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patients, increased fiber intake and the use of osmotic agents such as PEG may achieve satisfactory results. For patients with more severe constipation, a chloride channel opener (lubiprostone) or GC-C agonist (linaclotide or plecanatide) or NHE3 inhibitor (tenapanor) may be considered. For IBS patients with predominant gas and bloating, a low FODMAP diet may provide significant relief. Some patients may benefit from probiotics and rifaximin treatment. A small proportion of IBS patients have severe and refractory symptoms, are usually seen in referral centers, and frequently have constant pain and psychosocial difficulties. This group of patients is best managed with neuromodulators and other psychological treatments (Table 338-4). Clinical trials demonstrating success of a low FODMAP diet in improving IBS symptoms and quality of life provide strong evidence supporting the use of this dietary approach in the treatment of IBS. These observations, if confirmed, may lead to the use of the low FODMAP diet as the first line of treatment of IBS patients with moderate to severe symptoms.

■ ■ FURTHER READING Chang L et al: AGA Clinical Practice Guideline on the pharmacological management of irritable bowel syndrome with constipation. *Gastroenterology* 163:118, 2022. Dionne J et al: A systematic review and meta-analysis evaluating the efficacy of a gluten-free diet and a low FODMAP diet in treating symptoms of irritable bowel syndrome. *Am J Gastroenterol* 113:1290, 2018. Drossman DA: Functional gastrointestinal disorders: History, pathophysiology, clinical features, and Rome IV. *Gastroenterology* 150:1262, 2016. Lembo A et al: AGA Clinical Practice Guideline on the pharmacological management of irritable bowel syndrome with diarrhea. *Gastroenterology* 163:137, 2022. Mayer EA et al: Brain-gut microbiome interactions and functional bowel disorders. *Gastroenterology* 146:1500, 2014. Pittayanon R et al: Gut microbiota in patients with irritable bowel syndrome: A systematic review. *Gastroenterology* 157:97, 2019. Zhou SY et al: FODMAP diet modulates visceral nociception by lipopolysaccharide-mediated intestinal barrier dysfunction and intestinal inflammation. *J Clin Invest* 128:267, 2018. Susan L. Gearhart

Diverticular Disease

and Common Anorectal Disorders ■ ■DIVERTICULAR DISEASE Incidence and Epidemiology In the United States, diverticulosis affects one-half of the population aged >60 years, and the majority of affected individuals will have no associated symptoms. However, studies have shown that ~5% of individuals with diverticulosis will develop acute diverticular disease. In addition, 10–25% of individuals with diverticular disease will experience recurrent symptoms, and up to 10% will develop complications leading to surgery. Diverticular disease has become the fifth most costly gastrointestinal disorder in the United States and is the leading indication for elective colon resection. The incidence of diverticular disease is on the rise and most prevalent among middle-aged individuals. The majority of patients with diverticular disease report a lower health-related quality of life and more

depression as compared to matched controls, thus adding to health care costs. Formerly, diverticular disease was confined to developed countries; however, with the adoption of westernized diets in developing countries, diverticulosis is on the rise across the globe. Immigrants to the United States develop diverticular disease at the same rate as U.S. natives. Although the prevalence among females and males is similar, males tend to present at a younger age. Known risk factors for the development of diverticular disease include the use of nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin, steroids, opioids, smoking, and sedentary lifestyle. Anatomy and Pathophysiology Two types of diverticula occur in the intestine: true and false (or pseudodiverticula). A true diverticulum is a saclike herniation of the entire bowel wall, whereas a pseudodiverticulum involves only a protrusion of the mucosa and submucosa through the muscularis propria of the colon (Fig. 339-1). The type of diverticulum most commonly seen in the colon is the pseudodiverticulum. The diverticula occur at the point where the nutrient artery, or vasa recta, penetrates through the muscularis propria, resulting in a break in the integrity of the colonic wall. Diverticula are most commonly encountered in the sigmoid colon. This anatomic restriction may be a result of the relative high-pressure zone within the muscular sigmoid colon. Higher-amplitude contractions combined with constipated, high-fat-content stool within the sigmoid lumen in an area of weakness in the colonic wall result in the creation of these diverticula. FIGURE 339-1 Gross and microscopic view of sigmoid diverticular disease. Arrows mark an inflamed diverticulum with the diverticular wall made up only of mucosa.

Consequently, the vasa recta is either compressed or eroded, leading to either perforation or bleeding. As mentioned above, diverticula commonly affect the left and sigmoid colon; the rectum is always spared. However, in Asian populations, 70% of diverticula are seen in the right colon and cecum. Diverticulitis is inflammation of a diverticulum. Previous understanding of the pathogenesis of diverticulosis attributed the disease to poor dietary choices, and the onset of diverticulitis would occur acutely when these diverticula become obstructed. However, evidence now suggests that the pathogenesis is more complex and multifactorial. Better understanding of the gut microbiota suggests that dysbiosis is an important aspect of disease. Chronic low-grade inflammation is thought to play a key role in neuronal degeneration, leading to dysmotility and high intraluminal pressure. As a consequence, pockets or outpouchings develop in the colonic wall where it is weakest. Studies have also shown that it is more common for patients with diverticular disease to have abnormal collagen cross-linking. Researchers hypothesize that abnormal collagen cross-linking leads to loss of intestinal compliance and, therefore, higher intraluminal pressure, leading to

pockets or outpouchings. A newer approach to examine causality in diseases such as diverticu litis is to examine genome-wide associations (GWAs) using a variety of biobanks. Biobanks from Iceland, Denmark, and the United Kingdom have been examined for GWAs in diverticular disease and identified LAMB4 and TNFSF15 variants to be associate with early-onset, severe diverticular disease in otherwise healthy families. Although the exact function these genes play in the development of diverticular disease is unknown, the protein encoded by the gene LAMB4 is part of the extra cellular matrix lamin family, whereas the protein encoded by the gene TNFSF15 is a member of the tumor necrosis family, which has implica tions in inflammation.

