

# Diverticular Disease and Diverticulitis

Anish A. Sheth, M.D.,<sup>1</sup> Walter Longo, M.D.,<sup>2</sup> and Martin H. Floch, M.D.<sup>1</sup>

<sup>1</sup>Section of Digestive Diseases, and <sup>2</sup>Department of Surgery, Yale University School of Medicine, New Haven, CT

Diverticular disease is one of the most prevalent medical conditions to affect Western populations. Symptomatic diverticular disease can range from mild, low-level symptomatology similar to that seen in irritable bowel syndrome to acute bouts of diverticulitis complicated by abscess or frank perforation. This review discusses the epidemiology, pathophysiology, clinical presentation, and management of the spectrum of diverticular disease, including mention of recent advances in the treatment of chronic diverticular disease with aminosalicylates and probiotics.

(Am J Gastroenterol 2008;103:1550–1556)

## INTRODUCTION

Despite its prevalence, there remains a shroud of mystery surrounding the various aspects of diverticular disease. While epidemiologic studies have identified dietary factors as a cause for the western predominance of diverticulosis, there is much to be learned about the pathophysiology and treatment of diverticular inflammation (1, 2).

Diverticulitis is a wide-ranging disease that runs the spectrum from isolated, mild, acute attacks to severe, recurrent disease (3) (see Table 1). Even asymptomatic patients may have low-grade inflammation associated with colonic diverticula (4). Once thought to be a distinct clinical entity, emerging data suggest a possible overlap between chronic diverticular inflammation and inflammatory bowel disease (IBD) (5). This association has gathered more support with greater understanding of the entity known as segmental colitis associated with diverticula (SCAD) and the recent reports of therapeutic success with aminosalicylates (6, 7).

While the mainstays of diverticulitis treatment remain antibiotics and surgery, important questions remain with regard to the indications and timing of surgery both in the acute setting and for recurrent disease. Newer therapeutic strategies, including the use of oral aminosalicylates and probiotics, are making their way into the diverticulitis treatment algorithm (6, 8, 9).

This review will focus on the emerging evidence regarding the pathophysiology, diagnosis, and management of diverticulitis.

## EPIDEMIOLOGY

Diverticulosis is one of the most common conditions in western countries, but only a fraction of patients will ever experience an episode of diverticulitis (10). The prevalence of diverticular disease varies by study, with some quoting rates as high as 60% in those patients over the age of 70 (1). It is thought that approximately 20% of patients with diverticulosis will experience an inflammatory complication of the disease, ranging in severity from a single, mild, acute at-

tack of diverticulitis to more severe attacks characterized by perforation and abscess formation, occasionally resulting in chronic complications such as obstruction and fistula formation. Bleeding is another known complication of diverticular disease, but will not be addressed in this review.

There is a profound geographic variation between Asian and western countries with regard to the prevalence and pathophysiology of diverticular disease (11). Diverticulosis is much less common in Asian patients and is more likely to be found in the proximal colon when compared to their western counterparts, in whom the sigmoid colon is the most commonly affected segment. Several factors have been proposed to account for this difference, but observations that Asians who adopt a western-style diet have increased rates of diverticulosis lend support to the role of dietary fiber in diverticular formation.

## PATHOPHYSIOLOGY

In western populations, colonic diverticula are “pseudodiverticula” because herniation involves only the mucosal and submucosal layers (12). Observations by Burkitt and Painter lead to the theory that low-fiber diets played a role in the pathogenesis of diverticulosis (13). Subsequent epidemiologic studies of Asian populations and vegetarian diets have confirmed this association (14).

Diverticula form at weak points in the bowel wall, typically at sites where the vasa recta penetrate the colonic smooth muscle (15). Although not the focus of this review, it warrants mention that proximal colonic diverticula, most commonly seen in Asians, are true diverticula involving all layers of the colonic wall. Some have explained the colon's propensity for diverticular formation by its less muscular wall (12). In contrast to the two layers of smooth muscle (inner circular and outer muscular) found in other portions of the gastrointestinal tract, the colonic wall contains only a single complete layer of inner circular muscle; the outer longitudinal layer being divided into three bands, the taenia coli.

