



# Diverticular Disease

## *Traditional and Evolving Paradigms*

### **ABSTRACT**

Diverticular disease includes diverticulosis, which are sac protrusions of the intestinal mucosa, and diverticulitis, inflammation of the diverticula. Diverticular disease is listed as one of the top 10 leading physician diagnoses for gastrointestinal disorders in outpatient clinic visits in the United States. There are several classifications of diverticular disease ranging from asymptomatic diverticulosis to diverticulitis with complications. Several theories are linked to the development of diverticula which includes the physiology of the colon itself, collagen cross-linking, and recently challenged, low-fiber intake. The differential diagnoses of lower abdominal pain in addition to diverticular disease have overlapping signs and symptoms, which can make a diagnosis challenging. Identification of the distinct signs and symptoms of each classification will assist the practitioner in making the correct diagnosis and lead to appropriate management. The findings from recent studies have changed the paradigm of diverticular disease. The purpose of this article is to discuss traditional dogma and evolving concepts in the pathophysiology, prevention, and management of diverticular disease. Practitioners must be knowledgeable about diverticular disease for improved outcomes.

### **Case Study**

Marie Anderson, a 60-year African American woman, presents to a practitioner's office complaining of lower abdominal pain, fever of 101.2°F (maximum temperature), and nausea for 2 days. The practitioner obtains more information from Mrs. Anderson about the history of her present illness. Specifically, questions regarding the location of her abdominal pain, description, radiation, and associated symptoms are further investigated. The patient reports that her pain is located in the left lower quadrant (LLQ), is intermittent, and is accompanied by several episodes of diarrhea, in addition to the already reported nausea and fever. She denies recent travel, vomiting, melena,

hematochezia, dysuria, unintended weight loss, hematemesis, and radiation of pain. She reports that she had a colonoscopy 3 years ago and remembers that she had hemorrhoids and "pockets in her colon." Past medical history includes hypertension and past surgical history is unremarkable. Her current medications include hydrochlorothiazide, and she has no known allergies to medications or latex. She is a nonsmoker and lives with her husband of 40 years. Family history includes colon cancer in her mother diagnosed at 72 years of age. On physical examination, she is found to have localized tenderness in the LLQ and normal bowel sounds and no hepatosplenomegaly or masses. The rectal examination is within normal limits.

### **Differential Diagnoses for Lower Quadrant Abdominal Pain**

The patient's gender, age, comorbidities, family history, and social history should be considered in the differential diagnoses. The differential diagnosis of lower abdominal pain includes many conditions with overlapping signs and symptoms and can be challenging for the practitioner. The correct diagnosis can make a difference for the patient to be managed in an outpatient setting or be admitted to a hospital. Table 1 reviews common differential diagnoses for lower abdominal pain as well as the location, description of pain, associated signs and symptoms, and physical examination findings.

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**TABLE 1. Differential Diagnosis of Lower Abdominal Pain**