Presentation, Evaluation, and Management of Diverticular Bleeding

Hemorrhage from a colonic diverticulum is the most common cause of hematochezia in patients >60 years, yet only 20% of patients with diverticulosis will have gastrointestinal bleeding. Patients at increased risk for bleeding tend to be hypertensive, have atheroscle rosis, and regularly use anticoagulants and NSAIDs. Additional risk factors include obesity and a history of diabetes mellitus. Of note, due to increased use of anticoagulants in our aging population, there has been a rise in the incidence of diverticular bleeding. Most bleeds are self-limited and stop spontaneously with bowel rest. The lifetime risk of rebleeding is 25%. Initial localization of diverticular bleeding may include colonoscopy, multiplanar computed tomography (CT) angiogram, or nuclear medi cine tagged red cell scan. If the patient is stable, ongoing bleeding is best managed by angiography. If mesenteric angiography can localize the bleeding site, the vessel can be occluded successfully with a coil in 80% of cases. The patient can then be followed closely with repetitive colonoscopy, if necessary, looking for evidence of colonic ischemia. However, with highly selective coil embolization, the rate of colonic ischemia is <10%, and the risk of acute rebleeding is <25%. Longterm results (40 months) indicate that >50% of patients with acute diverticular bleeds treated with highly selective angiography have had definitive treatment. Alternatively, colonoscopic ligation with banding, placement of a detachable snare, and over-the-scope hemoclip have been shown to be effective methods to obtain hemostasis if the bleed ing site can be localized. These approaches have been shown to prevent rebleeding or the requirement of emergent surgery. In the event that these measures fail to achieve hemostasis, a segmental resection of the colon may be undertaken. This may be advantageous in patients on chronic anticoagulation and immunosuppression as delayed bleeding and perforation have been reported in this subpopulation. If the patient is refractory to angiographic or endoscopic treatments, unstable, or has required a large-volume transfusion, current recom mendations are that surgery should be performed. If the bleeding has been localized, a segmental resection can be performed. If the site of bleeding has not been definitively identified, a subtotal colectomy may be required. In patients without severe comorbidities, surgical

TABLE 339-1 Presentation of Diverticular Disease Uncomplicated Diverticular Disease—75% Abdominal pain Fever Leukocytosis Anorexia/obstipation Complicated Diverticular Disease—25% Abscess 16% Perforation 10% Stricture 5% Fistula 2% resection can be performed with a primary anastomosis. A higher anastomotic leak rate has been reported in patients who received

“ 10 units of blood. Presentation, Evaluation, and Staging of Diverticulitis

Acute uncomplicated diverticulitis characteristically presents with fever, anorexia, left lower quadrant abdominal pain, and obstipa tion (Table 339-1). The diagnosis of diverticulitis is best

made on a contrast-enhanced abdominal and pelvic CT scan demonstrating the following findings: sigmoid diverticula, thickened colonic wall >4 mm, and inflammation within the pericolic fat without the collection of contrast material or fluid. Additional syndromes have been identified that relate to the presence of diverticulosis or diverticular disease. Symptomatic colitis-associated diverticulosis (SCAD) is uncommon (<1% of patients with diverticulosis) and occurs when inflammation is identified between the diverticula on endoscopic assessment. Symptoms of SCAD mimic irritable bowel disease. Symptomatic uncomplicated diverticular disease (SUDD) is a condition seen in patients with known diverticular disease and ongoing abdominal pain without evidence of overt inflammation on radiographic imaging.

CHAPTER 339 Diverticular Disease and Common Anorectal Disorders Complicated diverticular disease is defined as diverticular disease associated with an abscess or perforation and less commonly with a fistula (Table 339-1). Symptoms of complicated diverticular disease may be similar to uncomplicated disease, or patients may exhibit signs of peritonitis indicating the presence of a diverticular perforation. If a pericolic abscess has formed, the patient may have abdominal distention and signs of localized peritonitis. Laboratory investigations often demonstrate a leukocytosis. Rarely, a patient may present with an air-fluid level in the left lower quadrant on plain abdominal film. This should raise a concern for a giant diverticulum of the sigmoid colon and is managed with resection to avoid impending perforation. Perforated diverticular disease is staged using the Hinchey classification system (Fig. 339-2). This staging system was developed to predict outcomes following the surgical management of complicated diverticular disease. The Hinchey staging system has been modified to include the development of a phlegmon or early abscess (Hinchey stage Ia). In complicated diverticular disease with fistula formation, common locations include cutaneous, vaginal, or vesicle fistulas. These conditions present with either passage of stool through the skin or vagina or the presence of air in the urinary stream (pneumaturia). Colovaginal fistulas are more common in women who have undergone a hysterectomy.

TREATMENT Medical Management of Diverticular Disease Asymptomatic diverticular disease discovered on imaging studies or at the time of colonoscopy is best managed by lifestyle changes. Although the data regarding dietary risks and symptomatic diverticular disease are limited (Table 339-2), patients may benefit from a fiber-enriched diet or supplements that include 30 g of fiber each day. The use of fiber decreases colonic transit time and, therefore, prevents increased intraluminal pressure, leading to the

Abscess I II Feces III IV **FIGURE 339-2 Hinchey classification of diverticulitis.** Stage I: Perforated diverticulitis with a confined paracolic abscess. Stage II: Perforated diverticulitis that has closed spontaneously with distant abscess formation. Stage III: Noncommunicating perforated diverticulitis with fecal peritonitis (the diverticular neck is closed off, and therefore, contrast will not freely expel on radiographic images). Stage IV: Perforation and free communication with the peritoneum, resulting in fecal peritonitis.