**Table 1.** Stages of Diverticular Disease

Stage 0	Development of diverticular disease
Stage I	Asymptomatic disease
Stage II	Symptomatic disease a. Single episode b. Recurrent c. Chronic (pain, diarrhea, IBD overlap/SCAD)
Stage III	Complicated Abscess Phlegmon Obstruction Fistulization Bleeding Sepsis Stricture

The most widely accepted theory of dietary fiber’s role in diverticular formation states that smaller-volume stool results in alterations in colonic motility that produce increased intraluminal pressures (16). High intraluminal pressures are generated when the sigmoid colon undergoes “segmentation,” a process during which smooth muscle contraction separates the colon into functionally distinct compartments (17). This normal physiologic process becomes exaggerated in those with low-volume stools, thereby generating markedly elevated intrasegmental colonic pressures that are then transmitted to the colonic wall. The law of LaPlace ( $P$  [pressure] =  $kT$  [wall tension]/ $R$ [radius]), which states that intraluminal pressure is inversely related to lumen diameter, has been used to explain the role of low-volume stools in the generation of increased luminal pressures and development of diverticula (12).

Examination of surgical and postmortem specimens has offered insight into the structural changes found in patients with diverticular disease. The term *mychosis* has been used to describe the contracted, thickened appearance of the colon folds (18). Originally thought to be secondary to circular muscle hypertrophy, the colon’s corrugated appearance is now known to be a secondary effect of bowel shortening and increased elastin deposition (19). The exact reason for elastin deposition in patients with diverticular disease is unknown,

although some have implicated increased uptake of proline from Western diets (12). Further support for the role of connective tissue changes in the pathophysiology of diverticulosis comes from the finding that patients with Marfan and Ehlers–Danlos syndromes develop diverticula at an early age.

Although much has been learned about the development of diverticula, much less is known about the pathogenesis of diverticular inflammation. As discussed earlier, only a minority of patients with diverticulosis will develop symptomatic disease.

Initial theories of diverticulitis focused on diverticular obstruction by a fecalith or food particle followed by increased intradiverticular pressure and perforation. Interest has been generated in the role of altered peridiverticular colonic flora and low-grade chronic inflammation leading to periods of symptomatic disease, similar to periods of exacerbation and remission in IBD (20). While altered colonic flora and low-grade inflammation may account for mild, chronic diverticular disease such as SCAD, the true triggers of an acute episode of diverticulitis remain unknown.

**CLINICAL FEATURES**

Acute diverticulitis has long been classified as uncomplicated or complicated based on the severity of clinical presentation and radiologic findings. On the other hand, the manifestations of chronic diverticular inflammation can be quite varied, ranging from asymptomatic disease to mild, intermittent abdominal pain mistakenly attributed to irritable bowel syndrome (IBS) to a clinical picture similar to that of IBD with chronic abdominal pain and hematochezia. Further complicating the picture of chronic diverticular inflammation is the clinical entity termed SCAD.

**Acute Diverticulitis**

Acute diverticulitis typically presents with fever, left lower quadrant abdominal pain, and leukocytosis. While the diagnosis of acute diverticulitis is often easily established, especially in those with a prior history of similar episodes, determining the severity of an attack is significantly more

**Table 2.** EAES Clinical Classification

	Clinical Description	Recommended Diagnostic Testing
Grade I symptomatic, uncomplicated disease	Fever, crampy abdominal pain	Colonoscopy vs barium enema to rule out malignancy, colitis
Grade II recurrent, symptomatic disease	Recurrence of above	CT scan vs barium enema
Grade III complicated disease	Abscess Hemorrhage Stricture Fistula Phelgmon Purulent and fecal peritonitis Perforation Obstruction	CT scan

**Table 3.** Buckley Classification

	CT Findings
Mild	Bowel wall thickening, fat stranding
Moderate	Bowel wall thickening >3 mm, phlegmon or small abscess
Severe	Bowel wall thickening >5 mm, frank perforation with subdiaphragmatic free air, abscess >5 cm

difficult. This assessment carries great importance as some patients can be easily treated with oral antibiotics in the outpatient setting, while others require hospitalization, intravenous (IV) antibiotics, and close observation for complications.

The European Association for Endoscopic Surgeons (EAES) has devised a clinical classification scheme to stratify patients with acute diverticulitis (21) (see Table 2). This classification based on clinical presentation can help guide subsequent steps in diagnostic testing. The addition of computed tomography (CT) findings to the clinical presentation allows clinicians to accurately determine the need for hospitalization and surgical intervention.

