for stones are in the distal ileum, followed by the jejunum and gastric outlet. The enterotomy should then be closed in a transverse orientation to avoid stricture. There is a two-stage procedure that involves the enterotomy and a subsequent cholecystectomy if the patient can tolerate it. Otherwise, the enterotomy can be done alone and an elective cholecystectomy can be done at a later date, usually in 4–6 weeks. The biliary-enteric fistula closes spontaneously in the majority of patients. The one-stage procedure carries a higher mortality (16.8%) than the two-stage procedure (11.8%) [3]. This is typically because of the age and other comorbidities of the patients presenting with this condition that adding operative time and complexity to the procedure should be a judicious consideration.

References

1. Mueller CL, Neville AA, Fried GM. Management of complicated gallstone disease. Sci. Am. Surg. 2013;122.
2. Rajkovic Z, Papes D, Altarac S, Arshani N. Differential diagnosis and clinical relevance of pneumobilia or portal vein gas on abdominal X-ray. Acta Clin Croat. 2013;52:369–73.
3. Reisner RM, Cohen JR. Gallstone ileus: a review of 1001 reported cases. Am Surgeon. 1994;60:441–6.

Pneumoperitoneum

18

Nicole Melchior

Treat the patient, not the scan

Learning Objectives

1. The learner will be able to formulate a differential diagnosis of pneumoperitoneum.
2. The learner will be able to identify patients who need immediate operative intervention.
3. The learner will be able to appropriately resuscitate the patient prior to the operating room.

Case Scenario

A 72-year-old male with a past medical history significant for hypertension presents to the emergency room complaining of a 3-day history of abdominal pain. The pain was initially localized to his left lower quadrant, but became diffuse this morning. He reports that the car ride to the hospital was very painful. He has had prior episodes of left lower quadrant pain previously, but they were all self-limited. He also complains of nausea and had one episode of non-bilious emesis this morning. His last colonoscopy was over 20 years ago, and he reports it was normal. He has

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never had upper endoscopy. He appears uncomfortable, and on physical exam, he is febrile, with a heart rate of 108 and blood pressure of 121/72 with a distended, diffusely tender abdomen with rebound. His white blood cell count is 15,000.

A computed tomography (CT) scan of the abdomen and pelvis was ordered by the emergency department and demonstrated pneumoperitoneum consistent with a perforated viscus, multiple sigmoid diverticula with pericolonic stranding of the sigmoid colon and areas of extraluminal air surrounding the small and large bowel. Large-bore IV's were placed, and the patient was resuscitated with normal saline. Broad-spectrum antibiotics were administered. He was taken to the operating room and underwent a Hartmann procedure for perforated diverticulitis. Postoperatively, he was initially monitored in the ICU and recovered slowly until he was ultimately discharged home on POD 10.

Epidemiology/Etiology/Pathophysiology

The incidence of gastrointestinal perforation in patients who are not taking nonsteroidal anti-inflammatory drugs is 0.1 per 1000 person-years. A perforated viscus is the most common etiology of pneumoperitoneum, which is defined as air in the peritoneal cavity; however, the differential diagnosis is broad. Of patients with visceral perforation who exhibit free air on imaging studies, the most common causes are peptic ulcer and diverticulitis, followed by trauma.

In cases of pneumoperitoneum due to perforated viscus, the gastrointestinal wall becomes inflamed which eventually leads to ischemia and then necrosis, resulting in full-thickness destruction of the gastrointestinal wall. Loss of gastrointestinal wall integrity then leads to leakage of enteric contents. Exposure of the peritoneal cavity to these intraluminal contents results in chemical peritonitis followed by secondary bacterial peritonitis, which triggers a systemic inflammatory response syndrome in the body.

Differential Diagnosis

As in other area of acute care surgery, early diagnosis and prompt treatment are paramount. The differential diagnosis list is exhaustive but most importantly includes perforated viscus as a result of peptic ulcer disease, diverticular disease, ischemic bowel disease or bowel obstruction, neoplasms, trauma or impacted foreign body or iatrogenic causes such as nasogastric tube placement, upper or lower endoscopy or esophageal dilation.

Additionally, benign, nonsurgical causes need to be kept in mind such as recent laparotomy (for up to 10 days postoperatively), laparoscopy (for up to 5 days postoperatively), or percutaneous gastrostomy tube placement or instrumentation

(e.g., endoscopy, stent placement, paracentesis), peritoneal dialysis, and occasionally mechanical ventilation with elevated positive end-expiratory pressure (PEEP) settings.

Less frequently, free air can also be observed after vaginal instrumentation or surgery, with bacterial infections such as typhoid fever or with pneumatosis cystoides intestinalis, spontaneous or idiopathic pneumoperitoneum is a diagnosis of exclusion.

Diagnosis

The preferred initial diagnostic study for cases of suspected pneumoperitoneum is upright (erect) chest X-ray, which is quick, inexpensive and universally available. Obtaining multiple views, such as an upright chest, supine abdominal and left lateral decubitus is ideal. The positive rate of free air is highest in a left lateral decubitus abdominal X-ray (98%), followed by erect chest X-ray (85.1%). The classic finding on plain film is a lucent stripe beneath the diaphragm, which is usually best identified under the right hemidiaphragm (lucent liver sign) (Fig. 18.1). Other signs which may be seen in cases of pneumoperitoneum include Rigler's sign, or the double wall sign, which is when air outlines both sides of the bowel wall and may be seen when at least 1 L of air is present.

When plain radiographs are non-diagnostic and the patient is stable and able to undergo additional imaging studies, computed tomography (CT) is the next study of choice for adult patients as it is a more sensitive study. A CT scan may also help to localize the site of perforation and hence aid in operative planning but should not delay surgical intervention in an acutely ill patient (Fig.18.2).

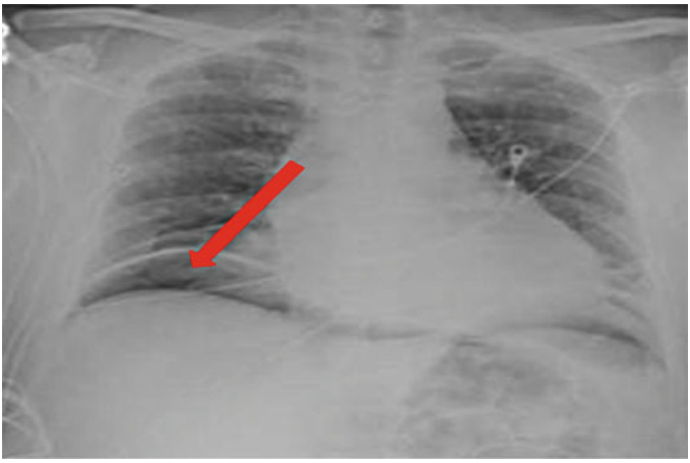

Fig. 18.1 "Free Air" is seen here under the right diaphragm. © Dale Dangleben, MD

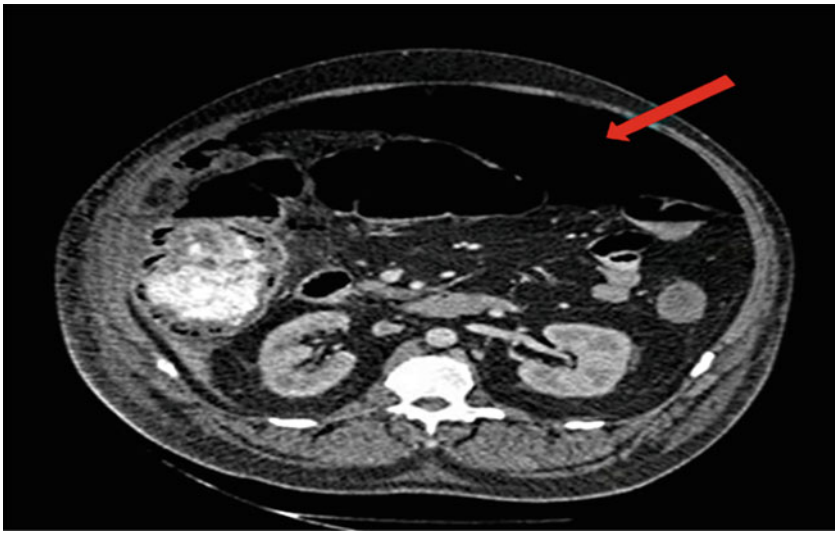

Fig. 18.2 Large amount of pneumoperitoneum "Free Air" is seen here outside the colon. There is also pneumatosis of the right colon. © Dale Dangleben, MD

In children or pregnant patients, one should consider ultrasonography, as in some cases ultrasound may be superior to plain films in these groups.

One should also be aware of "pseudopneumoperitoneum," or air in the abdominal cavity that appears to be intraperitoneal but is actually contained in an organ. An example of such a finding is Chilaiditi's sign: colonic interposition between the liver and diaphragm [1].

Complications

Complications of pneumoperitoneum due to perforated viscus include abscess or phlegmon formation, peritonitis, septic shock, multi-organ failure and death should the diagnosis be delayed or missed. Early intervention is crucial, as patients with perforated viscus and peritonitis have a mortality rate of greater than 20%.

Management

In a patient with pneumoperitoneum on imaging and peritonitis on examination, emergent operative intervention is warranted; however, resuscitation must be initiated as the operating room is being mobilized. In a patient with signs of sepsis or septic shock, adequate intravenous access must be obtained, either with two

large-bore IVs or with a central venous catheter if necessary as vasopressor support may be required. Arterial line placement should be strongly considered for hypotensive patients especially if vasopressor support is instituted. Intravenous fluids and broad-spectrum IV antibiotics should be given and electrolytes should be corrected. A nasogastric tube should be placed to decompress the stomach (and its position confirmed at operation), and a foley catheter should be placed for monitoring of urine output. It should be emphasized that the above lines, tubes and drains should not delay transport from the emergency department as they can be inserted in the operating room to save precious time [2, 3].

As resuscitation continues, surgical intervention should be instituted rapidly and will encompass a variety of procedures depending on the operative findings. For peptic ulcer disease, one ought to choose the easiest operation: Most commonly a Graham (omental) patch (without closure) is performed. Resection often adds morbidity without benefit. Acid will be controlled by medications; therefore, highly selective and selective vagotomies are no longer indicated. Postoperatively, patients should be placed on a proton-pump inhibitor. For cases due to perforated diverticular disease, a Hartmann procedure (sigmoidectomy with end colostomy) is typically required. Current studies investigating the utility of laparoscopic lavage or primary anastomosis with a protective, proximal, diverting loop ileostomy in this setting are promising, yet more studies are needed to formalize a clinical guideline. To summarize, the surgeon should do the simplest and most expeditious operation to get the patient off the operating table without engaging in acrobatics particularly in the acutely ill patient.

When encountered with a case of localized low-volume pneumoperitoneum that appears to be contained, non-operative management may be considered, provided the patient has stable hemodynamics and no peritoneal signs on abdominal examination.

References

1. Kumar, et al. The etiology of pneumoperitoneum in the 21st century. J Trauma Acute Care Surg. 2012;73(3):542–8.
2. Langell JT, Mulvihill SJ. Gastrointestinal perforation and the acute abdomen. Med Clin North Am. 2008;92(3):viii–ix, 599–625.
3. Reich H, Chou D, Melo N. Perforated Hollow Viscus. In: Butler Kathryn L, Harisinghan Mukesh, editors. Acute care surgery: imaging essentials for rapid diagnosis. McGraw-Hill Education: China; 2015.