PART 10 Disorders of the Gastrointestinal System development of diverticulosis. The incidence of complicated diverticular disease appears to also be increased in patients who smoke and are obese. Therefore, patients should be encouraged to refrain from smoking and to join a weight loss program. The historical recommendation to avoid eating nuts is based on no more than anecdotal data.

ANTIBIOTICS The routine use of antibiotics in uncomplicated diverticular disease does not appear to reduce time to symptom resolution or reduce the risk of complications or recurrence. Two large, randomized trials (the AVOD trial and the Diabolo trial) and a large meta-analysis demonstrated that immunocompetent patients with uncomplicated diverticular disease had no difference in time to symptom resolution, recurrence rates, development of complicated diverticular disease, or surgery if treated with or without

antibiotics. Currently, patients who are immunocompromised, have findings of extensive inflammation on radiographic studies, are at risk for disease progression, or have computed tomography (CT) findings of complicated diverticular disease should be treated with antibiotics. Known risk factors for disease progression included the American Society of Anesthesiologists (ASA) classification of III or IV, >5 days of TABLE 339-2 The Use of Fiber in the Management of Diverticular Disease (DD) JOURNAL, STUDY YEAR PATIENTS (N) INTERVENTION STUDY LENGTH FINDINGS Lancet, 1977

Wheat or bran crisp bread 3 months Significant reduction of symptoms score BMJ, 1981

Bran, ispaghula, placebo 16 weeks No difference J Gastroenterol, 1977

Methylcellulose 3 months Significant reduction in symptoms BMJ, 2011 47,033 Vegetarian vs nonvegetarian 11.6 years Vegetarians had a 31% lower risk of DD Gastroenterology, 2012

Fiber consumption 12 years Fiber associated with great risk of DD JAMA, 2008 47,288 Nut, corn, popcorn consumption 18 years Higher nut, corn, and popcorn had lower risk of recurrence Ann R Coll Surg Engl, 1985

Fiber consumption 66 months Higher fiber associated with 19% reduction in symptom recurrence Source: Modified from A Turis et al: Review article: The pathophysiology and medical management of diverticulosis and diverticular disease of the colon. Aliment Pharmacol Ther 42:664, 2015.

symptoms, and elevation of C-reactive protein (CRP) or white blood cell (WBC) count. If the use of antimicrobial therapy is desired, the current recommended antimicrobial coverage for uncomplicated acute diverticulitis is a third-generation cephalosporin (or ciprofloxacin if there is a known allergy to cephalosporins) and metronidazole targeting a mixed flora. Alternatively, single-agent therapy with a third-generation penicillin such as IV piperacillin or oral penicillin/clavulanic acid may be effective. The usual course of antibiotics is 5 days. A study compared the use of intravenous (IV) versus oral antibiotics in uncomplicated diverticular disease and noted no difference in recovery time or progression of the disease and recommended that safe home treatment on oral antibiotics after a 6-h observation in the emergency department is reasonable. Exclusion criteria for home treatment included complicated diverticular disease, immunocompromised patient, significant active comorbidities, poor social support, or a decline during observation. DIET AND OTHER MEDICAL THERAPIES Patients should remain on a limited diet until their pain resolves. The use of anti-inflammatory medications (mesalazine) in randomized clinical trials has shown them to be beneficial at reducing symptoms and disease recurrence in patients with SUD. However, when objective signs of inflammation such as CRP and computerized imaging are taken into consideration, no benefit for the use of mesalazine has been shown. Probiotics are increasingly used by gastroenterologists for multiple bowel disorders and may prevent recurrence of diverticulitis. Specifically, probiotics containing *Lactobacillus acidophilus* and *Bifidobacterium* strains may be beneficial; however, a systematic review was unable to show any benefit to the use of probiotics alone. The addition of fiber or mesalazine with probiotics has shown some promise in maintaining remission. Rifaximin (a poorly absorbed broad-spectrum antibiotic), when compared to fiber alone for the treatment of SUD, is associated with 30% less frequent recurrent symptoms from uncomplicated diverticular disease. COLONOSCOPY Colonoscopy should

be considered ~6 weeks after the first episode of uncomplicated diverticular disease and after the development of complicated diverticular disease as the overall prevalence of colon cancer is low in these patients (<2%) but higher incidence has been seen with complicated diverticulitis (6–8%). The parallel epidemiology of colorectal cancer and diverticular disease provides enough concern for an endoscopic evaluation before operative management.

SURGICAL MANAGEMENT OF DIVERTICULAR DISEASE

Preoperative risk factors influencing postoperative mortality rates include higher ASA physical status class (Table 339-3) and pre existing organ failure. In patients who are low risk (ASA P1 and P2), surgical therapy can be offered to those who do not rapidly improve on medical therapy. For uncomplicated diverticular disease, medical therapy can be continued beyond two attacks without an increased risk of perforation requiring a colostomy. However,

TABLE 339-3 American Society of Anesthesiologists Physical Status Classification System