Differential Diagnosis	Location of Pain	Pain Description	Associated Signs and Symptoms	Precipitating Events	Physical Examination Findings
Appendicitis	RLQ	Colicky	Vomiting once pain starts Constipation Fever	Movement and coughing	RLQ guarding Rebound to RLQ
Cholecystitis/Cholelithiasis	RUQ to Right scapula	Colicky	Nausea/Vomiting Dark urine Light stool Jaundice	Eating fatty foods	RUQ tenderness on palpation and percussion
Pancreatitis	LUG epigastric radiates to back	Steady mild to severe	Nausea, vomiting Diaphoresis	Lying supine	Abdominal distention Decreased bowel sounds
Salpingitis	RLQ or LLQ	Cramplike		Menses makes pain worse	Adnexa and cervix tender on palpation Increased bowel sounds
Ectopic pregnancy	Lower quadrant	Sudden onset of pain persistent	Tender adnexal mass Vaginal bleeding		
Mittelschmerz	Lower abdomen	Dull, crampy, sharp, and sudden	Mild vaginal bleeding		
Pelvic inflammatory disease	Lower abdomen and pelvis	Varying severity	Fever, heavy vaginal discharge, irregular menstrual bleeding, dysuria, dyspareunia		Cervical motion tenderness Uterine compression tenderness on bimanual examination
Abdominal aorta rupture or dissection	Chest or abdomen	Unbearable			Decreased femoral pulses
Inflammatory bowel disease	Generalized abdominal pain	Crampy	Diarrhea, mucus, fever, weight loss, aphthous ulcers, rectal bleeding		
Irritable bowel syndrome	Generalized abdominal pain	Symptoms relieved by bowel movement	Diarrhea, constipation, or both		Hypo- or hyperactive bowel sounds, distention on palpation
Ovarian cyst	Pelvic pain radiating to back, thighs	Dull, achy	If large, increased urination		
Colon cancer	Possible generalized abdominal pain	Achy, cramping	Weight loss, rectal bleeding	Radiation therapy for cancer, family history, IBD, age >50 years, race, personal history	Mass may be palpable
Incarcerated hernia	RLQ or LLQ			Coughing straining	Hernia or mass on palpation
Diverticulitis	LLQ or RLQ pain	Achy, crampy	Diarrhea, fever, nausea, vomiting, possibly dysuria	History of diverticula	LLQ or RLQ tenderness on palpation

Note. IBD = inflammatory bowel disease; LLQ = left lower quadrant; LUQ = left upper quadrant; RLQ = right lower quadrant; RUQ = right upper quadrant.

There are multiple differential diagnoses for lower abdominal pain with several that are gender specific. The most common differential diagnosis of lower quadrant pain is acute appendicitis if the patient complains of colicky right lower quadrant (RLQ) pain with vomiting, constipation, and fever. The patient will report pain is worse with movement while coughing and lying still often improves pain. Cholecystitis may be the cause if colicky right upper quadrant pain radiating to the right scapula, nausea/vomiting, dark urine, light stool, and jaundice are present. Pancreatitis should be suspected if the pain is located in the left upper quadrant/epigastric area and radiates to the patient's back with nausea/vomiting present. Often, the patient will report that leaning forward relieves pain.

Inflammatory bowel disease (IBD) should be suspected in patients who present with generalized abdominal pain, diarrhea, mucus, fever, weight loss, and occasionally aphthous ulcers. Another consideration in the differential of lower abdominal pain is irritable bowel syndrome (IBS). Irritable bowel syndrome is a chronic condition caused by visceral hypersensitivity, an excessive perception or response to stimuli. Presenting symptoms commonly include cramping, abdominal pain, bloating, flatus, and/or diarrhea/constipation.

Colon cancer should be suspected if unintended weight loss and/or rectal bleeding are present. In addition, in a female patient, differential diagnoses include ovarian cyst with a presentation of unilateral lower abdominal pain, salpingitis in a menstruating female with cramp-like pain, and mittelschmerz suspected in a patient who complains of dull, cramping pain with mild vaginal bleeding. Ectopic pregnancy should be considered with a sudden onset of persistent pain and vaginal bleeding. Abdominal aortic rupture or dissection, an acute emergency requiring immediate intervention, should be suspected if the patient complains of unbearable pain in the abdomen or the chest. An incarcerated hernia should be suspected in a patient who presents with RLQ or LLQ pain that worsens with cough or straining.

## Diverticular Disease

One common cause of LLQ pain associated with nausea and vomiting in adults is diverticular disease. Diverticular disease is an inclusive term that includes diverticulosis and diverticulitis. Diverticula (osis) are saclike protrusions of the intestinal mucosa that extend through the muscular wall of the colon. Diverticulitis is inflammation of the diverticula and is thought to occur when the necks of the diverticula become obstructed with thickened, dried food particles or trapped feces (Lutwak & Dill, 2013; Tursi & Papagrigroriadis, 2009). The openings of most diverticula are approximately 5–10 mm in size.