Portal Venous Gas

19

John Brady

Mesenteric ischemia until proven otherwise

Learning Objectives

1. Establish differential diagnosis of portal venous gas, with most common cause being intestinal ischemia.
2. Laparotomy is mandated for portal venous gas with acidosis or radiologic evidence of bowel ischemia.
3. Establish consideration for vascular causes of intestinal ischemia, and prompting appropriate intra-operative evaluation and consultation, if needed.
4. Establish low threshold for laparotomy or diagnostic laparoscopy in cases where diagnosis is unclear.

Case Scenario

A 74-year-old female with a past medical history of atrial fibrillation on warfarin therapy, hypertension, and diabetes presents with acute onset central abdominal pain that began last evening. She is febrile, tachycardic, and diaphoretic. Physical examination shows a rigid, diffusely tender abdomen. Laboratory studies demonstrate a white blood cell count of 16,000 with 10% bands, BUN of 63 with

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creatinine of 3.5, lactate of 6.7, and INR of 0.9. Computed tomography scan of the abdomen without contrast shows portal venous gas with thickening and stranding of right colon and large portion of the distal small bowel. A brief period of resuscitation was performed in the emergency department, and broad spectrum antibiotics were given.

She was taken to the operating room for exploratory laparotomy, and the distal ileum and right colon were found to be necrotic. Other portions of small bowel appeared viable. She underwent right hemicolectomy with ileal resection and was left in discontinuity, with temporary abdominal closure. Further resuscitation followed for the next 48 h in the intensive care unit. Second look laparotomy demonstrated viable bowel, and ileocolonic anastomosis was created followed by definitive abdominal closure. She was discharged to a rehabilitation facility on postoperative day eighteen, after complications of renal failure and need for intermittent hemodialysis.

Epidemiology/Etiology/Pathophysiology

Portal venous gas is a very rare radiologic finding that is estimated to be found on less than 0.1% of all CT scans of the abdomen. Men and women are equally likely to have this radiologic finding, and due to unclear reasons, it tends to be more common in the left lobe of the liver. Portal venous air is an ominous sign that could be suggestive of an acute intra-abdominal catastrophe requiring prompt surgical intervention, or it could be rapidly fatal. The mechanism and pathophysiology for the appearance of portal venous air is largely unknown. One of the most likely theories is that gas escapes from the bowel wall, either due to ischemia or gas producing organisms, and is then circulated into the hepatic portal system. It has been hypothesized that any disruption in mucosal integrity can lead to easier access of air into the bowel vasculature and subsequent portal venous air. Even less is known of the reason for this radiologic finding with benign causes. Classically, when seen in conjunction with bowel ischemia, portal venous gas is associated with transmural bowel necrosis in 91% of patients, with an associated mortality of 85%. Also, portal venous gas on plain films is associated with a mortality rate of 75%. However, recent studies suggest that the overall mortality for portal venous air is 29–39%, likely reflecting increasingly seen benign causes, or earlier recognition of surgical causes [1−3].

Differential Diagnosis

The differential of portal venous gas is vast. Interestingly, the differential ranges from fatal and life threatening causes requiring emergent operative intervention to completely benign causes with only supportive care required. The overwhelmingly

most common cause (43–70%) is intestinal ischemia with necrosis. Other common causes include ulcerative colitis (8%) and intra-abdominal abscesses (6%). Other etiologies include gastrointestinal dilation, gastrointestinal inflammation, sepsis, endoscopic procedures, peptic ulcer disease, blunt trauma, and abdominal malignancies. Benign causes are innumerable, but include current chemotherapy, bronchopneumonia, cystic fibrosis, diabetic ketoacidosis, seizures, hyperbaric decompression, intra-aortic balloon pulsation, hemodialysis, caustic ingestion, and cryptosporidium infections. Differentiation between these diagnoses requires a careful history and a physical examination by an experienced clinician.

Diagnosis

Patient evaluation with emphasis on history and physical examination is important to first exclude gastrointestinal tract ischemia and, then, evaluate for other more common, and likely benign causes. Yet diagnosis is most often made based on abdominal CT imaging, but can be evident on abdominal plain films or ultrasonography. Portal venous air is important to discern from pneumobilia. Differential diagnoses and in turn management between the two radiologic entities differ. Portal venous gas extends to the *periphery* of the liver, while pneumobilia localizes *centrally*. This is due to the flow of the system in which the air is present, with portal venous flow centrifugal (away from the liver) and hepatic biliary flow centripetal (toward the liver). The mainstay imaging modality is CT scanning with the ability to obtain subsequent CT angiography (CTA) should there be clinical suspicious of mesenteric ischemia (Fig. 19.1).

Complications

Significant mortality is associated with merely the presence of portal venous gas. Complications germane to the need for emergent surgery in often contaminated fields include a high incidence of wound infections. Given the vast array of causes of portal venous gas, conservative management can be chosen for a patient who requires operative intervention. The conversion of conservative to operative management should be considered a misdiagnosis and, thus, a potential complication as well.

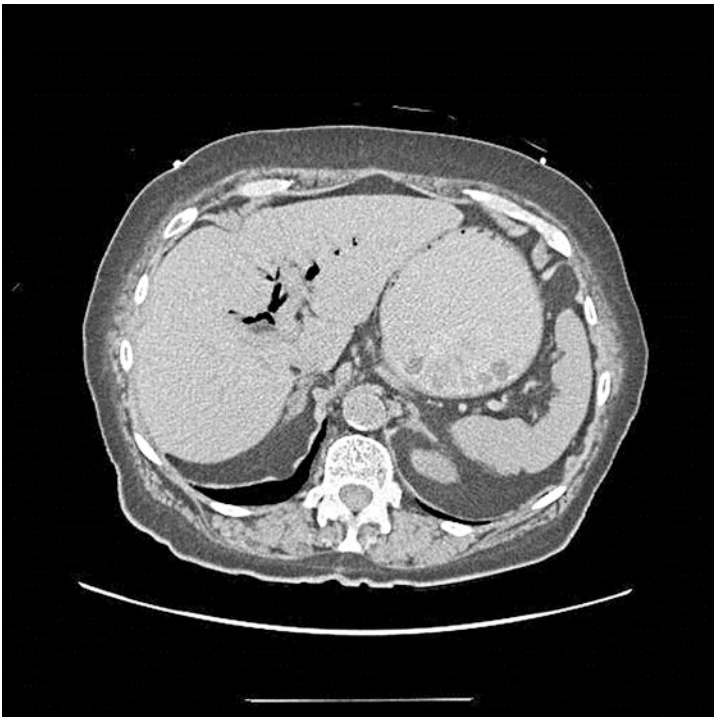

Fig. 19.1 CT scan illustrating portal venous gas. © Dale Dangleben, MD

Management

Early treatment of the underlying cause of portal venous gas is critical. As the most common cause is intestinal ischemia, the focus of management should be to clinically and radiologically evaluate for possible bowel ischemia or rule it out. With the multitude of differential diagnoses, all of which have to be considered, the management occasionally becomes one of the surgical gestalts. Some authors have attempted to approach this in a systematic fashion based on the historic mortalities, which parallel the incidence of bowel ischemia.

Portal venous gas with concomitant radiologic findings of bowel ischemia mandates laparotomy. These include, but are not limited to, pneumatosis intestinalis and pneumoperitoneum. Some authors have recommended laparotomy in the absence of other testing if portal venous gas is present on plain abdominal film, again due to the documented high associated mortality, but this remains controversial as benign non-ischemic causes need to be considered. Acidosis suggesting

ischemia should also be viewed as a relative indication for exploration. Perioperative management includes broad spectrum antibiotic therapy and aggressive preoperative resuscitation. Operation consists of exploratory laparotomy with assessment of bowel viability and need for resection. Non-viable bowel is resected and left in discontinuity, as damage control laparotomy is usually required, given critical illness and the unfavorable conditions for anastomosis creation. Careful inspection of remaining bowel is performed, noting any ischemic areas to be assessed at a second look laparotomy. Second look laparotomy tends to be the norm in these cases. Given the possibility of embolic, thrombotic, or non-occlusive mesenteric ischemia as the cause of bowel ischemia, attention to the abdominal vasculature is necessary. The distribution of the ischemia may help elucidate this as proximal jejunal sparing typically reflects and superior mesenteric artery (SMA) embolus whereas diffuse ischemia of the entire midgut (ampulla to distal transverse colon) suggests thrombosis. In cases requiring SMA exposure and exploration, revascularization should be pursued prior to bowel resection [4].

Asymptomatic patients with portal venous gas are more challenging from a diagnostic and management standpoint. In the absence of clear indications for laparotomy, asymptomatic patients can usually undergo conservative management with nil per os (NPO), antibiotics, nasogastric tube (NGT) decompression, as indicated, along with serial abdominal examination. Any concern for intestinal ischemia should prompt extensive workup, which may necessitate diagnostic laparoscopy or laparotomy to definitively rule out bowel ischemia.

References

1. Abboud B. Hepatic portal venous gas: physiopathology, etiology, prognosis and treatment. World J Gastroenterol WJG. 2009;15(29):3585.
2. McElvanna K. Hepatic portal venous gas—three non-fatal cases and review of the literature. Ulster Med J. 2012;81(2):74–8.
3. Nelson AL. Hepatic portal venous gas: the ABCs of management. Arch Surg. 2009;144 (6):575.
4. Peloponissios N. Hepatic portal gas in adults: review of the literature and presentation of a consecutive series of 11 cases. Arch Surg. 2003;138(12):1367–70.

Small Bowel Diverticulitis

20

Stefanie Haynes

Rare and usually asymptomatic, but be prepared when it rears its ugly head

Learning Objectives

1. The learner will be able to formulate a differential diagnosis for small bowel diverticulitis.
2. The learner will be able to describe the evaluation and management of small bowel diverticulitis based on anatomic location and symptomatology.
3. The learner will be able to diagnose and treat symptomatic and incidental Meckel's diverticula.

Case Scenario

A 67-year-old female presents to the Emergency Department with sudden onset, severe epigastric abdominal pain radiating to her back. Associated symptoms of subjective fevers, nausea and emesis are present. She has a past medical history of HTN, HLD, colonic diverticulosis and denies any surgical history. The patient does note vague intermittent symptoms of crampy abdominal pain, postprandial bloating and nausea for many years. Her vital signs are normal except for a tachycardia of 111. On physical examination, her abdomen is diffusely tender. Laboratory

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evaluation demonstrated WBC 23,000, BUN 20, and Cr 1.15. A CT scan of her abdomen and pelvis showed thickening of the wall of the duodenum, surrounding fat stranding and a small amount of extraluminal air. The patient was admitted to the medical surgical floor and treated nonoperatively with bowel rest, IV antibiotics and serial abdominal examinations for a contained duodenal diverticular perforation.