P1 A normal healthy patient
P2 A patient with mild systemic disease
P3 A patient with severe systemic disease
P4 A patient with severe systemic disease that is a constant threat to life
P5 A moribund patient who is not expected to survive without the operation
P6 A declared brain-dead patient whose organs are being removed for donor purposes

patients on immunosuppressive therapy, in chronic renal failure, or with a collagen-vascular disease have a fivefold greater risk of perforation during recurrent attacks. A multicentered randomized clinical trial (DIRECT trial) comparing surgery with conservative management for recurrent SUDD demonstrated that elective surgical resection was associated with an improved quality of life and was more cost-effective at 5 years following resection as compared to conservative management. Surgical therapy is generally indicated in all low-surgical-risk patients with complicated diverticular disease. A randomized trial (LASER trial) examined the use of laparoscopic surgery for refractory or complicated diverticular disease and demonstrated improved quality of life and a reduction in recurrent symptoms by 50% when compared to conservative treatment. The goals of surgical management of diverticular disease include controlling sepsis, eliminating complications such as fistula or obstruction, removing the diseased colonic segment, and restoring intestinal continuity. Table 339-4 lists the operations most commonly indicated based on the Hinchey classification and the predicted postoperative outcomes. The current options for uncomplicated diverticular disease include an open or a minimally invasive resection of the diseased area with reanastomosis to the rectosigmoid. Preservation of portions of the sigmoid colon may lead to early recurrence of the disease. The benefits of minimally invasive resection over open surgical techniques include early discharge (by at least 1 day), less narcotic use, less postoperative complications, and an earlier return to work. The options for the surgical management of complicated diverticular disease (Fig. 339-3) include the following open or minimally invasive procedures: (1) proximal diversion of the fecal stream with an ileostomy or colostomy and sutured omental patch with drainage, (2) resection with colostomy and mucous fistula or closure of distal bowel with formation of a Hartmann's pouch (Hartmann's procedure), (3) resection with anastomosis (coloproctostomy), or (4) resection with anastomosis and diversion (coloproctostomy with loop ileostomy or colostomy). (5) Laparoscopic technique of washout and drainage without diversion has been described for Hinchey III patients; however, a threefold increased risk of

TABLE 339-4 Outcome Following Surgical Therapy for Complicated Diverticular Disease Based on Modified Hinchey Staging

HINCHEY STAGE	OPERATIVE PROCEDURE	ANASTOMOTIC LEAK RATE, %	OVERALL MORBIDITY RATE, %	Ia (pericolic phlegmon)
Laparoscopic	or open colon resection			

Ib (pericolic abscess) Percutaneous drainage followed by laparoscopic or open colon resection II Percutaneous drainage followed by laparoscopic or open colon resection +/- proximal diversion with an ostomy III Laparoscopic washout and drainage or Laparoscopic or open resection with proximal diversion (ostomy) or Hartmann's procedure IV Hartmann's procedure or Washout with proximal diversion

recurrent peritonitis requiring reoperation with washout alone has been reported. Robotic surgery resection for complicated diverticular disease is associated with a lower rate of conversion to an open procedure.

Patients with Hinchey stage Ia may be managed with antibiotic therapy only or followed by resection with anastomosis following further workup and symptom resolution. Patients with Hinchey stages Ib and II disease are managed with percutaneous drainage followed by resection with anastomosis following further evaluation and symptom resolution. Current guidelines put forth by the American Society of Colon and Rectal Surgeons suggest, in addition to antibiotic therapy, CT-guided percutaneous drainage of diverticular abscesses that are >3 cm and have a well-defined wall. Abscesses that are <5 cm may resolve with antibiotic therapy alone. Contraindications to percutaneous drainage are no percutaneous access route, pneumoperitoneum, and fecal peritonitis. Drainage of a diverticular abscess is associated with a 20–25% failure rate. Urgent operative intervention is undertaken if percutaneous drainage fails and patients develop generalized peritonitis, and most will need to be managed with a Hartmann's procedure (resection of the sigmoid colon with end colostomy and stapling of the rectosigmoid distal to the diseased segment). In selected cases, nonoperative therapy may be considered. The management of Hinchey stage III disease is under debate. In this population of patients, no fecal peritonitis is present, and it is presumed that the perforation has sealed. Historically, Hinchey stage III has been managed with a Hartmann's procedure or with primary anastomosis and proximal diversion. Several studies have examined short- and long-term outcomes for laparoscopic peritoneal lavage to remove the peritoneal contamination and place drainage catheters should a communication to the bowel still exist. However, this procedure has been associated with an increased risk of requiring reoperation for ongoing peritonitis. Overall, ostomy rates are lower with the use of laparoscopic peritoneal lavage. No anastomosis of any type should be attempted in Hinchey stage IV disease or in the presence of fecal peritonitis. A limited approach to these patients is associated with a decreased mortality rate. CHAPTER 339 Diverticular Disease and Common Anorectal Disorders Recurrent Symptoms Recurrent abdominal symptoms following surgical resection for diverticular disease occur in 10% of patients. Recurrent diverticular disease develops in patients following inadequate surgical resection. A retained segment of diseased rectosigmoid colon is associated with twice the incidence of recurrence. The presence of irritable bowel syndrome may also cause recurrence of initial symptoms. Patients undergoing surgical resection for presumed diverticulitis and symptoms of chronic abdominal cramping and irregular loose bowel movements consistent with irritable bowel syndrome have poorer functional outcomes.