Although not relevant to acute bouts of diverticulitis, several recent studies have utilized symptom scores in patients with chronic diverticular disease to assess responses to therapy (6, 7). Questionnaires assigned quantitative scores with regard to symptoms such as pain, bloating, fever, diarrhea/constipation, and general well-being.

There are two radiologic classifications that attempt to assess the severity of acute diverticulitis. Both the Buckley and Hinchey systems use CT scanning to stage disease (see Tables 3 and 4). While neither scoring system has been prospectively validated against clinical outcomes, a single retrospective study found good correlation between the Hinchey CT classification and intraoperative findings in cases of perforated diverticulitis requiring surgery (22).

Another retrospective study assessed the role of CT in predicting the need for surgery and found that patients with CT evidence of abscess more often failed conservative treatment and were more likely to develop recurrent disease. The authors suggest that CT can be used to stratify patients who should be referred to early surgical intervention (23).

### Complicated Diverticulitis

Acute diverticulitis can result in both immediate and long-term complications. Immediate complications include abscess formation, peritonitis, obstruction, fistula formation, and, rarely, hemorrhage. Infection can spread locally to involve nearby structures such as the ovary and hip joint, or travel via the portal vein to cause hepatic abscesses. Rarely,

recurrent infection of the hip joint with enteric bacteria can be the presenting sign of otherwise asymptomatic chronic diverticular disease (24).

Fistulas occur in 12% of patients with diverticulitis, most commonly involving the bladder (25). Other common fistulas include colovaginal, colocutaneous, and enterocolic fistulas. Some authors have attributed the increasing incidence of the fistulizing disease to the overall lower rates of surgical intervention in diverticulitis (26). Recurrent attacks of diverticulitis can also result in colonic stricture formation requiring periodic dilatation or surgical resection.

### Immunocompromised Patients

It is not clear that immunocompromised patients have a higher rate of diverticulitis, but studies do support that their episodes are more likely to be complicated (27). Patients who have received organ transplants, or are receiving chemotherapy or taking chronic immunosuppressive medications, are at higher risk for perforation and abscess formation. The clinical presentation of these patients is further complicated by the often indolent, smoldering nature of acute episodes of diverticulitis and the lack of usual signs of the disease such as fever and elevated white blood cell count. Some authors have advocated surgical intervention after the first episode of diverticulitis, but caution should be used as immunocompromised patients have increased perioperative mortality (28).

### Chronic Pain

Many patients with diverticulosis suffer from chronic abdominal pain, typically located in the left lower quadrant. Many of these patients are labeled as having IBS. A review by Horgan *et al.* suggests that a subset of patients has marked improvement in symptoms with sigmoid resection. In this series, three-fourths of resected specimens contained acute and chronic inflammatory changes (29).

Visceral hypersensitivity may account for symptoms in patients with chronic pain and diverticular disease (30). Animal studies have shown that chronic colonic inflammation can result in altered nerve regeneration and neurotransmitter release, resulting in increased sensitivity to stimuli (31).

Altered colonic motility plays a role in the pathogenesis of chronic diverticular pain. Several studies have demonstrated an increase in sigmoid intraluminal pressure in patients with symptomatic diverticular disease, especially after administration of provocative agents (32). However, in a study by Bassotti *et al.*, there was no difference in the amplitude or frequency of colonic contractions between symptomatic patients and controls (33). Interestingly, despite this lack of difference, there was a marked increase in the reported symptomatic contractions in patients with diverticular disease as compared to controls. This may further support the role of visceral hypersensitivity in the pathophysiology of chronic pain in diverticular disease.

Finally, low-level mucosal inflammation may cause chronic diverticular symptoms such as abdominal pain and diarrhea (3, 5, 7). Increase in the relative levels of

**Table 4.** Hinchey Classification (Perforated Diverticulitis)

	CT Findings
Stage I	Pericolic abscess or phlegmon
Stage II	Pelvic, intra-abdominal or retroperitoneal abscess
Stage III	Generalized purulent peritonitis
Stage IV	Generalized fecal peritonitis

proinflammatory cytokines (IL [interleukin]-1, TNF [tumor necrosis factor]), triggered, in part, by altered peridiverticular microflora, can give rise to mucosal inflammation and result in symptoms such as lower abdominal pain/discomfort, bloating, tenesmus, and diarrhea (34). Further evidence for this association comes from the recent studies demonstrating the efficacy of 5-ASA (aminosalicylate) compounds in achieving symptom relief (discussed under “Management”) (6–8).