Epidemiology/Etiology/Pathophysiology

Diverticular disease of the small intestine is relatively uncommon. Due to its usual asymptomatic presentation, it is often an incidental finding or a diagnosis of exclusion. They are classified based on their location, whether they are congenital or acquired and whether they are true (consisting of all intestinal wall layers) or false diverticula (consisting of mucosa, submucosa and serosa). Forty-five percent of all small bowel diverticula are found in the duodenum, making it the most common location, followed by the ileum and then the jejunum. Less than 4% of small bowel diverticula cause symptoms with the incidence of diverticulitis being 0.3–2.5%.

Acquired diverticula develop from dysregulated motility of the intestinal smooth muscle, leading to an increase in intraluminal pressure and herniation at a weakening in the intestinal wall. Bacterial overgrowth and stasis of intestinal transit may lead to a similar pathophysiologic process. Duodenal diverticula are usually solitary in nature and found between the ages 50 and 75. They are most commonly located in the periampullary region, which is within 2 cm of the ampulla in the second portion of the duodenum at the mesenteric border. Jejunoileal diverticula are usually larger in size, multiple and found in the sixth and seventh decade of life (Fig. 20.1).

A Meckel's diverticulum is the most common true congenital malformation of the gastrointestinal tract. It is a result of the failure of complete obliteration of the omphalomesenteric duct during gestation. Inflammatory changes consistent with diverticulitis are present in 10–20% of those with a Meckel's diverticulum and can sometimes be clinically indistinguishable from appendicitis.

Differential Diagnosis

Due to the nonspecific presentation associated with small bowel diverticula and their associated complications, the differential includes but is not limited to pancreatitis, cholecystitis, cholangitis, peptic ulcer disease, gastroenteritis and bowel obstruction.

Diagnosis

The diagnosis of small bowel diverticular disease and subsequent development of diverticulitis is often challenging. As with all presentations, a thorough history and physical examination is essential. Signs and symptoms may include nausea, vomiting, abdominal pain, fever and chills. Radiologic studies including plain abdominal films and ultrasound may be helpful in excluding other differential diagnoses. A CT scan of the abdomen is the imaging modality of choice for small bowel diverticulitis and may demonstrate thickening of the bowel wall and surrounding fat inflammation. In the presence of a perforation, extraluminal air or the extravasation of contrast may be visualized. In cases which CT imaging is not able to identify the etiology of a patient's symptoms, additional studies include esophagogastroduodenoscopy, endoscopic retrograde cholangiopancreatography, fluoroscopic upper gastrointestinal swallow study, or small bowel follow-through may be utilized [1].

Complications

Small bowel diverticula may lead to complications consistent with inflammation, obstruction or hemorrhage. The incidence of these complications ranges from 6 to 10%. Inflammation consistent with diverticulitis may lead to perforation or bleeding, posteriorly into the retroperitoneal space and anteriorly into the peritoneal space or aorta. Mortality associated with complicated small bowel diverticulitis is reported to be as high as 40%.

Management

As with colonic diverticulitis, small bowel diverticulitis may be managed nonoperatively depending on the individual presentation of the patient. An uncomplicated episode of small bowel diverticulitis, including those with a contained perforation, may be treated with bowel rest, antibiotics, serial abdominal examinations and percutaneous drainage if an abscess is present. If the patient presents with hemodynamic instability, generalized peritonitis or failure of medical management, then operative intervention is required. The choice of surgical procedure will depend on the location of the diverticulum and intraoperative findings [2, 3].

Generally, operative intervention for symptomatic duodenal diverticula is reserved for cases not successfully treated with endoscopy (sphincterotomy and stent placement). A simple perforation with limited amount of inflammation may be amenable to a primary closure or diverticulectomy. This is performed after an extensive Kocher mobilization, if located in the duodenum, then primary closure (transversely or obliquely) in a one- or two-layer fashion with permanent suture and

drainage of the repair (Fig. 20.2). In situations where a large defect is encountered, a defunctionalized segment of jejunum may be used as a serosal (Thal) patch.

If the diverticulum involves the ampulla of Vater, extensive inflammatory changes are present or it is not anatomically amenable to resection, a diversion procedure is indicated. Diversion may be accomplished with a gastrojejunostomy and pyloric exclusion, Billroth II reconstruction or Roux-en-Y gastrojejunostomy. A segmental resection with primary anastomosis may be utilized for rare cases located in the third or fourth segments of the duodenum or for jejunoileal diverticula. A pancreaticoduodenectomy may also be considered if the inflammation is too severe for safe diversion or if the diverticulum is involving the common bile duct or pancreatic ducts.

Lastly, it is generally agreed that in an adult, incidentally discovered Meckel's diverticula ought to be left alone, whereas they are resected in a child. Relative indications for adult resections include palpable evidence of ectopic tissue, prior diverticulitis, hemorrhage, intussusception, or the presence of a mesodiverticular band.

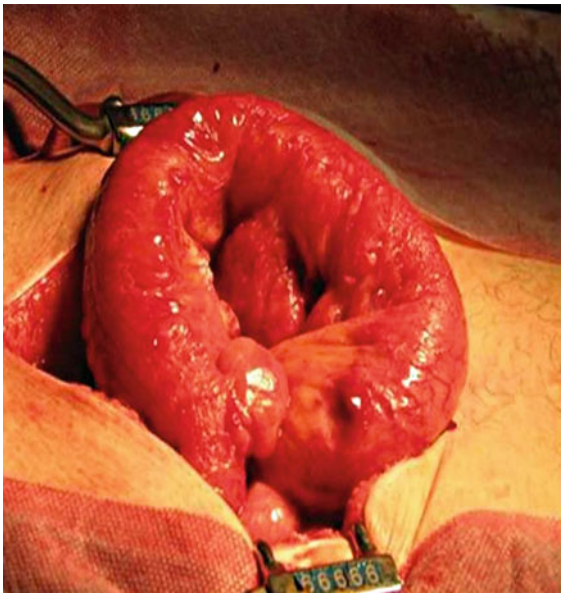

Fig. 20.1 Small bowel seen here with large jejunal diverticulum which tends to perforate into the mesentery. © Dale Dangleben, MD

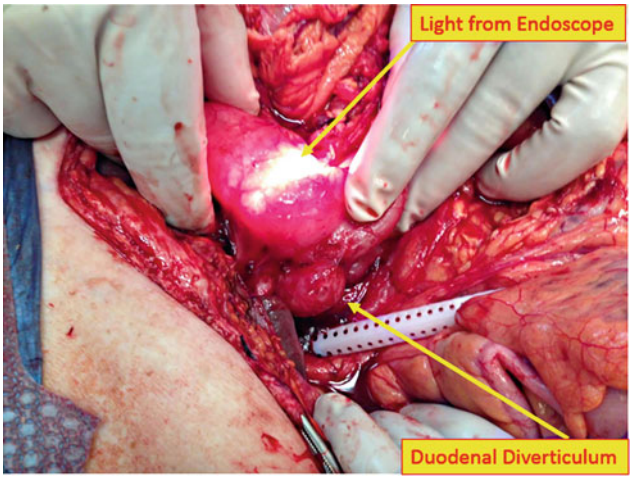

Fig. 20.2 Duodenal diverticulum. © Dale Dangleben, MD

Symptomatic Meckel's diverticulitis (presenting with bleeding, obstruction or inflammation) is treated with surgical management including segmental resection (**not** simple diverticulectomy as ectopic tissue is often present on the antimesenteric side) and primary ileoileostomy [4]. Appendectomy is generally performed concurrently in order to avoid diagnostic confusion in the future (Figs. 20.1 and 20.2).

References

1. Akhrass R, Yaffe MB, Fischer C, et al. Small-bowel diverticulosis: perceptions and reality. J Am Coll Surg. 1997;184(4):383–8.
2. Chiu EJ, Shyr YM, Su CH, et al. Diverticular disease of the small bowel. Hepatogastroenterology. 2000;47(31):181–4.
3. Kouraklis G, Glinavou A, Mantas D, et al. Clinical implications of small bowel diverticula. Imaj. 2002;4(6):431–3.
4. Zani A, Eaton S, Rees CM, et al. Incidentally detected Meckel diverticulum: to resect or not to resect? Ann Surg. 2008;247:276–81.

Small Bowel Obstruction

21

Firas G. Madbak

Predictably unpredictable

Learning Objectives

1. Review the various etiologies of small bowel obstruction.
2. Review the workup and evaluation of a patient with suspected small bowel obstruction.
3. Establish a systematic approach to managing small bowel obstruction.

Case Scenario

A 65-year-old female who underwent an uncomplicated hysterectomy 15 years ago presents to the emergency department with a two-day history of crampy abdominal pain, bilious vomiting and obstipation. On physical exam, her abdomen was distended, diffusely tender but nonperitoneal and had hyperactive bowel sounds. A nasogastric tube (NGT) was inserted and drained 1.5 L of bilious output. Laboratory evaluation showed a white blood cell count of 17,000, BUN of 40 and creatinine of 1.8. A plain film of the abdomen demonstrated multiple dilated loops of small bowel with air–fluid levels and no air in the rectum. A computed tomography (CT) scan of the abdomen and pelvis showed dilated small bowel loops with a transition point, the proximal ileum and no evidence of bowel ischemia.

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Two days of nonoperative treatment with NGT decompression, bowel rest, persistent high output and obstipation prompted for laparoscopic exploration and adhesiolysis. Postoperatively, the patient had bowel function return on POD 4 and was discharged home after tolerating diet and ambulating without difficulty on POD 6.

Epidemiology/Etiology/Pathophysiology

Small bowel obstruction (SBO) represents 15% of emergency department admissions for abdominal pain. Complete SBO occurs when the bowel lumen is obliterated, and partial SBO allows some restricted passage of contents distally. Closed-loop obstruction results when there is obliteration of bowel lumen at two points proximally and distally with rapid strangulation of the involved segment. Complete SBO is less likely to resolve with nonoperative management and is associated with a high risk of strangulation. The most common cause in developed countries is adhesions from previous abdominal surgery. Approximately 60–85% of cases are due to adhesions. It is also estimated that 34% of patients who have had a laparotomy will be admitted to the hospital within 10 years for adhesive disease. There are multiple risk factors largely dependent upon the original operation. About 80% of SBOs develop following pelvic and lower abdominal surgery (mainly colorectal and gynecologic operations). The extent of surgery also affects the density of adhesions. Previous history of admissions for SBO increases recurrence likelihood. Multiple operations are also associated with a greater number of adhesions. The need for operative intervention in adhesive obstructions ranges from 20 to 40%, but it is largely dependent on threshold for surgical intervention. Additionally, 20% of explorations for SBO reveal gangrenous bowel that requires resection. Recurrence rates tend to be lower in the long term for patients managed operatively although this finding is based on studies with significant selection bias. The recurrence rates of SBO range from 19 to 53% [1, 2].

Patients who develop SBO without prior history of abdominal surgery (virgin abdomen) classically have mechanical obstruction that requires urgent operation. Nonadhesive SBO is primarily due to hernias (10% of SBO, most commonly inguinal hernias) and masses (tumors). There is a high risk of strangulation associated with SBO secondary to hernias. Notably, the vast majority of pediatric SBO cases are due to hernias. Most neoplasms causing SBO are metastatic lesions or peritoneal carcinomatosis, whereas primary small bowel tumors are a less likely cause. Crohn's disease patients often present with obstruction due to phlegmon or stricture. Presentation is usually chronic and typically managed nonoperatively unless there is evidence of strangulation or perforation. Other less common causes include bezoars, radiation enteritis, foreign bodies, intussusception, congenital adhesions, gallstone ileus, SMA syndrome, diverticular disease, cystic fibrosis, abscess and volvulus.