30% risk of peritonitis requiring reoperation if no resection is performed. Overall morbidity 50% Overall mortality 15% — Overall morbidity 50% Overall mortality 15%

FIGURE 339-3 Methods of surgical management of complicated diverticular disease. 1. Drainage, omental pedicle graft, and proximal diversion. 2. Hartmann's procedure. 3. Sigmoid resection with

coloproctostomy. 4. Sigmoid resection with coloproctostomy and proximal diversion. PART 10 Disorders of the Gastrointestinal System COMMON DISEASES OF THE ANORECTUM ■ ■ RECTAL PROLAPSE (PROCIDENTIA) Incidence and Epidemiology Rectal prolapse is six times more common in women than in men. The incidence of rectal prolapse peaks in women >60 years. Women with rectal prolapse have a higher incidence of associated pelvic floor disorders including urinary incontinence, rectocele, cystocele, and enterocele. About 20% of children with rectal prolapse will have cystic fibrosis. All children presenting with prolapse should undergo a sweat chloride test. Less common associations include Ehlers-Danlos syndrome, solitary rectal ulcer syndrome, congenital hypothyroidism, Hirschsprung's disease, dementia, cognitively impaired, and schizophrenia. Anatomy and Pathophysiology Rectal prolapse (procidentia) is a circumferential, full-thickness protrusion of the rectal wall through the anal orifice. It is often associated with a redundant sigmoid colon, pelvic laxity, and a deep rectovaginal septum (pouch of Douglas). Initially, rectal prolapse was believed to be the result of early internal rectal intussusception, which occurs in the upper to mid rectum. This was considered to be the first step in an inevitable progression to full-thickness external prolapse. However, only 1 of 38 patients with internal prolapse followed for >5 years developed full-thickness prolapse. Others have suggested that full-thickness prolapse is the result of damage to the nerve supply to the pelvic floor muscles or pudendal nerves from repeated stretching with straining to defecate. Damage to the pudendal nerves would weaken the pelvic floor muscles, including the external anal sphincter muscles. Bilateral pudendal nerve injury is identified significantly more with full-thickness prolapse and fecal incontinence than unilateral injury. Presentation and Evaluation In external prolapse, patient complaints include a palpable anal mass, bleeding per rectum, leakage of blood and mucus, and poor perianal hygiene. Prolapse of the rectum usually occurs following defecation and will spontaneously reduce or require the patient to manually reduce the prolapse. Constipation occurs in ~30–67% of patients with rectal prolapse. Differing degrees

A C B D FIGURE 339-4 Degrees of rectal prolapse. Mucosal prolapse only (A, B, sagittal view). Full-thickness prolapse associated with redundant rectosigmoid and deep pouch of Douglas (C, D, sagittal view). of fecal incontinence occur in 50–70% of patients. Patients with internal rectal prolapse will present with symptoms of both constipation and incontinence. Other associated findings include outlet obstruction (anismus) in 30%, colonic inertia in 10%, and solitary rectal ulcer syndrome in 12%. Office evaluation is best performed after the patient has been given an enema, which enables the prolapse to protrude. An important distinction should be made between full-thickness rectal prolapse and isolated mucosal prolapse associated with hemorrhoidal disease (Fig. 339-4). Mucosal prolapse is known for radial grooves rather than circumferential folds around the anus and is due to increased laxity of the connective tissue between the submucosa and underlying muscle of the anal canal. The evaluation of prolapse should also include cystoproctography and colonoscopy. These examinations evaluate for associated pelvic floor disorders and rule out a malignancy or a polyp as the lead point for prolapse. TREATMENT Rectal Prolapse The medical approach to the management of rectal prolapse is limited and includes stool-bulking agents or fiber supplementation to ease the process of evacuation. Surgical correction of rectal prolapse is the mainstay of therapy. Previously, the presence of internal rectal prolapse identified on imaging studies has been considered a nonsurgical disorder, and biofeedback was recommended. However, only one-third of patients will have successful resolution of symptoms from biofeedback. Two approaches are commonly considered: transabdominal and transperineal. Transabdominal approaches have been associated with lower recurrence rates, but some patients

with significant comorbidities are better served by a transperineal approach. Common transperineal approaches include a transanal proctectomy (Altmeier procedure), mucosal proctectomy (Delorme procedure), or placement of a Tirsch wire encircling the anus. The goal of the transperineal approach is to remove the redundant rectosigmoid

colon. Common transabdominal approaches include presacral suture or mesh rectopexy (Ripstein) with (Frykman-Goldberg) or without resection of the redundant sigmoid. Colon resection, in general, is reserved for patients with constipation and outlet obstruction. Ventral rectopexy is an effective method of abdominal repair of internal and full-thickness prolapse that does not require sigmoid resection. This repair has been shown to have improved pelvic floor functional results over other abdominal repairs. Transabdominal procedures can be performed effectively with laparoscopic and robotic techniques. Short- and long-term recurrence rates are low (<10%), and symptoms improved in more than three-fourths of patients. ■ ■FECAL INCONTINENCE Incidence and Epidemiology Fecal incontinence is the involuntary passage of fecal material or the inability to control the initiation of defecation. The prevalence of fecal incontinence in adults in the United States approaches 15% and is expected to increase given our aging population. A higher incidence of incontinence is seen among older parous women. One-half of patients with fecal incontinence also suffer from urinary incontinence. The cause of fecal incontinence is often multifactorial; however, the majority of women with fecal incontinence are parous and may have experienced obstetrical injury to the pelvic floor, either while carrying a fetus or during the delivery. An anatomic sphincter defect may occur in up to 32% of women following childbirth regardless of visible damage to the perineum. Risk factors at the time of delivery include prolonged labor, the use of forceps, and the need for an episiotomy. Symptoms of incontinence can present two or more decades after obstetric injury. Medical conditions known to contribute to the development of fecal incontinence are listed in Table 339-5. Anatomy and Pathophysiology The anal sphincter complex is made up of the internal and external anal sphincter. The internal sphincter is smooth muscle and a continuation of the circular fibers of the rectal wall. It is innervated by the intestinal myenteric plexus and is therefore not under voluntary control. The external anal sphincter is formed in continuation with the levator ani muscles and is under voluntary control. The pudendal nerve supplies motor innervation to the external anal sphincter. Obstetric injury may result in tearing of the muscle fibers anteriorly at the time of the delivery. This results in an obvious anterior defect on endoanal ultrasound. Injury may also be the result of stretching of the pudendal nerves during pregnancy or delivery of the fetus through the birth canal. Presentation and Evaluation Patients may suffer with varying degrees of fecal incontinence. Fecal incontinence is classified into three categories based on the clinical presentation. These categories include TABLE 339-5 Medical Conditions That Contribute to Symptoms of Fecal Incontinence Neurologic Disorders • Dementia • Brain tumor • Stroke • Multiple sclerosis • Tabes dorsalis • Cauda equina lesions Skeletal Muscle Disorders • Myasthenia gravis • Myopathies, muscular dystrophy Miscellaneous • Hypothyroidism • Irritable bowel syndrome • Diabetes • Severe diarrhea • Scleroderma