### **SCAD and IBD**

SCAD is a unique form of chronic colitis limited to areas of the colon with diverticular formation (4, 35). Gore *et al.* first reported on the presence of an entity they termed “diverticular colitis” in 1.5% of colonoscopies/flexible sigmoidoscopies (36). Often mistakenly diagnosed as either ulcerative colitis or Crohn’s disease, patients typically present with pain and intermittent rectal bleeding. Colonoscopic evaluation reveals friable mucosa in the region of diverticular disease, with a notable absence of the aphthous ulcerations typically found in Crohn’s disease. The inflammatory reaction is characterized pathologically by a focal chronic colitis without granulomas and characteristically involves the interdiverticular colonic mucosa, sparing the diverticula themselves (37). Rectal sparing, when present, can help distinguish SCAD from ulcerative colitis. Small studies have shown benefit to both medical therapy with antibiotics and 5-ASA compounds as well as surgical resection (38).

It is generally accepted that SCAD is a distinct clinicopathologic entity that has much in common with the idiopathic IBDs, Crohn’s disease, and ulcerative colitis (4, 39, 40). Not only do these entities share similar histologic changes, but also numerous case reports have described patients initially diagnosed with SCAD who go on to develop either ulcerative colitis or Crohn’s disease months to years after resection of the affected segment (41). Indeed, an “overlap hypothesis” has been proposed to explain the similarity in clinical features between diverticular disease-associated colitis and IBD (5, 12, 42). The pathophysiology is unknown, but some have theorized that a “blind pouch effect” may play a role in the development of both IBD and SCAD (43). This hypothesis states that chronic inflammation develops in areas such as the appendix, surgically-created J pouches, and segments with diverticular disease because of fecal stasis and resultant alteration in bacterial microflora. This, in turn, causes dysregulation of inflammatory mediators and increased levels of proinflammatory cytokines.

## **MANAGEMENT**

### **Acute Diverticulitis**

Several society guidelines have been published regarding the management of diverticulitis (44,45). The mainstay of treatment of acute uncomplicated diverticulitis is antibiotic therapy, usually consisting of agents that broadly cover aerobic and anaerobic Gram-negative organisms. The decision on

whether to hospitalize patients is based on clinical criteria such as the severity of the episode as assessed by physical examination and laboratory testing, the ability to tolerate oral antibiotics and hydration, and the presence or absence of significant comorbid conditions.

The diagnosis of complicated diverticular disease is based on clinically evident generalized peritonitis or CT evidence of abscess, phlegmon, perforation, fistula, obstruction, or hemorrhage. Treatment in these situations is initiated with IV antibiotics, bowel rest, and pain control, but definitive management usually requires more invasive techniques such as percutaneous drainage or surgery (46). Percutaneous drainage has been advocated for peridiverticular abscesses measuring greater than 4 cm in diameter (Hinchey stage 2) (47).

The timing and type of surgery for acute complicated diverticulitis is controversial (21, 46, 48). The major dilemma is whether or not primary colonic anastomosis can be safely performed in the acute setting (49). Traditionally, double- or triple-staged procedures were performed for complicated diverticulitis. Diverting or end colostomies were created over concern that primary anastomoses would break down and that operative complications would be greater (50, 51). The 2006 American Society of Colon and Rectal Surgeons practice guidelines reflect the ongoing uncertainty regarding the optimal surgical procedure in complicated diverticulitis; “the precise role and safety of primary anastomosis, especially without proximal diversion, remain unsettled” (44).

While some patients especially those with frank fecal peritonitis and sepsis (Hinchey stage 4) still receive sigmoid resection with the Hartmann’s pouch and end colostomy, recent studies have called into question this strategy for patients with less fulminant presentations (52, 53). The ability to perform CT-guided drainage of diverticular abscesses allows for delay in surgical resection, thereby increasing the chances of a single-stage procedure. Several studies have demonstrated the safety of single-stage operations in the setting of pericolic abscesses and even in selected cases of frank peritonitis (49, 54). Another study highlighted the feasibility in performing laparoscopic resection and anastomosis when undertaken in the “inflammation-free” period after treatment with antibiotics (55).