Differential Diagnosis

Amongst the broad diagnostic considerations in the workup of acute abdominal pain, a distinction between small and large bowel obstruction is critical since the diagnosis, management and treatment are different.

Generally, biliary and pancreatic etiologies need to be considered along with inflammatory bowel disease, mesenteric ischemia, gastroenteritis, gynecologic causes such as ovarian torsion or endometriosis and most notably, a non-mechanical paralytic ileus picture.

Diagnosis

History should illicit previous abdominal operations, malignancy or prior SBO presentations. Emphasis on hernia evaluation and noting abdominal scars during physical examination is important. Laboratory studies may be normal but with disease progression may show leukocytosis, hemoconcentration and metabolic alkalosis with protracted vomiting. Lactic acidosis should raise suspicion for strangulation and ischemia. Radiologic studies will show characteristic air–fluid levels (preferentially on an upright film), no gas in the colon and dilated small bowel loops (>3 cm in diameter) (Fig. 21.1). CT scans help identify causes and possibility of bowel ischemia (suggestive signs are wall edema, pneumatosis, poor enhancement with contrast of the bowel wall and portal venous gas) (Fig. 21.2).

Complications

The most common complication of adhesiolysis is inadvertent bowel injury. Iatrogenic injury could potentially occur upon entry in the abdomen or during lysis of dense adhesions. Full-thickness enterotomies should be repaired or resected with attempts to minimize contamination. The necessity of repairing serosal tears remains debatable.

Complete obstruction or vascular occlusion may lead to ischemia and ultimately perforation with risks of acute intraabdominal sepsis which needs quick diagnosis and prompt treatment.

Management

Fluid resuscitation is the mainstay of initial therapy. Electrolyte abnormalities should be corrected. Unless there is evidence of active ischemia or strangulation, antibiotics are generally not indicated. Nasogastric tube decompression and bowel rest have traditionally been used to relieve overdistension due to swallowed air and

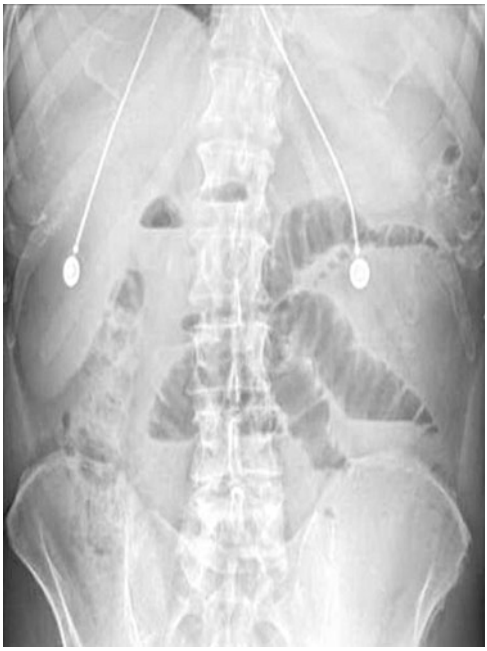

Fig. 21.1 Classic X-ray finding of SBO-dilated small bowel with air–fluid level within the bowel. © Dale Dangleben, MD

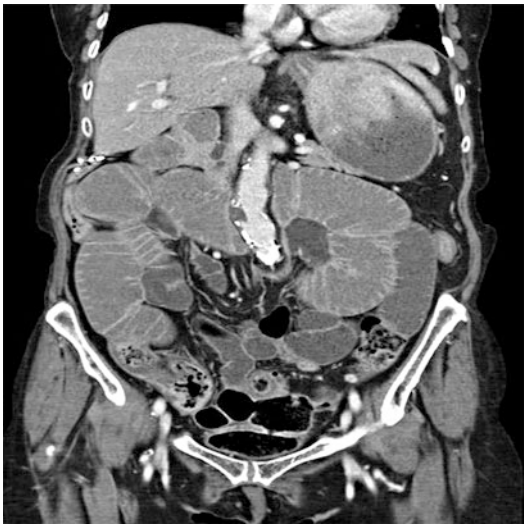

Fig. 21.2 CT scan with extensive small bowel dilatation and fluid filled consistently with a distal small bowel obstruction. © Dale Dangleben, MD

foregut secretions in all cases. More recently, patients with partial obstruction without nausea or vomiting have been nonoperatively managed with success without an NGT. Narcotic analgesia is used judiciously. In complete SBO, the classic teaching has been to operate early [3].

There is debate regarding the duration of nonoperative treatment of partial SBO until deciding to intervene surgically. Given the fact that 80% of adhesive bowel obstruction cases will resolve with a trial of nonoperative therapy, the decision making can be challenging. Recent retrospective studies have shown that 88% of cases will resolve within 48 h with the remaining cases resolving in 72 h. Most surgeons will wait 48–72 h before proceeding with operation. Others have advocated even longer times of up to five days before operating. Evidence of worsening exam or strangulation should prompt surgical intervention regardless of duration. Newer approaches to achieve early resolution have included upper gastrointestinal series with small bowel follow-through using water-soluble contrast if bowel obstruction is diagnosed previously on an unenhanced CT scan. Typically, surgery is indicated if no contrast is seen in the colon after 24 h. This protocol has been proven useful given the push to avoid the delays associated with CT scanning with oral contrast enhancement in emergency departments.

Goals of operation are lysis of adhesions and assessing bowel viability using visual inspection of color and peristalsis, Doppler or fluorescein examination. Nonviable bowel is resected with consideration of a second look operation (Figs. 21.3, 21.4, 21.5). Laparoscopy can be quite difficult but has been increasingly used with reasonable success.

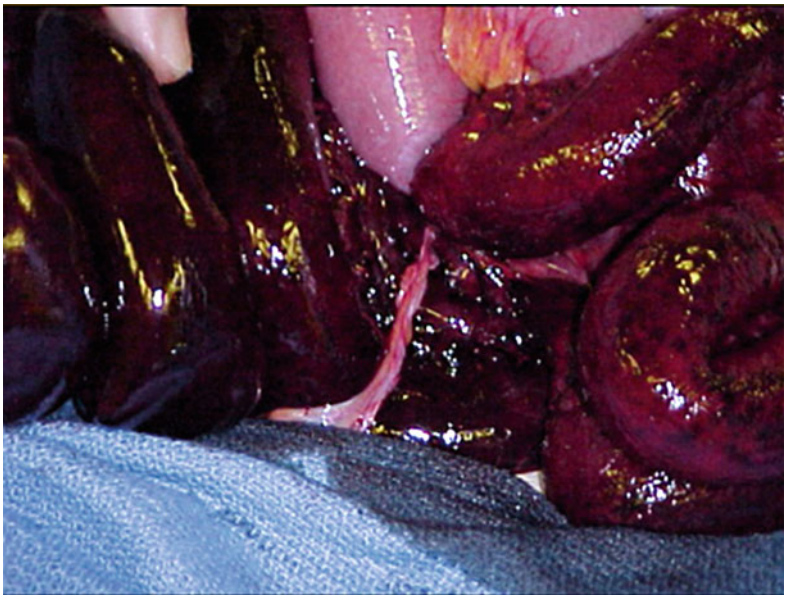

Fig. 21.3 Complete small bowel obstruction from an adhesive band resulting in strangulation. © Dale Dangleben, MD

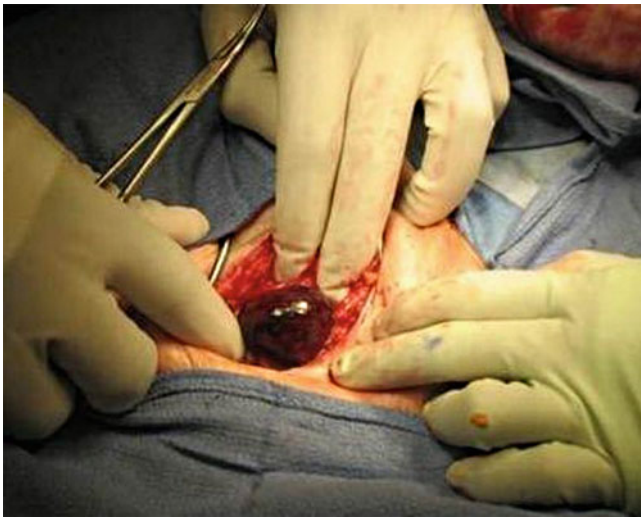

Fig. 21.4 A loop of strangulated small bowel is seen here in this strangulated inguinal hernia. The patient presented with excruciating groin pain and bowel obstruction. © Dale Dangleben, MD

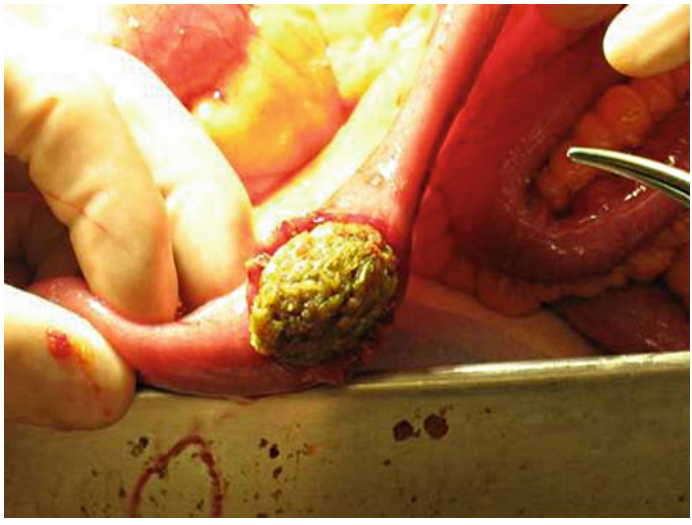

Fig. 21.5 This patient presented with a complete small bowel obstruction after eating large amounts of raw carrots, potatoes and celery. He was found to have a phytobezoar. © Dale Dangleben, MD

Hernias are associated with an increased risk of strangulation in the setting of SBO. Therefore, there is a low threshold for operative management. Unless a hernia can be reduced at the bedside with the aid of conscious sedation, patients with

evidence of incarceration or strangulation should undergo surgery. Similarly, bowel that is not viable is resected with a primary anastomosis and primary tissue hernia repair. In cases where there is bowel strangulation without necrosis, most surgeons will prefer not using prosthetic mesh for hernia repair; however, others believe it is acceptable. Alternatively, biologic mesh can be utilized with mitigated prosthetic mesh infections but with clear higher long-term recurrence rates and questionable durability.

References

1. Foster NM, McGory ML, Zingmond DS, et al. Small bowel obstruction: a population-based appraisal. J Am Coll Surg. 2006;203:170.
2. Kumar S, Wong PF, Leaper DJ. Intra-peritoneal prophylactic agents for pre venting adhesions and adhesive intestinal obstruction after non-gynaecological abdominal surgery. *Cochrane Database Syst Rev*. 2009:CD005080.
3. Loftus T, Moore F, VanZant E, et al. A protocol for the management of adhesive small bowel obstruction. J Trauma Acute Care Surg. 2015;78(1):13–9.