passive incontinence (passage of stool without awareness), urge incontinence (leakage of stool despite attempts at holding), and fecal seepage (seepage of residue generally following defecation associated with normal continence). Beyond the immediate problems associated with fecal incontinence, these patients are often withdrawn and suffer from depression. For this reason, quality-of-life measures are an important component in the evaluation of patients with fecal incontinence.

The evaluation of fecal incontinence should include a thorough history and physical examination including digital rectal examination (DRE). Weak sphincter tone on DRE and loss of the “anal wink” reflex (S1-level control) may indicate a neurogenic dysfunction. Perianal scars may represent surgical injury. Other studies helpful in the diagnosis of fecal incontinence include anal manometry, pudendal nerve terminal motor latency (PNTML), and endoanal ultrasound. Centers that care for patients with fecal incontinence will have an anorectal physiology laboratory that uses standardized methods of evaluating anorectal physiology. Pudendal nerve studies evaluate the function of the nerves innervating the anal canal using a finger electrode placed in the anal canal. Stretch injuries to these nerves will result in a delayed response of the sphincter muscle to a stimulus, indicating a prolonged latency. Finally, endoanal ultrasound will evaluate the extent of the injury to the sphincter muscles before surgical repair. Unfortunately, all of these investigations are user-dependent, and very few studies demonstrate that these studies predict outcome following an intervention. Magnetic resonance imaging (MRI) has also been utilized in the evaluation of the sphincter muscle complex in treatment planning for fecal incontinence, but its role has not been well-established. Rarely does a pelvic floor disorder exist alone. The majority of patients with fecal incontinence will have some degree of urinary incontinence. Similarly, fecal incontinence is a part of the spectrum of pelvic organ prolapse. For this reason, patients may present with symptoms of obstructed defecation as well as fecal incontinence. Careful evaluation including dynamic MRI or cine-defecography should be performed to search for other associated defects. Surgical repair of incontinence without attention to other associated defects may decrease the success of the repair.

CHAPTER 339 Diverticular Disease and Common Anorectal Disorders

TREATMENT Fecal Incontinence Medical management of fecal incontinence includes strategies to bulk up the stool, which help in increasing fecal sensation and complete evacuation. Stool bulking agents include fiber supplementation, loperamide, diphenoxylate, and bile acid binders. These agents help to bind the stool, resulting in more complete evacuation and decreasing the frequency of bowel movements. This can be particularly helpful in patients with mild symptoms of fecal incontinence. Patients may be offered a form of physical therapy called biofeedback. This therapy helps strengthen the external sphincter muscle while training the patient to relax with defecation to avoid unnecessary straining and further injury to the sphincter muscles. At a minimum, biofeedback is risk-free and most patients will have some improvement. For this reason, it should be incorporated into the initial recommendation to all patients with fecal incontinence. It is important to note that there is no medical or surgical therapy to reduce incontinence of flatus. Dietary alterations to reduce gas production and the use of probiotics are the only antidotal remedies. Historically, the “gold standard” for the treatment of fecal incontinence with an isolated sphincter defect has been the overlapping sphincteroplasty. The external anal sphincter muscle and scar tissue, as well as any identifiable internal sphincter muscle, are dissected free from the surrounding adipose and connective tissue. An overlapping sphincter repair is performed in an attempt to rebuild the muscular ring and restore its function. However, long-term results following overlapping sphincteroplasty have been poor, with a 50% failure rate over 5 years. Alternative therapies for the treatment of fecal incontinence included sacral nerve modulation, collagen-enhancing injectables,

and anal implantable self-expanding prostheses (THD Gatekeeper and Sphinkeeper). Sacral nerve stimulation (SNS) was the first implantable pacemaker and is a U.S. Food and Drug Administration (FDA)-approved adaptation of a procedure developed for the management of urinary incontinence. SNS is indicated in patients with two or more episodes of frank fecal incontinence per week. Long-

term results for SNS have been promising, with nearly 80% of patients having a reduction in incontinence episodes by at least 50%. There are inherent challenges with the SNS device, and to avoid these challenges, percutaneous or transcutaneous tibial nerve stimulation (PTNS or TTNS) is being offered to patients with fecal incontinence. PTNS is offered in a clinical setting, while TTNS can be performed at home. Both require weekly sessions of stimulation of the tibial nerve, which serves to feedback to the third sacral nerve root. Collagenenhancing injectables and anal self-expanding implants have been around for several years. Results from randomized trials have been variable but support the use of injectables/implantables in patients who are not candidates for sacral nerve modulation.

Finally, the use of stem cells to increase the bulk of the sphincter muscles is currently being tested. Stem cells can be harvested from the patient's own muscle, grown, and then implanted into their sphincter complex. Concern for cost and the need for an additional procedure have dampened enthusiasm.