In Western countries, diverticular disease is mostly left sided (descending colon), with 95% involving the sigmoid colon (World Gastroenterology Organisation, 2007). Left-sided diverticulosis poses a higher risk of inflammatory complications, mainly diverticulitis (Tursi, 2015). Right-sided diverticular disease (ascending colon) is more common in Asia and is associated with a higher risk of bleeding (Tursi, 2015). It is believed to be related to a genetic predisposition. However, the incidence of both right- and left-sided diverticular disease is significantly increasing in Chinese people. This may be related to greater affluence, change in lifestyle, and an aging population (Fong, Tan, Foo, Sim, & Cheong, 2011).

## Epidemiology

In 2009, diverticular disease was the sixth leading physician diagnosis for gastrointestinal disorders in outpatient clinical visits in the United States (U.S.) (Peery et al., 2012b). The prevalence of diverticulosis increases with age, as 70% of individuals 80 years of age or older are affected (Gaglia & Probert, 2015). The lifetime risk of individuals with diverticulosis who will develop acute diverticulitis is less than 5% (Shahedi et al., 2012). Following recovery from a first episode of diverticulitis, 13.3%–36% of patients will experience recurrence (Gaglia & Probert, 2015). Complicated diverticulitis, which is considered diverticulitis that leads to issues such as perforation, fistulas, or obstruction, has an uncommon recurrence (Hall et al., 2011).

Although diverticulitis is often associated as a condition of the elderly, it is increasingly diagnosed in patients younger than 40 years (Etzioni, Mack, Beart, & Kaiser, 2009). The reason younger patients are diagnosed with diverticulitis remains unclear. Most younger patients with diverticulitis are obese, but a causal link is currently unknown and in need of further evaluation (Pilgrim, Hart, & Speakman, 2013).

## Pathophysiology

There are several theories linked to the development of diverticula which includes the physiology of the colon itself, collagen cross-linking, and possibly, low-fiber intake. The colon, unlike the small intestine, contains only one layer of muscle, the inner circular muscle. The outer layer comprises three bands, called “taeniae coli.” One of the taeniae coli is the taenia mesocolic. The other two bands, taeniae omentalis and taenia libera, are located on the medial and lateral parts of the colon. The vasa recta supply blood to the mucosa and submucosa (West, 2006).

The colonic wall is weakest between the mesenteric and antimesenteric teniae where microscopic studies found muscle atrophy in patients with diverticular disease. Diverticula often form in these areas of weakness

through the circular muscle (Hughes, 1969). In addition, patients with diverticular disease, especially after 40 years of age, have been observed to have abnormal collagen cross-linking. Collagen gives the colon its expandability, strength, and ability to maintain its shape. Collagen cross-linking causes tissue to be more rigid, leaving it more prone to tearing, especially under increased colonic pressure and, therefore, more vulnerable for diverticula to form (Matrana & Margolin, 2009; Wess, Eastwood, Wess, Busuttill, & Miller, 1995; World Gastroenterology Organisation, 2007).

It is long believed that a deficiency of dietary fiber can cause diverticula to develop. During the Industrial Revolution, much of the fiber in flour was removed by stone grinding, and in 1880, the introduction of roller milling removed the remaining two-thirds of fiber leading to a decline in fiber intake. Diverticular disease incidence rose in the century following the Industrial Revolution in the U.S. and Europe to a common colonic disorder (Painter & Burkitt, 1971).

Diverticular disease was rare in Africa and Asia where the diet was typically high in fiber resulting in greater stool weights and faster transit times. With the adoption of a western diet, African and Japanese immigrants developed diverticular disease. These observations led Painter and Burkitt (1971) in a landmark study to theorize that low dietary fiber was associated with smaller caliber stools, smaller colon diameters, and higher colon pressures, leading to segmentation and the development of diverticular disease. Segmentation is the process when the colon is separated into distinct compartments of increased colonic pressure, leading to herniation and diverticula (Painter & Burkitt, 1971). Patients with diverticular disease also display slow wave motility patterns, and intake of fiber has been shown to normalize motility patterns (Matrana & Margolin, 2009).