Small Bowel Perforation

22

David Lapham and James Giannone

The road to the operating room does not always pass through the CT scanner

Learning Objectives

1. The learner will recognize concerning signs and symptoms on the abdominal examination prompting surgical exploration.
2. The learner will understand the large spectrum of pathologies that can manifest as a small bowel perforation.
3. The learner will be able to describe the surgical options when presented with a small bowel perforation.
4. The learner can discuss any further workup or treatment necessary depending on the underlying cause of perforation.

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Case Scenario

A 56-year-old male presents to the emergency department with an acute onset of central abdominal pain. He has a history of metastatic colorectal cancer and has been receiving a chemotherapy regimen consisting of 5-fluorouracil, leucovorin, and oxaliplatin (FOLFOX) plus bevacizumab. He also has end-stage COPD for which he is on daily steroids. On examination, his vitals are normal with the exception of mild hypertension. His abdomen is diffusely rigid and tender with peritoneal signs. He has a mild leukocytosis and anemia on his laboratory studies. An obstruction series is performed, demonstrating air under the diaphragm. He is brought to the operating room for laparotomy and is found to have ulceration with perforation of the mid-jejunum. Following resection and primary anastomosis, the abdomen is copiously irrigated and the small bowel is run in its entirety. The patient's abdomen is closed and he is brought to the intensive care unit for postoperative care.

Epidemiology/Etiology/Pathophysiology

Perforation of the small bowel can be a manifestation of a myriad of diverse pathologies which can be broadly categorized as infectious, inflammatory, ischemic, medication related, neoplastic, foreign body related, or iatrogenic (Table 22.1). Regardless of etiology, the presentation of a perforated abdominal viscus causing peritonitis tends to be fairly uniform. Patients will usually complain of pain that began acutely and will increase in severity based on chronicity. Pertinent positives in a focused review of systems may demonstrate nausea, fever, and emesis. Leakage of enteric contents will initially irritate the peritoneum focally and subsequently promulgate to the entire abdomen if not promptly controlled. The physical examination findings will mirror this progression with focal tenderness to palpation and localized guarding and rebound tenderness, quickly turning to generalized, diffuse tenderness, involuntary guarding, and rigidity.

Differential Diagnosis

When a patient presents with acute onset of pain, diffuse tenderness to palpation and free air on imaging the diagnosis of a perforated small bowel is easily entertained; however, the underlying cause is often more elusive. Broad etiologies should be considered first. Bowel obstruction and inflammatory processes are the most common cause of small bowel perforation in developed countries, while infectious etiologies are the most common worldwide.

It is important to emphasize that oftentimes the precise cause may not be known, but this should not delay definitive therapy which usually includes prompt surgical intervention. Axiomatically, time should not be wasted obtaining other imaging and laboratory studies that will not change management.

Table 22.1 Differential diagnosis of small bowel perforation by category

Category	Specific causes
Trauma	Penetrating Blunt
Infection	Cytomegalovirus Salmonella Tuberculosis Ascaris lumbricoides Entameba histolytica
Obstruction	Adhesions Hernia Neoplasm Closed-loop obstruction
Medication related	NSAIDs Potassium chloride Steroids Chemotherapy Immune modulators (bevacizumab) (2)
Foreign body	Direct injury (sharp/long objects) Stercoral ulcer from impaction
Inflammatory	Crohn's disease Ulcerative colitis Celiac disease Graft versus host disease
Iatrogenic	Radiation enteritis Trocar insertion Inadvertent surgical enterotomy Endoscopic rupture
Ischemic	Thrombus Embolism Venous congestion Vascular diseases

Diagnosis

Because the natural history of intestinal perforation leads to peritonitis, it is not common to require extensive imaging before laparotomy. However, given the acute nature of most presentations of bowel perforation, there are instances where pre-operative imaging may be helpful. The erect chest X-ray radiograph is best to demonstrate free air under the diaphragm, which, in the absence of a secondary contributing cause, is an indication for surgical exploration (Fig. 22.1). Abdominal radiographs may show free air as well. A left lateral decubitus radiograph can be helpful to distinguish from any air that may be seen in the stomach and could be helpful if other films are equivocal. In obtunded or immobilized patients, a chest X-ray obtained in the reverse Trendelenburg position may closely resemble an upright projection. Many patients with non-specific abdominal pain may have

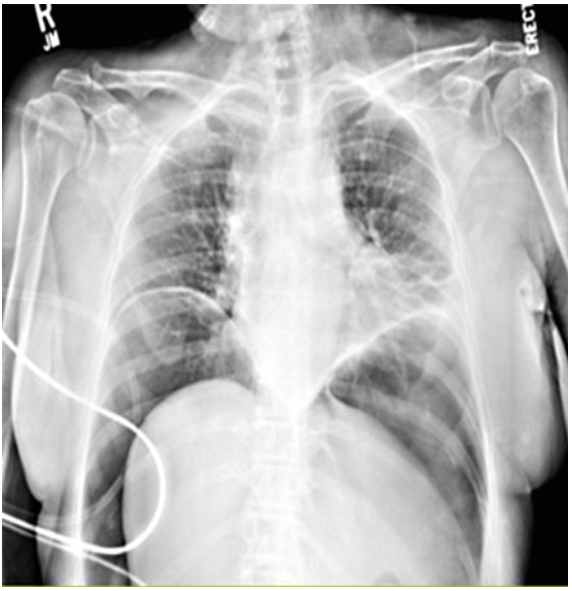

Fig. 22.1 Patient was having EGD and developed massive pneumoperitoneum during the intubation of the second part of the duodenum. © Dale Dangleben, MD

cross-sectional imaging of the abdomen and pelvis in the ED prior to surgical consultation, which may clearly yield a diagnosis of pneumoperitoneum. While the advantage of a preoperative CT scan is that it may also indicate the location or underlying pathology of the perforation, it is often unnecessary as it may waste valuable time. If the patient has received oral contrast, extraluminal contrast can be seen in addition to air. Depending on the acuity of the perforation, the patient may or may not have peritonitis. If peritoneal signs are present, they can aid in the confirmation of perforated viscus [1, 2].

Complications

Acute complications associated with a perforated bowel include sepsis, septic shock, and death. Once suspected, a true small bowel perforation mandates surgery. Long-term complications often manifest after the cause of the perforation is elucidated. Possible findings as outlined in Table 22.1 such as inflammatory bowel conditions, vascular insufficiency, or malignancy will require different long-term management after immediate control of the perforation.

Management

The principles of management of a perforated viscus focus on three tenets. First, one must identify the acuity of the patient and provide the necessary resuscitative and medical therapies in anticipation of operative exploration. Second, the operative approach to a perforated viscus is the same regardless of the location and starts with identification and source control. Third, the underlying pathology will dictate any further postoperative medical or surgical intervention.

The presentation of patients with a small bowel perforation can be placed on a wide spectrum of acuity. Patients may present with mild localized abdominal tenderness and imaging to support a small local process. Other patients may present in extremis with hemodynamic instability and abnormal laboratory studies.

Once in the operating room, thorough exploration of the abdomen is undertaken. Whether the exploration is approached as an open procedure or laparoscopically, the following items must be considered and successfully achieved. The abdomen and all quadrants must be fully explored as well as all solid organs. The entire bowel must be inspected from the ligament of Treitz to the rectum. Cultures should be taken if there is concerning fluid or material and biopsies should be sent as needed. Once the perforation is identified, the affected small bowel portion will require resection in almost all cases. The suspected etiology becomes imperative at this point. If, for example, the cause was blunt trauma, there may be no other abnormalities through the abdomen and all that is required is control of the bowel. If the cause of perforation was a complete small bowel obstruction, then correction of the obstruction is also required including possible lysis of adhesions, repair of hernias, or any of the other reasons on the long list of obstructive causes. Ingested foreign bodies require removal of the object and possibly another enterotomy to remove it from the bowel lumen. Mesenteric ischemia is particularly challenging because in addition to gaining source control of the perforation, vascular evaluation must also be entertained with angiography or embolectomy. As mentioned in Chap. 19, revascularization should be pursued prior to resection in these cases [3].

Once the bowel has been resected, the abdomen explored, and all other secondary causes accounted for, restoration of bowel continuity is considered (Figs. 22.2 and 22.3). Particularly in the setting of peritonitis and intraabdominal sepsis, it is advised to leave the bowel in discontinuity with a temporary abdominal closure with plans for a second-look operation. In other cases, diversion versus primary anastomosis can be performed depending on the factors including favorability of a tension-free anastomosis, blood supply, and overall breach of the physiological envelope and the patient's underlying disease and comorbidities. Based on the recent data, if the abdomen cannot be closed on the initial re-exploration, the editors favor diversion.

Postoperatively, the patient will undergo routine care, but further medical workup or surgical intervention will depend on the pathologic examination of collected specimen. If a diagnosis of inflammatory disease is made, which was the cause of perforation, medical treatment will need to be initiated for disease control.

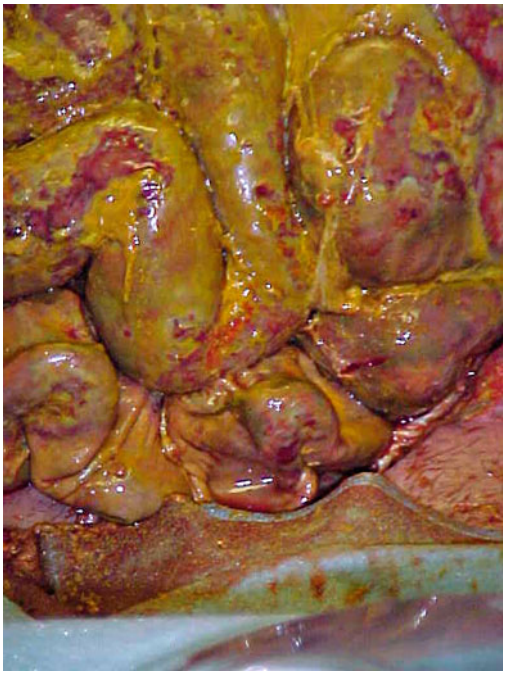

Fig. 22.2 Fecal peritonitis from the extensive contamination of small bowel content from perforation. © Dale Dangleben, MD

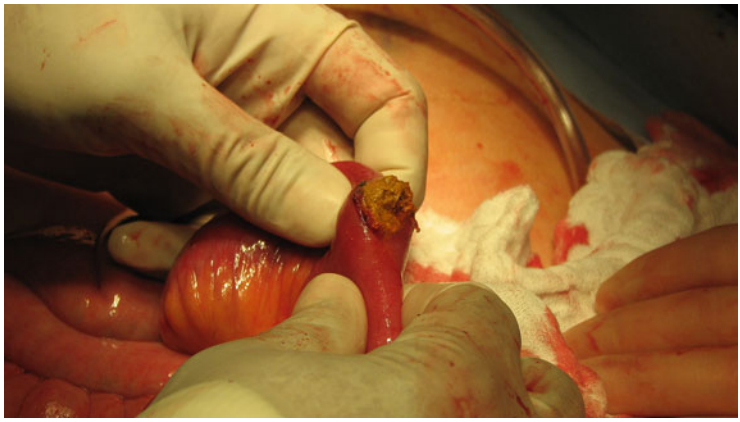

Fig. 22.3 Small bowel perforation with thick feculent material. © Dale Dangleben, MD

Malignancy, either primary or metastatic, is another finding that will require further workup and staging, possible chemotherapy, or additional surgery. If the etiology was found to be infectious, then antibiotics will need to be tailored accordingly.