MIXED CONSTIPATION AND FECAL INCONTINENCE It is estimated that up to 20% of patients presenting for evaluation of fecal incontinence have mixed constipation and fecal incontinence. Fecal incontinence, in this instance, is related to the large number of laxatives required to produce stool evacuation. This condition is more difficult to manage and often requires close collaboration with different specialties. One promising intervention is the use of a transanal irrigation system (Peristeen by Coloplast). This system provides daily irrigations of the left side of the colon to assist with evacuations and has shown promising results. The daily assistance with evacuation leads to less need for oral laxatives and reduced episodes of incontinence. One drawback is long-term adherence to the irrigations.

PART 10 Disorders of the Gastrointestinal System

HEMORRHOIDAL DISEASE Incidence and Epidemiology Symptomatic hemorrhoids affect >1 million individuals in the Western world per year. Hemorrhoidal disease can occur at any age and affects females and males at similar rates. The prevalence of hemorrhoidal disease is less in developing countries. The typical low-fiber, high-fat diet prominent in developed countries is associated with constipation and straining and the development of symptomatic hemorrhoids. Other risk factors for hemorrhoids include pregnancy and high-impact activities (bike riding).

Anatomy and Pathophysiology Hemorrhoidal cushions are a normal part of the anal canal. The vascular structures contained within this tissue aid in continence by preventing damage to the sphincter muscle. Three main hemorrhoidal complexes traversing the anal canal include the left lateral, the right anterior, and the right posterior. Prolonged engorgement with straining leads to prolapse of this tissue into the anal canal. Over time, the anatomic support system of the hemorrhoidal complex weakens, exposing this tissue to the outside of the anal canal where it is susceptible to injury. Hemorrhoids are commonly classified as external or internal. External hemorrhoids originate below the dentate line and are covered with squamous epithelium and are not associated with an internal component. Internal hemorrhoids originate above the dentate line and are covered with mucosa and transitional zone epithelium and represent the majority of hemorrhoids.

Presentation and Evaluation Patients commonly present to a physician for two reasons: bleeding and protrusion. Pain is less common than with fissures and, if present, is described as a dull ache from engorgement of the hemorrhoidal tissue. Severe pain may indicate a thrombosed external hemorrhoid. Hemorrhoidal bleeding is described

as painless bright red blood seen either in the toilet or upon wiping. Occasional patients can present with significant bleeding, which may be a cause of anemia; however, the presence of a colonic neoplasm must be ruled out in anemic patients, especially given the noted rise in young

patients with colorectal cancer. Patients who present with protruding tissue complain about inability to maintain perianal hygiene and anal itching (pruritis) and are often concerned about the presence of a malignancy. The diagnosis of hemorrhoidal disease is made on physical examination. Inspection of the perianal region for evidence of thrombosis or excoriation is performed, followed by a careful digital examination. Anoscopy is performed paying particular attention to the known position of hemorrhoidal disease. The patient is asked to strain. It is important to differentiate the circumferential appearance of a full-thickness rectal prolapse from the radial nature of prolapsing hemorrhoids (see "Rectal Prolapse," above). The stage and location of the hemorrhoidal complexes are defined.

TREATMENT Hemorrhoidal Disease The treatment for bleeding hemorrhoids is based on the stage of the disease (Table 339-6). In all patients with bleeding, the possibility of other causes must be considered, and further studies should include endoscopic assessments. With rare exceptions, the acutely thrombosed hemorrhoid can be excised within the first 48 h by performing an elliptical excision and clot extraction. This can often be performed at the bedside with local anesthesia; sitz baths, fiber, and stool softeners are prescribed. Additional nonoperative therapies for bleeding hemorrhoids include rubber band ligation, infrared coagulation, and sclerotherapy. Sensation begins at the dentate line; therefore, all procedures can be performed without discomfort either endoscopically or in the office. Bands are placed around the engorged tissue, causing ischemia and fibrosis. This aids in fixing the tissue proximally in the anal canal. Patients may complain of a dull ache for 24 h following band application. During sclerotherapy, 1–2 mL of a sclerosant (usually sodium tetradecyl sulfate) is injected using a 25-gauge needle into the submucosa of the hemorrhoidal complex. Care must be taken not to inject the anal canal circumferentially, or stenosis may occur. For surgical management of hemorrhoidal disease, excisional hemorrhoidectomy with sharp dissection (Milligan-Morgan hemorrhoidectomy), bipolar electrocautery ligation (LigaSure), transhemorrhoidal dearterialization (THD), and stapled hemorrhoidectomy ("the procedure for prolapse or hemorrhoids" [PPH]) are the procedures of choice. All surgical methods of management

TABLE 339-6 The Staging and Treatment of Hemorrhoids

DESCRIPTION OF CLASSIFICATION	TREATMENT
STAGE I Enlargement with bleeding	Fiber supplementation Short course of cortisone suppository Sclerotherapy Infrared coagulation
II Protrusion with spontaneous reduction	Fiber supplementation Short course of cortisone suppository Sclerotherapy Infrared coagulation
III Protrusion requiring manual reduction	Fiber supplementation Short course of cortisone suppository Rubber band ligation Operative hemorrhoidectomy
IV Irreducible protrusion	Fiber supplementation Cortisone suppository Operative hemorrhoidectomy