### **Chronic Symptoms**

It is now appreciated that the spectrum of diverticular disease includes patients with chronic, low-level symptoms. Patients with chronic symptomatology have favorable long-term outcomes, but impaired quality of life (56). As discussed above, chronic inflammation, similar to that occurring in the chronic idiopathic IBDs, can give rise to low-level symptomatology in patients with diverticular disease, and this suggests that some patients may benefit from treatment with anti-inflammatory medications (3, 5, 7).

Uncontrolled studies have examined the potential benefit of 5-ASA compounds in the treatment and prevention of symptomatic diverticular disease. Three Italian studies examined the role of mesalamine (+/– rifaximin) in

the treatment of uncomplicated diverticular disease (7, 8, 57). These studies assessed patient symptoms during and following attacks of diverticular disease, finding that mesalamine significantly improved patients' symptoms and global sense of well-being. These studies also demonstrated a decrease in recurrent episodes of diverticulitis, suggesting that low-level chronic inflammation does play a role in the development of acute diverticulitis.

Given the possible role of altered peridiverticular microflora in the development of chronic mucosal inflammation, two studies have been performed to evaluate the role of probiotic therapy in symptomatic, uncomplicated diverticular disease (6, 58). The first study of 15 patients with recurrent, symptomatic diverticular disease found that patients treated with *Escherichia coli* Nissle 1917 had a longer period of remission than those receiving standard therapy (14.10 months vs 2.43 months,  $P < 0.001$ ) (58). A second study by Tursi *et al.* found that patients who received a combination of mesalamine and *Lactobacillus casei* were more likely to remain in remission (based on the Global Symptom Score) after 1 yr when compared to patients who received either treatment alone (6). While these results appear promising, it should be noted that both studies were small in sample size and neither included a placebo arm.

## SPECIAL SCENARIOS

### Recurrent Diverticulitis

Approximately 25% of patients will suffer more than one attack of acute diverticulitis (59). A landmark study by Parks *et al.*, published in 1969, showed that recurrent diverticulitis was a more virulent disease, with subsequent attacks being more likely to fail medical therapy and require emergency surgery (60). This study had formed the basis for the major surgical societies' recommendations to perform sigmoid resection after the second episode of uncomplicated acute diverticulitis in patients over the age of 50 and after the initial episode in patients under the age of 50. More recent data suggest that patients with recurrent diverticulitis (> two episodes) do not have higher morbidity and mortality when compared to patients with less than two attacks (61).

There is a lack of randomized data on the optimal timing for colectomy and this has resulted in reliance on observational data, as summarized in expert opinion pieces and various professional society practice guidelines (20). While some authors recommend elective sigmoid resection after two episodes of diverticulitis, others favor a more conservative approach (62–64). The American Society of Colon and Rectal Surgeons amended their previous guidelines by commenting in their 2006 report that “The decision to recommend elective colectomy after recovery from acute diverticulitis should be made on a case-by-case basis” (44). A decision analysis suggests that surgical resection should be postponed until after the fourth attack of uncomplicated diverticulitis (53). When compared to colectomy after the second episode, this strategy would result in 0.5% less deaths and 0.7% less colostomies.

### Young Patients

Previously thought to be uncommon, there is now an increasing incidence of diverticular disease in patients under 40 yr of age (65). Rising rates of obesity in this population may be a contributing factor to the earlier development of diverticular complications (66).

Similar to recurrent diverticulitis, diverticulitis in the young (age <40 yr) has historically been thought of as a more virulent disease and, therefore, has been more aggressively managed by surgical resection (67). Recent data suggest that rates of frank perforation and surgical resection during the initial attack of diverticulitis are lower in younger patients (68).

The optimal management of young patients with diverticular disease, particularly those presenting without frank perforation, remains unclear. A retrospective review of patients with diverticular disease who were less than 50 yr of age found that only 1 of the 196 medically managed patients presented with frank perforation during a median 5 yr of follow-up (69). The higher lifetime rate of surgery in young patients with diverticulitis may be due to their longer life expectancy and, therefore, the statistically greater risk of recurrence (70).

## CONCLUSIONS

Our understanding of diverticular disease has greatly increased in the past five decades since Parks' initial characterization of the disease. This increasingly common affliction now encompasses mild, chronic symptomatology often attributed to IBS and overlap syndromes similar to the idiopathic IBDs. The mainstays in management still include age-old medical and surgical therapies, but ever-evolving guidelines with regard to the optimal timing of surgical intervention and newer medical treatments including aminosallylates and probiotics will hopefully continue to improve the management of this most challenging clinical condition.