Recent data on the role of dietary fiber is conflicting. Strate (2012) and Peery and Sandler (2013) concluded that Painter and Burkitt's study was flawed because dietary data were not collected on the subjects studied and differences in the distribution of the ages of Western and African subjects was not addressed. As a result, longer life span of individuals (age) in Western versus African and Asian countries may have accounted for the difference in incidence of diverticular disease (Peery & Sandler, 2013; Strate, 2012).

Conversely, other studies have found fiber to be beneficial for diverticular disease. In a large study of 43,881 American males, between the ages of 40 and 75 years, high intake of dietary fiber was found to reduce the risk of diverticular disease. Furthermore, fiber from fruit and vegetable sources was particularly associated with a reduced risk of diverticular disease (Aldoori et al., 1998). Crowe, Appleby, Allen and Key (2011) also found that those with the highest fiber

consumption were less likely to have complications from diverticular disease. In contrast, Peery and Sandler (2013) found that a high-fiber diet was associated with a higher prevalence of diverticula. More research is necessary as guidelines for fiber intake in diverticular disease are unclear. A fiber-rich diet is associated with many benefits such as reduced cholesterol, control of blood glucose, and assistance in achieving a healthy weight among others (Ye, Chacko, Chou, Kugizaki, & Liu, 2012). It is therefore, prudent to consider this when discussing dietary fiber with patients.

### Signs and Symptoms of Diverticular Disease

There are several classifications of diverticular disease: diverticulosis, symptomatic uncomplicated diverticular disease (SUDD), acute uncomplicated diverticulitis, and complicated diverticulitis. Identification is largely based on symptoms. Identification of the distinct signs and symptoms of each classification will assist the practitioner in making the correct diagnosis and lead to appropriate management. Table 2 provides the classification of diverticular disease, signs and symptoms, and traditional management. New strategies are evolving; however, high-quality randomized controlled studies are needed to justify their use (Boynton & Floch, 2013).

#### Diverticulosis

Most patients with diverticulosis are asymptomatic and are unaware of its existence. The diverticula are often discovered as an incidental finding during a colonoscopy. Patients are sometimes puzzled as to how they developed diverticula. Such a time period represents an opportunity for practitioners to provide education.

#### Symptomatic Uncomplicated Diverticular Disease

It is with the onset of symptoms that diverticular disease comes to light. Recent evidence suggests that SUDD occurs in the presence of low-grade chronic inflammation in patients with symptomatic diverticular disease but without severe or acute symptoms (Mosadeghi, Bhuket, & Stollman, 2015). Typically, patients will present with mild nonspecific abdominal pain, most likely left-sided, without fever or abnormal laboratory studies (Weizman & Nguyen, 2011). Patients may also present with colicky abdominal pain, relieved by defecation or passing flatus, and/or bloating and altered bowel habits (Symeonidis et al., 2011). The physical examination is often unremarkable (Weizman & Nguyen, 2011).

#### Acute Uncomplicated Diverticulitis

The clinical presentation of acute uncomplicated diverticulitis may include LLQ or suprapubic pain; however;

**TABLE 2.** Diverticular Disease: Signs and Symptoms With Traditional Management

Diverticular Disease	Signs and Symptoms	Traditional Management
Diverticulosis	Asymptomatic	Diet high in fiber
Symptomatic uncomplicated diverticulosis	Discomfort in LL abdomen Vague lower abdominal or suprapubic discomfort Bloating, nausea, diarrhea, and mucus	Bowel rest Clear liquid diet
Acute uncomplicated diverticulitis	LLQ or RLQ pain Diarrhea, constipation Nausea, vomiting, anorexia Dysuria and urinary frequency Fever	Outpatient basis for those with good oral intake, supportive social structure—mild symptoms and no peritoneal signs Clear liquid diet advanced with symptom improvement Broad-spectrum antibiotic such as amoxicillin plus clavulanic acid, sulfamethoxazole–trimethoprim, or a quinolone with metronidazole for 7–10 days After resolution of the inflammatory process, colonoscopy should be done 4–6 weeks later Hospitalization for the very elderly, immunocompromised, severe comorbid disease, high fevers, and significant leukocytosis
Complicated diverticulitis	Abscess formation, perforation with peritonitis, fistula, intestinal obstruction, or stricture	Operative intervention should be based on clinical scenario, physiologic status, physical examination, and response to ongoing therapy

*Note.* LL = left lower; LLQ = left lower quadrant; RLQ = right lower quadrant.