are equally effective in the treatment of symptomatic third- and fourth-degree hemorrhoids. Current postprocedure outcomes following hemorrhoidectomy target improvements in bleeding, pain, and recurrence rates. The sutured hemorrhoidectomy involves the removal of redundant tissue down to the anal verge, and unpleasant anal skin tags are removed as well. However, pain and postoperative bleeding are more common. The stapled hemorrhoidectomy is associated with less discomfort; however, this procedure does not remove anal skin tags, and an increased number of complications are associated with use of the stapling device. THD uses ultrasound guidance to ligate the blood supply to the anal tissue, hence reducing hemorrhoidal engorgement. Ligation with electrocautery uses a bipolar device (LigaSure) to remove the unwanted prolapsed tissue. Both THD and ligation with electrocautery have demonstrated similar short-term and long-term results; however, ligation with electrocautery requires less operative time. No procedures on hemorrhoids should be done in patients who are immunocompromised or who have active proctitis. Emergent hemorrhoidectomy for bleeding hemorrhoids is associated with a higher complication

rate. Acute complications associated with the treatment of hemorrhoids include pain, infection, recurrent bleeding, and urinary retention. Care should be taken to place bands properly and to avoid overhydration in patients undergoing operative hemorrhoidectomy. Late complications include fecal incontinence as a result of injury to the sphincter during the dissection. Anal stenosis may develop from overzealous excision, with loss of mucosal skin bridges for reepithelialization. Finally, an ectropion (prolapse of rectal mucosa from the anal canal) may develop. Patients with an ectropion complain of a “wet” anus as a result of inability to prevent soiling once the rectal mucosa is exposed below the dentate line. ■ ■

ANORECTAL ABSCESS Incidence and Epidemiology The development of a perianal abscess is more common in men than women by a ratio of 3:1. The peak incidence is in the third to fifth decade of life. Perianal pain associated with the presence of an abscess accounts for 15% of office visits to a colorectal surgeon. The disease is more prevalent in immunocompromised patients such as those with diabetes, hematologic disorders, or inflammatory bowel disease (IBD) and persons who are HIV positive. These disorders should be considered in patients with recurrent perianal infections.

Anatomy and Pathophysiology An anorectal abscess is an abnormal fluid-containing cavity in the anorectal region. Anorectal abscess results from an infection involving the glands surrounding the anal canal. Normally, these glands release mucus into the anal canal, which aids in defecation. When stool accidentally enters the anal glands, the glands become infected, and an abscess develops. Anorectal abscesses are perianal in 40–50% of patients, ischiorectal in 20–25%, intersphincteric in 2–5%, and supralelevator in 2.5% (Fig. 339-5).

Presentation and Evaluation Perianal pain and fever are the hallmarks of an abscess. Patients may have difficulty voiding and have blood in the stool. On physical examination, a large fluctuant area is usually readily visible. Routine laboratory evaluation shows an elevated WBC count. Diagnostic procedures are rarely necessary unless evaluating a recurrent abscess. A CT scan or MRI has an accuracy of 80% in determining incomplete drainage. If there is a concern about the presence of IBD, a rigid or flexible sigmoidoscopic examination may be done at the time of drainage to evaluate for inflammation within the rectosigmoid region. A more complete evaluation for Crohn’s disease would include a full colonoscopy and small-bowel enterography.

TREATMENT Anorectal Abscess As with all abscesses, the “gold standard” is drainage. Office drainage of an uncomplicated anorectal abscess may suffice. A small incision

Fistula tracts Abscesses Supralelevator

Intersphincteric Ischiorectal

Perianal

Intersphincteric

Extrasphincteric

Trans-sphincteric

Suprasphincteric

FIGURE 339-5 Common locations of anorectal abscess (left) and fistula in ano (right). A small incision close to the anal verge is made, and a Mallenkot drain is advanced into the abscess cavity. The Mallenkot catheter (a mushroom-shaped catheter with grooves) can be maintained by the patient for up to

1 week before being removed to avoid tissue ingrowth. For patients with a complicated abscess or who are diabetic or immunocompromised, drainage should be performed in an operating room under anesthesia. These patients are at greater risk for developing necrotizing fasciitis. The role of antibiotics in the management of anorectal abscesses is limited. Antibiotics are only warranted in patients who are immunocompromised or have obvious cellulitis on physical exam.

CHAPTER 339

■ ■ FISTULA IN ANO

Diverticular Disease and Common Anorectal Disorders

Incidence and Epidemiology The incidence of fistulizing perianal disease parallels the incidence of anorectal abscess and is estimated to be 1 in 10,000 individuals. Some 30–40% of abscesses will give rise to an anal fistula. Although the majority of the fistulas are cryptoglandular in origin, 10% are associated with IBD, tuberculosis, malignancy, and radiation.

Anatomy and Pathophysiology An anal fistula is defined as a communication of an abscess cavity with an identifiable internal opening within the anal canal. This identifiable opening is commonly located at the dentate line where the anal glands enter the anal canal. Patients experiencing continuous drainage at 1 month following the treatment of a perianal abscess likely have an anal fistula. These fistulas are classified by their relationship to the anal sphincter muscles, with 70% being intersphincteric, 23% transsphincteric, 5% suprasphincteric, and 2% extrasphincteric (Fig. 339-5).

Presentation and Evaluation A patient with an anal fistula will complain of constant drainage from the perianal region associated with tenderness. Examination under anesthesia is the best way to evaluate a fistula. However, in cases with a complex fistula, a preoperative MRI will identify tracts with an accuracy of 80%. During surgery, hydrogen peroxide injected through the external opening will aid in identifying the internal opening. Goodsall's rule states that a posterior external fistula will enter the anal canal in the posterior midline, whereas an anterior fistula will enter at the nearest crypt. A fistula exiting >3 cm from the anal verge may have a complicated upward extension and may not obey Goodsall's rule.

TREATMENT **Fistula in Ano** A newly diagnosed draining fistula is best managed with an operative intervention including initial placement of a seton catheter (a vessel loop or silk tie placed through the fistula tract), which

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