The unfortunate surgeon will occasionally encounter a scenario where a perforation cannot be identified on exploration. In that situation, one of the editors has found it useful to flood the abdominal cavity and perform concurrent upper and/or lower endoscopy in the hope of a positive bubble test.

References

1. Brown C. Small bowel and colon perforation. Surg Clin N Am. 2014;94:471–5.
2. Brunicardi FC. Schwartz's principles of surgery. 10th ed. New York, USA: The McGraw-Hill Companies Inc.; 2010.
3. Freeman HJ. Spontaneous free perforation of the small intestine in adults. World J Gastroenterol. 2014;7:9990–7.

Toxic Megacolon

23

Aaron N. Sachs

Put your knife on hold, but not for long

Learning Objectives

1. Understand the causes of toxic megacolon and the seriousness of the condition.
2. Identify criteria utilized to diagnose toxic megacolon and the differential diagnosis.
3. Recognize that successful management requires a team-based approach.
4. Know the indications for surgical management in the disease process.
5. Learn the complications of toxic megacolon, including death.

Case Scenario

A 49-year-old man with a history of Crohn's disease presents to the hospital complaining of increasing abdominal pain in the setting of ongoing treatment for an acute colitis flare. On examination he has a temperature of 39.5 °C, heart rate of 132, and blood pressure of 89/40. He has diffuse abdominal tenderness and marked abdominal distention. Laboratory studies show a leukocytosis as well as evidence of acute kidney injury and hypokalemia. A plain abdominal radiograph demonstrates a dilated colon approximately 6.2 cm in diameter. He is admitted to the medical intensive care unit for nasogastric tube decompression and is started on IV antibiotics and high-dose steroids. General surgery is called to evaluate the patient

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since he continued to have worsening hypotension requiring vasopressor infusions as well as increasing abdominal pain and leukocytosis.

Epidemiology/Etiology/Pathophysiology

Toxic megacolon is a life-threatening condition that can complicate colitis due to virtually any pathology although it has been more commonly associated with inflammatory bowel disease most notably ulcerative colitis. It is defined as colonic distension (segmental or total) of at least 6 cm associated with signs of systemic toxicity and acute colitis. Causes are inflammatory (such as ulcerative colitis or Crohn's disease), infectious (which can be bacterial, viral, or parasitic), ischemic, or malignant. It is most commonly reported in inflammatory bowel disease and occurs in 1–5% of these patients during their lifetime. A mean of 3–5 years of active disease typically precedes the development of toxic megacolon. The most common infectious cause is pseudomembranous colitis, in which it complicates 0.4–3% of patients. Other than *Clostridium difficile*, other bacterial infectious agents include Salmonella, Shigella, Campylobacter, and Yersinia. Parasitic and viral causes include Cryptosporidium and cytomegalovirus, respectively [1, 2].

It is thought that factors related to release of inflammatory mediators and mucosal colonic damage extending into smooth muscle lead to toxic megacolon. Hypokalemia, antimotility agents (e.g., opiates and anticholinergics), colonoscopy, and enemas can exacerbate the problem. The colon dilates proximal to the diseased inflamed segment, and the dilated colon is not actually the toxic segment nor a cause of the syndrome making toxic megacolon a confusing term. This massive colonic distension causes a functional obstruction to ensue. The inflamed, thickened colon becomes vulnerable to perforation, most commonly at the splenic flexure.

Differential Diagnosis

The differential diagnosis of toxic megacolon is broad, essentially including any colon obstruction or gastrointestinal illness. It is distinguished from colonic pseudo obstruction and Hirschsprung's disease by the presence of systemic toxicity. Gastroenteritis/ileus, diverticulitis, stricture, ischemic colitis, and Crohn's/ulcerative colitis exacerbation are also in the differential. A mechanical large bowel obstruction such as cancer or volvulus should be excluded.

Diagnosis

Toxic megacolon should be suspected in patients presenting with abdominal distension, diarrhea, and a significant systemic inflammatory response. Typically, is it preceded or accompanied by fever, chills, abdominal pain, or bloody diarrhea.

On examination, patients are tachycardic, hypotensive, and have a distended, tender abdomen. A history of previous bouts of inflammatory bowel disease, foreign travel, immunosuppression, antibiotics, or chemotherapy may be present. Jalan et al. established criteria for diagnosis, in which any three of the following must be present: fever of 101.5 °F (or 38.6 °C), tachycardia greater than 120, or leukocytosis 10.5 K or greater. If one of the above is present with dehydration, altered mental status, electrolyte disturbances, or hypotension, toxic megacolon is likely. A thorough history and physical is therefore paramount to accurate diagnosis.

Abdominal radiographs help solidify the diagnosis and follow course in the disease process. A dilation of the ascending or transverse colon ranging from 6 to 15 cm may be seen. Perforation may also be seen as free air. The rate of colonic expansion and clinical condition of the patient are more important than the actual size of the colon seen on imaging. CT can confirm the diagnosis as well as exclude pneumatosis or pneumoperitoneum (Fig. 23.1). One can also exclude an obstructing cancer or diverticular stricture with CT.

Important laboratories to obtain include a complete blood count and electrolyte panel. Stool samples to exclude infectious etiologies such as *C. difficile* and blood cultures to exclude bacteremia should be considered. Serum lactate should be obtained. Limited endoscopy can help assist in the diagnosis, though it is important to avoid complete colonoscopy due to the risk of perforation. Pseudomembranous may be seen, and biopsy can show CMV inclusion bodies.

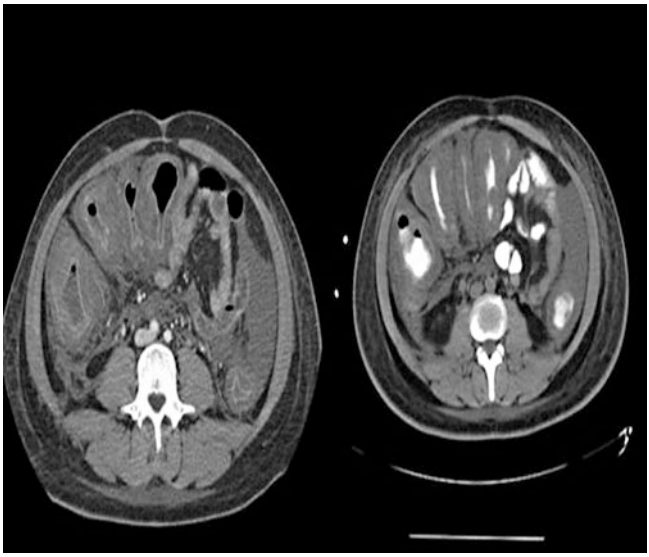

Fig. 23.1 A patient with fulminant *C. difficile* colitis developed toxic megacolon with marked thickening of the colonic wall. © Dale Dangleben, MD

Complications

Toxic megacolon is a serious disease. Complications include sepsis, septic shock, multi-organ failure, GI bleeding, colon perforation, and death. The mortality rate is about 20%, and slightly higher (in the range of 35–60%) with fulminant *C. difficile* colitis. A white blood cell count higher than 50 K and lactate higher than 5 are predictors of mortality. Depending on the extent of operative intervention, wound infection, staple line dehiscence, and ostomy complications are the main perioperative risks in patients undergoing surgical therapy.

Management

Successful management of toxic megacolon requires a multi-disciplinary approach from medical and surgical teams. Early surgical consultation is crucial, though an aggressive initial attempt at medical management is appropriate. Typically, these patients should be admitted to an intensive care unit. Bowel rest, nasogastric decompression, resuscitation, and electrolyte repletion are key starting points. Frequent serial abdominal examinations, laboratory studies, and radiographs are used to follow their clinical course. Broad spectrum antibiotics are initiated to reduce septic complications. If there is a history of inflammatory bowel disease, high-dose hydrocortisone can be given and does not result in higher risk of perforation. If *C. difficile* is present, discontinuing the offending antibiotic and administering rectal vancomycin (500 mg in 100 mL saline as retention enema every 6 h) as the oral route (500 mg four times daily) may be ineffective in most patients and with intravenous metronidazole (500 mg three times daily) is appropriate. Any antimotility agents, anticholinergics, and opiates should also be discontinued. Medical therapy is successful in 50–75% of patients.

Free perforation, massive GI bleeding, or continued progression of colonic dilation is operative indications, while failure to improve within 48 h is a relative indication. Some consider up to a week of medical management if there are no hard indications for operation. However, if perforation occurs, the mortality rate increases significantly. Subtotal colectomy with end ileostomy is the treatment of choice. Enterostomal therapists should be involved early. Mechanical bowel preparation should be avoided [3, 4].

The operation is approached with a midline incision. The colon should be handled gently to avoid perforation intraoperatively (Figs. 23.2 and 23.3). The hepatic and splenic flexures are mobilized, and the rectum is divided as low as possible. A suture is used to mark the staple line for future ileostomy takedown and restoration of bowel continuity. Drainage of the closed rectal stump with a mushroom type catheter is recommended. No intraperitoneal drain is necessary.

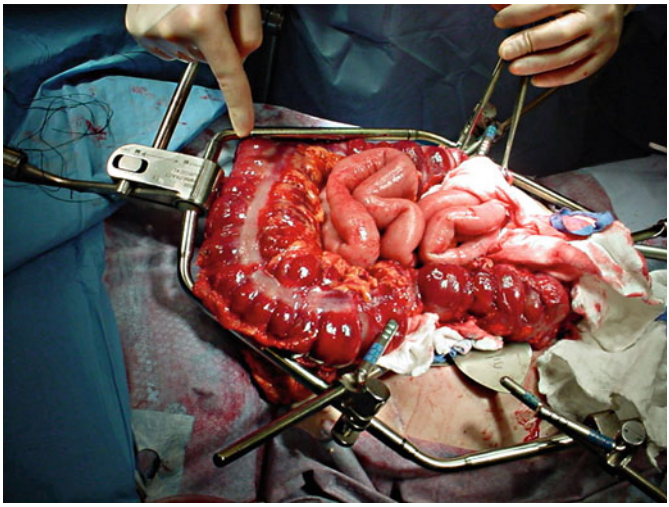

Fig. 23.2 **Toxic** megacolon with "beefy red" colon and normal appearing small intestines. © Dale Dangleben, MD

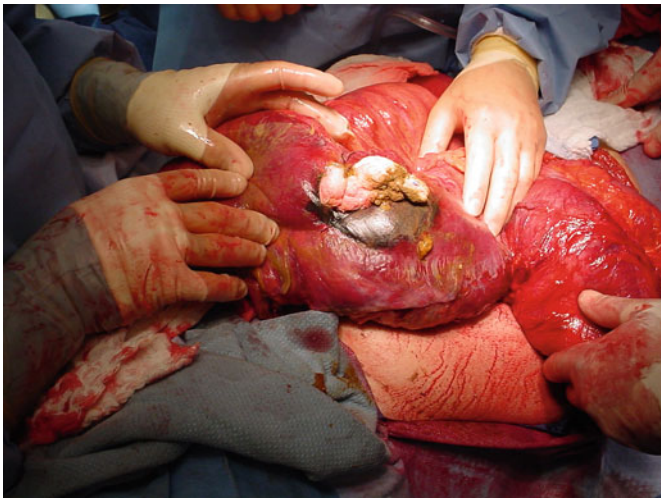

Fig. 23.3 Toxic megacolon with perforation. © Dale Dangleben, MD

Intravenous steroids should be weaned to the patient's preoperative dose over several days. In the hemodynamically unstable patient, it may occasionally be necessary to perform an abbreviated damage control laparotomy and leave the patient in discontinuity and an open abdomen until the intra-abdominal sepsis can be controlled.