---

**Reprint requests and correspondence:** Martin H. Floch, M.D., M.A.C.G., Digestive Disease Center, Yale University School of Medicine, 40 Temple Street-1A, New Haven, CT 06510.

*Received December 5, 2007; accepted January 16, 2008.*

---

## REFERENCES

1. Parra-Blanco A. Colonic diverticular disease: Pathophysiology and clinical picture. *Digestion* 2006;73(Suppl 1):47–57.
2. Tursi A. New physiopathological and therapeutic approaches to diverticular disease of the colon. *Expert Opin Pharmacother* 2007;8:299–307.
3. Floch MH. A hypothesis: Is diverticulitis a type of inflammatory bowel disease? *J Clin Gastroenterol* 2006;40(Suppl 3):S121–5.
4. Lamps LW, Knapple WL. Diverticular disease-associated segmental colitis. *Clin Gastroenterol Hepatol* 2007;5:27–31.
5. Peppercorn MA. The overlap of inflammatory bowel disease and diverticular disease. *J Clin Gastroenterol* 2004;38(5 Suppl):S8–10.

6. Tursi A, Brandimarte G, Giorgetti GM, et al. Mesalazine and/or lactobacillus casei in preventing recurrence of symptomatic uncomplicated diverticular disease of the colon: A prospective, randomized, open-label study. *J Clin Gastroenterol* 2006;40:312–6.
7. Di Mario F, Comparato G, Fanigliulo L, et al. Use of mesalazine in diverticular disease. *J Clin Gastroenterol* 2006;40(Suppl 3):S155–9.
8. Brandimarte G, Tursi A. Rifaximin plus mesalazine followed by mesalazine alone is highly effective in obtaining remission of symptomatic uncomplicated diverticular disease. *Med Sci Monit* 2004;10:PI70–3.
9. Floch MH, White JA. Management of diverticular disease is changing. *World J Gastroenterol* 2006;12:3225–8.
10. Bogardus ST Jr. What do we know about diverticular disease? A brief overview. *J Clin Gastroenterol* 2006;40(Suppl 3):S108–11.
11. Painter NS, Burkitt DP. Diverticular disease of the colon: A deficiency disease of western civilization. *BMJ* 1971;2:450–4.
12. West AB, Losada M. The pathology of diverticulosis coli. *J Clin Gastroenterol* 2004;38(5 Suppl):S11–6.
13. Burkitt D. Diverticular disease of the colon epidemiological evidence relating it to fibre-depleted diets. *Trans Med Soc Lond* 1973;89:81–4.
14. Gear JS, Ware A, Fursdon P, et al. Symptomless diverticular disease and intake of dietary fibre. *Lancet* 1979;1:511–4.
15. Ferzoco LB, Raptopoulos V, Silen W. Acute diverticulitis. *N Engl J Med* 1998;338:1521–6.
16. Painter NS, Burkitt DP. Diverticular disease of the colon: A 20th century problem. *Clin Gastroenterol* 1975;4:3–21.
17. Painter NS, Truelove SC, Ardran GM, et al. Segmentation and the localization of intraluminal pressure in the human colon, with special reference to the pathogenesis of colonic diverticula. *Gastroenterology* 1968;54(4 Suppl):778–80.
18. Kelly JK. Polypoid prolapsing mucosal folds in diverticular disease. *Am J Surg Pathol* 1991;15:871–8.
19. Whiteway J, Morson BC. Elastosis in diverticular disease of the sigmoid colon. *Gut* 1985;26:258–66.
20. Floch CL. Diagnosis and management of acute diverticulitis. *J Clin Gastroenterol* 2006;40(Suppl 3):S136–44.
21. Kohler L, Sauerland S, Neugebauer E. Diagnosis and treatment of diverticular disease: Results of a consensus development conference. The Scientific Committee of the European Association for Endoscopic Surgery. *Surg Endosc* 1999;13:430–6.
22. Lohrmann C, Ghanem N, Pache G, et al. CT in acute perforated sigmoid diverticulitis. *Eur J Radiol* 2005;56:78–83.
23. Kaiser AM, Jiang JK, Lake JP, et al. The management of complicated diverticulitis and the role of computed tomography. *Am J Gastroenterol* 2005;100:910–7.
24. Ravo B, Khan SA, Ger R, et al. Unusual extraperitoneal presentations of diverticulitis. *Am J Gastroenterol* 1985;80:346–51.
25. Woods RJ, Lavery IC, Fazio VW, et al. Internal fistulas in diverticular disease. *Dis Colon Rectum* 1988;31:591–6.
26. Bordeianou L, Hodin R. Controversies in the surgical management of sigmoid diverticulitis. *J Gastrointest Surg* 2007;11:542–8.
27. Tyau ES, Prystowsky JB, Joehl RJ, et al. Acute diverticulitis. A complicated problem in the immunocompromised patient. *Arch Surg* 1991;126:855–8; discussion 858–9.
28. Fratini J, Longo WE. Diagnosis and treatment of chronic and recurrent diverticulitis. *J Clin Gastroenterol* 2006;40(Suppl 3):S145–9.
29. Horgan AF, McConnell EJ, Wolff BG, et al. Atypical diverticular disease: Surgical results. *Dis Colon Rectum* 2001;44:1315–8.
30. Spiller R. How inflammation changes neuromuscular function and its relevance to symptoms in diverticular disease. *J Clin Gastroenterol* 2006;40(Suppl 3):S117–20.
31. Mayer EA, Collins SM. Evolving pathophysiologic models of functional gastrointestinal disorders. *Gastroenterology* 2002;122:2032–48.
32. Attisha RP, Smith AN. Pressure activity of the colon and rectum in diverticular disease before and after sigmoid myotomy. *Br J Surg* 1969;56:891–4.
33. Bassotti G, Battaglia E, De Roberto G, et al. Alterations in colonic motility and relationship to pain in colonic diverticulosis. *Clin Gastroenterol Hepatol* 2005;3:248–53.
34. Rogler G, Andus T. Cytokines in inflammatory bowel disease. *World J Surg* 1998;22:382–9.
35. Peppercorn MA. Drug-responsive chronic segmental colitis associated with diverticula: A clinical syndrome in the elderly. *Am J Gastroenterol* 1992;87:609–12.
36. Gore S, Shepherd NA, Wilkinson SP. Endoscopic crescentic fold disease of the sigmoid colon: The clinical and histopathological spectrum of a distinctive endoscopic appearance. *Int J Colorectal Dis* 1992;7:76–81.
37. Jani N, Finkelstein S, Blumberg D, et al. Segmental colitis associated with diverticulosis. *Dig Dis Sci* 2002;47:1175–81.
38. Sultan K, Fields S, Panagopoulos G, et al. The nature of inflammatory bowel disease in patients with coexistent colonic diverticulosis. *J Clin Gastroenterol* 2006;40:317–21.
39. Makapugay LM, Dean PJ. Diverticular disease-associated chronic colitis. *Am J Surg Pathol* 1996;20:94–102.
40. Shepherd NA. Diverticular disease and chronic idiopathic inflammatory bowel disease: Associations and masquerades. *Gut* 1996;38:801–2.
41. Pereira MC. Diverticular disease-associated colitis: Progression to severe chronic ulcerative colitis after sigmoid surgery. *Gastrointest Endosc* 1998;48:520–3.
42. Harpaz N, Sachar DB. Segmental colitis associated with diverticular disease and other IBD look-alikes. *J Clin Gastroenterol* 2006;40(Suppl 3):S132–5.
43. Goldblum JR, Appelman HD. Appendiceal involvement in ulcerative colitis. *Mod Pathol* 1992;5:607–10.
44. Rafferty J, Shellito P, Hyman NH, et al. Practice parameters for sigmoid diverticulitis. *Dis Colon Rectum* 2006;49:939–44.
45. Stollman NH, Raskin JB. Diagnosis and management of diverticular disease of the colon in adults. Ad Hoc Practice Parameters Committee of the American College of Gastroenterology. *Am J Gastroenterol* 1999;94:3110–21.
46. Wolff BG, Devine RM. Surgical management of diverticulitis. *Am Surg* 2000;66:153–6.
47. Ambrosetti P, Robert J, Witzig JA, et al. Incidence, outcome, and proposed management of isolated abscesses complicating acute left-sided colonic diverticulitis. A prospective study of 140 patients. *Dis Colon Rectum* 1992;35:1072–6.
48. Wong WD, Wexner SD, Lowry A, et al. Practice parameters for the treatment of sigmoid diverticulitis—supporting documentation. The Standards Task Force. The American Society of Colon and Rectal Surgeons. *Dis Colon Rectum* 2000;43:290–7.
49. Wasvary H, Turfah F, Kadro O, et al. Same hospitalization resection for acute diverticulitis. *Am Surg* 1999;65:632–5; discussion 636.
50. Auguste LJ, Wise L. Surgical management of perforated diverticulitis. *Am J Surg* 1981;141:122–7.