References

1. Cameron JL, Cameron AM. The management of toxic megacolon. In: Current surgical therapy. 11th ed. Philadelphia: Elsevier Saunders; 2014.
2. Dimick JB, Upchurch GR, Sonneday CJ. Fulminant *Clostridium difficile* colitis. In: Clinical scenarios in surgery: decision making and operative technique. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2012.
3. Jalan K, Sircus W, Card WI, et al. An experience of ulcerative colitis: toxic dilatation in 55 cases. Gastroenterology. 1969;57:68–82.
4. Strong S. Toxic colitis and toxic megacolon. Epocrates Online. Epocrates, Inc.; 2015. Web.

Acute Pancreatitis

24

Syrell J. Rodriguez Carreras and Christie Hirsch-Reilly

Buckle up and just ride out the storm

Learning Objectives

1. Be able to diagnose acute pancreatitis and severe acute pancreatitis.
2. Identify the different etiologies that cause acute pancreatitis.
3. Recognize complications associated with acute pancreatitis and their management.
4. Be able to recognize and diagnose chronic pancreatitis.
5. Recognize the etiologies of chronic pancreatitis.
6. Recognize complications of chronic pancreatitis and how to manage them.
7. Become familiar with the surgical procedures and their indications for the treatment of chronic pancreatitis.

Case Scenario

A 45-year-old male presents to the emergency department complaining of severe abdominal pain, nausea, and vomiting. His medical history is unremarkable, and he denies a surgical history. He strongly denies alcohol use and does not take daily

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medications. His family history is only remarkable for cardiac disease. On physical examination, he has mid-epigastric tenderness to palpation. His laboratory studies reveal a leukocytosis, an elevated lipase, but a normal hepatic function panel. He is diagnosed with acute pancreatitis.

He is admitted, made NPO, and receives fluid resuscitation. His triglyceride levels come back as over 1000 mg/Dl, and he is diagnosed with hyperlipidemia. He is treated appropriately and discharged. However, 2 weeks later, he is brought by ambulance with altered mental status, hyperthermia, tachycardia, and tachypnea. A CT scan of his abdomen reveals findings associated with pancreatic necrosis. He undergoes percutaneous drainage successfully. He recuperates and is discharged with fenofibrate and high-dose lovastatin to prevent future pancreatitis episodes.

Epidemiology/Etiology/Pathophysiology

Acute Pancreatitis

Acute pancreatitis is defined as inflammation of the pancreas caused by a wide range of different etiologies, with gallstones and alcohol consumption being the two most common causes in the USA. The third most common etiology is iatrogenic, namely undergoing ERCP. Other less common etiologies include hyperlipidemia, hypercalcemia, hereditary, autoimmune, malignancy, medications, scorpion sting, and lastly idiopathic. Medications that have been implicated in the pathophysiology of pancreatitis include thiazide diuretics, azathioprine, estrogen, valproic acid, acetylcholinesterase inhibitors, furosemide, L-asparaginase, 6-mercaptopurine, methyldopa, sulfonamides, propofol, tetracycline, pentamidine, procainamide, dideoxyinosine, and nitrofurantoin.

It is believed that the pathophysiology of pancreatitis is due to the activation of digestive zymogens located inside acinar cells after an insult. This leads to acinar cell injury and the activation of inflammation, including inflammatory cells and cytokine activation and recruitment.

Chronic Pancreatitis

Chronic pancreatitis is continued inflammation of the pancreas resulting in permanent structural damage. Multiple etiologies have been implicated, the most common one being alcohol abuse. Additional etiologies include genetics (PRSS1 mutation), duct obstruction secondary to trauma, gallstones and malignancies, repeated attacks of acute pancreatitis, hyperlipidemia, hypercalcemia/hyperparathyroidism, autoimmune (IgG4 and associated autoimmune disease), and idiopathic. A good way to remember these is the classification system TIGAR-O, which stands for toxic-metabolic, idiopathic, genetic, autoimmune, recurrent and severe acute pancreatitis and obstructive.

Differential Diagnosis

Acute Pancreatitis—Acute mesenteric ischemia, cholangitis, cholecystitis, colon cancer, colonic obstruction, chronic pancreatitis, gastric cancer, pancreatic cancer, peptic ulcer disease, pancreatic pseudocyst, irritable bowel syndrome, viral hepatitis, bacterial pneumonia, acute respiratory distress syndrome, visceral perforation, and ruptured abdominal aortic aneurysm.

Chronic Pancreatitis—Pancreatic cancer, chronic gastritis, cholangitis, peptic ulcer disease, Crohn disease, intestinal perforation, and chronic mesenteric ischemia.

Diagnosis

Acute Pancreatitis

Commonly presents as epigastric pain accompanied by nausea and vomiting. Severe acute pancreatitis is associated with organ failure and typically presents with depressed mental status, tachycardia, tachypnea, and hypoxia.

Diagnosing acute pancreatitis depends on the fulfillment of the following two out of three criteria: elevation of lipase or amylase three times its normal level, characteristic abdominal pain, and/or imaging findings characteristic of acute pancreatitis.

Ranson's criteria have been classically used to identify patients at risk of severe acute pancreatitis although other scoring systems like the APACHE II and BISAP can be found on the literature. Ranson's criteria include age, white blood cell count, glucose, LDH, and ALT measurements at admission. Also, the measurement of a fall in hematocrit, fluid sequestration, hypocalcemia, PaO_2, increase in BUN and base deficit is performed in 48 h. A score equal or greater than 3 indicates severe acute pancreatitis.

Laboratory tests include lipase and amylase (lipase being more specific) as well as liver function tests, chemistries, complete blood count, and lipid panels. Imaging tests include abdominal ultrasound and CT scan [1].

Chronic Pancreatitis

The most common presenting symptom in chronic pancreatitis is abdominal pain followed by anorexia. As the disease progresses, patients will experience endocrine and exocrine function impairment, for which steatorrhea may be the first presenting symptom. Other common findings include biliary strictures and pseudocysts, pseudoaneurysms, duodenal obstruction, mesenteric venous thrombosis, and malignancy (both pancreatic and extrapancreatic).

Abdominal ultrasound is commonly the first imaging study obtained. The pancreatic changes observed are dilated pancreatic ducts, heterogeneous texture, cystic changes, and intraductal filling defects. Intraductal calcification is seen on plan abdominal films, and it is considered pathognomonic for the disease. Other imaging tests include CT, MRI, MRCP (magnetic resonance cholangiopancreatography) and EUS (endoscopic ultrasound), in which characteristic beading of the pancreatic duct (representing the calcifications mentioned above) will be seen. ERCP has also been used for diagnosis but its invasiveness makes it more of a therapeutic choice for managing ductal stones and strictures. However, it is considered the gold standard for diagnosis and staging.

The classification of chronic pancreatitis, from normal to severe, can be classified by Cambridge class, ranging from normal to severe depending on main pancreatic duct and on the presence and number of abnormal side branches.

Management

Acute Pancreatitis

Aggressive fluid resuscitation, NPO status, pain control, and identification and treatment of its cause are the mainstay of treatment. However, patients suffering from severe acute pancreatitis benefit from early institution of artificial nutrition (enteral) as it has been associated with decreased inflammation and the decrease in sequelae associated with this disease severity.

Complications of acute pancreatitis include pancreatic fluid collections and pancreatic necrosis. If peripancreatic fluid collections persist for more than 2 weeks and are accompanied by symptoms of infection, fluid should be drained and antibiotics given if its analysis reveals infection. Pancreatic necrosis is found in patients suffering from severe acute pancreatitis. CT scan is helpful to diagnose infected pancreatic necrosis as it can reveal gas in the cavity. Infected pancreatic necrosis should be aspirated and interventions should be undertaken when a clear demarcation between the devitalized and the normal tissues is observed. Available interventions include percutaneous drainage, endoscopic necrosectomy, video-assisted retroperitoneal debridement, laparoscopic necrosectomy, and open necrosectomy, with the minimally invasive procedures being the first choice [2].

Chronic Pancreatitis

The main priority in management of chronic pancreatitis is pain control and the reduction of progressive inflammatory changes, meaning discontinuing alcohol and tobacco consumption and treating any factors that exacerbate the disease. Also, supplementation of pancreatic enzymes should be used in patients suffering from malabsorption. Oral CCK (cholecystokinin) inhibitors have been proposed to

decrease the frequency of attacks, slow the progression, and improve pain. However, data are lacking regarding this intervention.

Complications of chronic pancreatitis include risk of pancreatic carcinoma, pseudocyst development (most common), pancreatic ascites, pancreatic enteric fistula, mass at head of pancreas, and splenic and portal vein thrombosis.

Endoscopic treatment for pain control consists of stricture dilation, stone removal and pancreatic duct stenting. It is considered a temporary solution as pain typically recurs and it is mainly used for patients suffering from multiple comorbidities [3].

Surgical treatment is indicated for management of biliary or duodenal obstruction, pseudocysts, and pain management. A variety of surgical treatments are available and include drainage, resection, combination of drainage and resection and denervation. Often, a drainage procedure such as the Puestow procedure is indicated if the duct diameter is greater than 7 mm. If the head of pancreas is greater than 4 cm, resection is indicated and procedures include the Whipple or its pylorus-preserving variant. If both the diameter and the head of the pancreas are enlarged, then a procedure that combines the two is indicated. Combined drainage and resection procedures include the Beger procedure (pancreatic head resection with preservation of duodenum) and the Frey operation (resection of pancreatic head with lateral pancreaticojejunostomy). Last but not least, thoracoscopic splanchnicectomy is a denervation procedure typically utilized for patient that typically does not have surgical targets.

References

1. Brunicardi FC, et al. Schwartz's principles of surgery. 10th ed. New York: McGraw-Hill Education; 2015.
2. Cameron J, Cameron AM. Current surgical therapy. 11th ed. Philadelphia: Elsevier Saunders; 2013.
3. Gardner TB, Anand BS. Acute pancreatitis. Medscape. 2015. http://emedicine.medscape.com/article/181364-overview. Accessed Jan 2016.
4. Huffman JL, Anand BS. Chronic pancreatitis. Medscape. 2015. http://emedicine.medscape.com/article/181554-overview. Accessed Jan 2016.
5. Mulholland MW, et al. Greenfield's surgery: scientific principles and practice. 5th ed. Philadelphia: LWW; 2010.