51. Graves HA Jr, Franklin RM, Robbins LB 2nd, et al. Surgical management of perforated diverticulitis of the colon. *Am Surg* 1973;39:142–7.
52. Bahadursingh AM, Virgo KS, Kaminski DL, et al. Spectrum of disease and outcome of complicated diverticular disease. *Am J Surg* 2003;186:696–701.
53. Salem L, Veenstra DL, Sullivan SD, et al. The timing of elective colectomy in diverticulitis: A decision analysis. *J Am Coll Surg* 2004;199:904–12.
54. Constantinides VA, Tekkis PP, Athanasiou T, et al. Primary resection with anastomosis versus Hartmann's procedure in nonelective surgery for acute colonic diverticulitis: A systematic review. *Dis Colon Rectum* 2006;49:966–81.
55. Reissfelder C, Buhr HJ, Ritz JP. What is the optimal time of surgical intervention after an acute attack of sigmoid diverticulitis: Early or late elective laparoscopic resection? *Dis Colon Rectum* 2006;49:1842–8.
56. Salem TA, Molloy RG, O'Dwyer PJ. Prospective, five-year follow-up study of patients with symptomatic uncomplicated diverticular disease. *Dis Colon Rectum* 2007;50:1460–4.
57. Tursi A, Brandimarte G, Daffina R. Long-term treatment with mesalazine and rifaximin versus rifaximin alone for patients with recurrent attacks of acute diverticulitis of colon. *Dig Liver Dis* 2002;34:510–5.
58. Fric P, Zavoral M. The effect of non-pathogenic *Escherichia coli* in symptomatic uncomplicated diverticular disease of the colon. *Eur J Gastroenterol Hepatol* 2003;15:313–5.
59. Janes S, Meagher A, Frizelle FA. Elective surgery after acute diverticulitis. *Br J Surg* 2005;92:133–42.
60. Parks TG. Natural history of diverticular disease of the colon. A review of 521 cases. *BMJ* 1969;4:639–42.
61. Chapman JR, Dozois EJ, Wolff BG, et al. Diverticulitis: A progressive disease? Do multiple recurrences predict less favorable outcomes? *Ann Surg* 2006;243:876–80.
62. Bordeianou L, Hodin R. Controversies in the surgical management of sigmoid diverticulitis. *J Gastrointest Surg* 2007;11:542–8.
63. Ibele A, Heise CP. Diverticular disease: Update. *Curr Treat Options Gastroenterol* 2007;10:248–56.
64. Makela JT, Kiviniemi HO, Laitinen ST. Elective surgery for recurrent diverticulitis. *Hepatogastroenterology* 2007;54:1412–6.
65. Konvolinka CW. Acute diverticulitis under age forty. *Am J Surg* 1994;167:562–5.
66. Schauer PR, Ramos R, Ghiatas AA, et al. Virulent diverticular disease in young obese men. *Am J Surg* 1992;164:443–6; discussion 446–8.
67. Cunningham MA, Davis JW, Kaups KL. Medical versus surgical management of diverticulitis in patients under age 40. *Am J Surg* 1997;174:733–5; discussion 735–6.
68. Ambrosetti P, Robert JH, Witzig JA, et al. Acute left colonic diverticulitis in young patients. *J Am Coll Surg* 1994;179:156–60.
69. Guzzo J, Hyman N. Diverticulitis in young patients: Is resection after a single attack always warranted? *Dis Colon Rectum* 2004;47:1187–90; discussion 1190–1.
70. Peppas G, Bliziotis IA, Oikonomaki D, et al. Outcomes after medical and surgical treatment of diverticulitis: A systematic review of the available evidence. *J Gastroenterol Hepatol* 2007;22:1360–8.

---

#### CONFLICT OF INTEREST

**Guarantor of the article:** Martin H. Floch, M.D.

**Specific author contributions:** Anish A. Sheth was the primary preparer of the manuscript; Walter Longo was involved in the manuscript review and editing of surgical topics; and Martin H. Floch was involved in the manuscript organization, editing, and review.

**Financial support:** None.

**Potential competing interests:** None.

---