Peptic Ulcer Disease

25

Jordan M. Kirsch and Christie Hirsch-Reilly

Don't close it, patch it

Learning Objectives

1. The learner will be able to formulate a differential diagnosis for a patient presenting with peptic ulcer disease.
2. The learner will be able to use the Modified Johnson Classification System for gastric ulcers.
3. The learner will be able to formulate a management plan for a patient with peptic ulcer disease.

Case Scenario

A 43-year-old male presents to the emergency department with 3 h of sudden onset abdominal pain which began in the epigastrium. He reports a history of episodic epigastric pain, which occurs several hours after eating for the past 6 months. He has a 20 pack-year history and does not routinely see a physician. Laboratory studies show a WBC of 17,000. An upright chest X-ray demonstrates

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pneumoperitoneum after which he is taken to the operating room for an exploratory laparotomy. A 0.5 cm perforated ulcer on the anterior surface of the first portion of the duodenum is found. The defect is repaired with a Graham omental patch. The patient recovers uneventful and is discharged on omeprazole on postoperative day 6.

Epidemiology/Etiology/Pathophysiology

Peptic ulcer disease, one of the most common gastrointestinal diseases in the USA, has a prevalence of approximately 2%. It is most commonly seen in men with a peak incidence between 55 and 65 years of age. Risk factors include *Helicobacter pylori* infection (in 80–90% of both gastric and duodenal ulcers), NSAID use, smoking and cocaine use. Less common causes include Zollinger-Ellison Syndrome (gastrinoma); antral G-cell hyperfunction or hyperplasia, trauma, burns, and major physiologic stress. Peptic ulcers occur when there is an acid oversecretion affecting the gastroduodenal mucosal barrier.

Gastric Ulcer Classification

Type I ulcers are located on the lesser curvature and are the most common comprising approximately 60% of all gastric ulcers. Type II is a combined gastric body ulcer near the incisura and a duodenal ulcer. Type III ulcer are prepyloric, and Type IV ulcers are located high on the lesser curvature near the gastroesophageal junction. Type V ulcers are associated with NSAID use and may be at any location. Both Type II and Type III ulcers are associated with acid hypersecretion.

Differential Diagnosis

Differential diagnosis includes gastritis, duodenitis, cholelithiasis, cholecystitis, and pancreatitis.

Diagnosis

Upper endoscopy is the mainstay for diagnosing peptic ulcer disease in the inpatient setting. The gold standard test is the histologic examination of a mucosal biopsy using special stains. Thus, all gastric ulcers should be biopsied at the time of operation or endoscopy. Other tests for *H. pylori* infection include a serologic test (unreliable if the patient has already been treated with antibiotics for *H. pylori* and cannot be used to confirm disease eradication because the serum will remain

positive for an indeterminate length of time), a urea breath test, a rapid urease test, and the quick fecal antigen test. Mainly used to confirm disease eradication, the urea breath test is a newer modality and involves the ingestion of radio-labeled urea with subsequent analysis of expired air. The rapid urease test requires a biopsy specimen and is a simple laboratory test but has a high false-negative rate (especially after PPIs or antibiotics have been given). The fecal antigen test that detects active infection is a very simple test and can be used for diagnosis or confirmation of cure. It can be used preoperatively particularly in the emergent setting if the etiology of ulcer disease is unclear.

The acute care surgeon is often called upon to address emergencies associated with peptic ulcer disease, namely bleeding or perforation.

As with other acute care surgical problems, resuscitation and stabilization should begin without delay as a treatment modality is being planned. In bleeding patients, if transfusion requirements have escalated or the patient has continued hemodynamic instability despite less invasive endoscopic or radiographic interventions then surgery is indicated. Based on endoscopic findings, a validated scored system such as the Forrest Classification can be used to predict the risk of rebleeding should an operation not be emergently required [1–3].

For perforated ulcers, patients will present with peritoneal signs on exam and signs of sepsis with a fever, leukocytosis and abdominal imaging showing free air suggestive of a perforated viscus.

Complications

Bleeding Ulcers

Bleeding is the most common complication of peptic ulcer disease requiring hospitalization, and bleeding peptic ulcers are the most common cause of upper gastrointestinal bleeding overall. The vast majority of these cases can be treated nonoperatively with endoscopy and medical management.

Perforated Ulcers

Perforation occurs in a minority of patients requiring hospitalization for peptic ulcer disease; however, nearly all of these patients require operative intervention.

Gastric Outlet Obstruction

Gastric outlet obstruction is the least common of the major sequelae of peptic ulcer disease and is mostly associated with duodenal and pyloric channel ulcers.

Management

In the elective setting, patients who have never been treated for *H. pylori* and those whose ulcers result from NSAID use do not need to undergo acid reducing procedures. However, for patients who failed medical therapy (with evidence of nonhealing after 12 weeks), an acid reducing procedure is required at the time of operation.

The management of peptic ulcer disease has changed drastically over the past several decades. With the advent of proton pump inhibitors and therapy for the eradication of *H. pylori*, surgical intervention is only required in the most refractory cases and in those patients who experience complications of the disease which are discussed below. In these refractory cases, more uncommon causes of ulcer disease including gastrinoma must be considered. In general, malignancy must always be considered in gastric ulcers (making biopsy mandatory) but is exceedingly rare in duodenal ulcers. Locations near the greater curve have the highest propensity for malignancy.

Bleeding Ulcers

Initial therapy as with all upper GI bleeding should begin with endoscopy. If bleeding continues, angioembolization may be attempted if interventional radiology capability is available although significant collateralization makes this modality less successful for control when compared to lower GI bleeding. Bleeding gastric ulcers are often self-limited yet operative repair may be required in refractory cases (Figs. 25.1 and 25.2). Especially in emergencies where preoperative localization is not possible, gastric ulcers are exposed with an anterior gastrotomy and the ulcer is oversewn.

For bleeding duodenal ulcers, longitudinal duodenotomy should be performed and the ulcer be oversewn with ligation of the gastroduodenal artery if involved. Classically, this is performed with a "U-stich" where sutures (heavy silk) are placed on the proximal, superior, and inferior (3, 6 and 12 o'clock) but not distal borders of the ulcer to avoid potential injury to the ampulla of Vater. If truncal vagotomy is performed, the incision can then be closed as a pyloroplasty. Postoperative treatment for the eradication of *H. pylori* is essential as rebleeding rates are approximately 7 times higher in those not treated. For gastric ulcers, biopsies should be taken to rule out malignancy and evaluate for *H. pylori.*

Perforated Ulcers

In selected nonseptic, nonperitoneal patients, nonoperative management is appropriate if perforation is contained without free extravasation of contrast on enhanced imaging. Rapid exploration and repair in the operating room is the cornerstone of treatment for patients who present critically ill with pneumoperitoneum. Even with

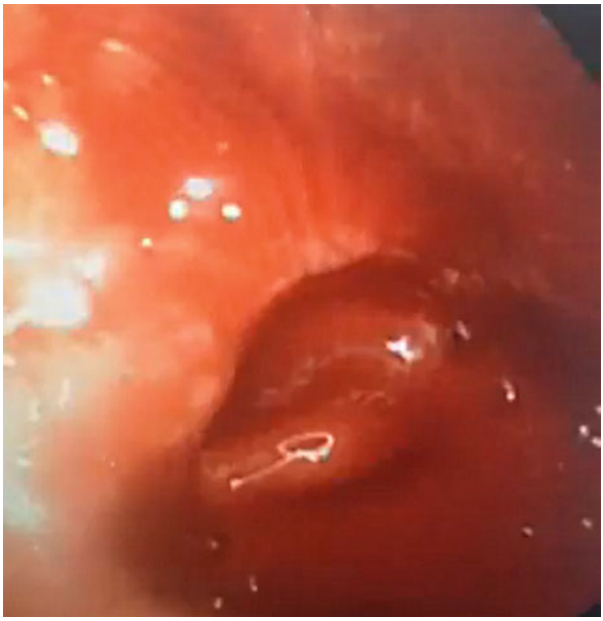

Fig. 25.1 Active bleeding from the base of an ulcer. © Dale Dangleben, MD

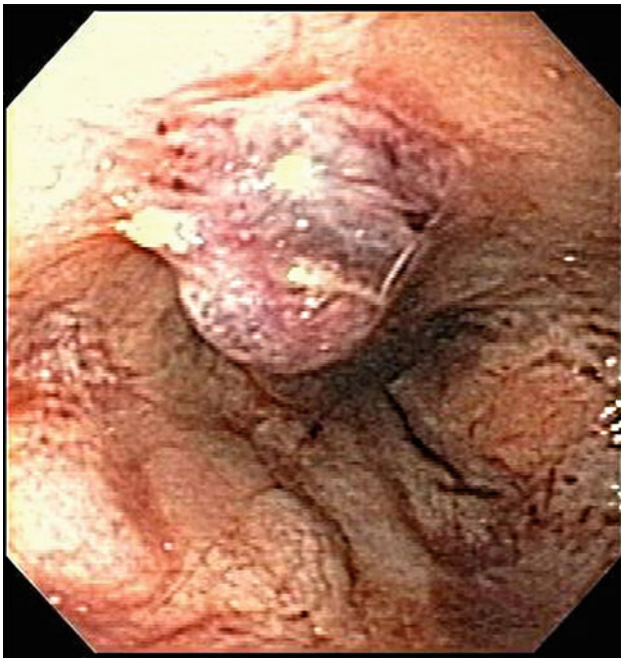

Fig. 25.2 Blood clot overlying an ulcer. © Dale Dangleben, MD

prompt care, mortality reaches 15%. While the open approach remains the gold standard, laparoscopy has been described for omental patching and vagotomy procedure with good outcomes. Limited studies are available as this continues to be an evolving technique. In patients with profound shock or hemodynamic instability, open repair is required. For most small (<1 cm) duodenal ulcers patching, the defect with omentum without primary closure is satisfactory. Although some surgeons prefer primary closure and omentoplasty, the editors do not favor primary closure given the edema and friability of the ulcer's edges. The same technique may be used in small gastric ulcers if the suspicion for malignancy is low or if the patient is too unstable to tolerate a more complex repair. Gastric ulcers should always be biopsied intraoperatively. For larger or more suspicious lesions, a distal gastrectomy including the ulcer bed should be performed. Ideally, a Billroth I (antrectomy with gastroduodenostomy) reconstruction is preferred but a Billroth II (antrectomy with gastrojejunostomy) may be required depending on condition of the duodenal stump and the extent of the gastrectomy.

Gastric Outlet Obstruction

In the acute setting, nasogastric decompression should be done first with corrections of electrolyte imbalances followed by endoscopy. Endoscopic pneumatic balloon dilation or stenting may offer temporary relief, but definitive surgical treatment is the mainstay. An antrectomy is the procedure of choice in chronic gastric outlet obstruction.

References

1. Brunicardi FR, et al. Schwartz's principles of surgery. New York, NY: McGraw-Hill; 2009.
2. Cameron J, Cameron AM. Current surgical therapy. 11th ed. Philadelphia: Elsevier Saunders; 2013.
3. Heldwein W, Schreiner J, Pedrazzoli J, Lehnert P. Is the forrest classification a useful tool for planning endoscopic therapy of bleeding peptic ulcers? Endoscopy. 1989;21(6):258–62.

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