RLQ pain, particularly in Asian patients, may be the presenting area of pain. Pain may be described as intermittent or constant with diarrhea or constipation. Fever and leukocytosis may also be present. Patients may report nausea, vomiting, anorexia, or hematochezia. As a result of bladder irritation from the close proximity of the bladder to the inflamed colon, patients may complain of dysuria and frequency. On examination, localized tenderness, guarding, and rebound tenderness may be present. Bowel sounds may be normal or depressed (Gaglia & Probert, 2015; Mehta & Probert, 2011; Tursi & Papagrigoriadis, 2009).

### Complicated Diverticulitis

Complications from diverticulitis include perforation with peritonitis, abscess formation, fistula, intestinal obstruction, or stricture (Symeonidis et al., 2011). On abdominal or rectal examination, a tender mass may be present in addition to persistent fever, even when treatment is given for the onset of symptoms of diverticulitis. Rupture of the diverticular abscess can lead to purulent peritonitis or fistulas that extend to a nearby organ. The most common fistulas are colovesical fistulas, followed by colovaginal fistulas. Other fistulas that develop less frequently are between the colon, small intestine, uterus, gallbladder, and skin (Symeonidis et al., 2011). Another complication that can result from diverticulitis is

obstruction of the small and large bowel. Complete obstruction can occur as a result of strictures and fibrosis that narrow the colonic lumen from frequent attacks of diverticulitis (Symeonidis et al., 2011).

Segmental colitis associated with diverticular disease, a distinct and pathological disorder, is a rare form of chronic diverticulitis with an unknown etiology that affects the elderly, particularly males. It develops in the sigmoid colon of those with diverticular disease and has a benign course with endoscopic findings that show nonspecific inflammation in the sigmoid colon, particularly, in those patients who present with abdominal pain and bloody diarrhea. It can be difficult to differentiate from IBD and hypothesized to be a form of IBD. Biopsies are essential to distinguish between the two disorders (Gaglia & Probert, 2015).

### Prevention Strategies of Diverticular Disease

For many years, patients with diverticulosis were advised to avoid nuts, corn, and seeds in their diet to reduce the risk of developing diverticulitis. However, no evidence exists that these foods are harmful and may actually decrease risk of diverticular disease because of their high-residue nature. Until recently, recommendations for prevention of diverticulitis were well established. Strate (2012) reported a high-fat diet, red meat intake

with a low-fiber diet, or obesity increases risk of symptomatic diverticular disease. However, other studies found evidence that a high-fiber diet may protect against diverticular disease.

Fiber is believed to lower intracolonic pressures, speed transit times, increase stool weight, and aide in more frequent bowel movements (Matrana & Margolin, 2009). Other studies found that physical activity, particularly vigorous activity, such as running, was the only specific activity to decrease the risk of diverticular disease (Aldoori et al., 1995; Williams, 2009). Conversely, Peery et al. (2012a) found no association between physical activity and symptomatic diverticulosis and, interestingly, found that a high-fiber diet was associated with a higher prevalence of diverticulosis. The study also found that participants with greater than 15 bowel movements per week had a 70% greater risk for diverticulosis than participants with fewer than seven bowel movements per week (Peery et al., 2012a). Further studies are necessary for conclusive evidence as results are unclear.

### Diagnosis of Diverticular Disease

The diagnosis of symptomatic diverticular disease (diverticulosis) can be made based on a patient's history and physical examination. It is important to ask about location and character of pain as well as associated symptoms such as fever, chills, nausea, vomiting, or change in bowel habits that may indicate acute diverticulitis (Touzios & Dozois, 2009). For symptoms suggesting acute diverticulitis, computed tomographic (CT) scan of the abdomen and the pelvis with contrast should be obtained (Gaglia & Probert, 2015; Moorman, 2012). A CT scan is useful to help confirm the diagnosis, grade severity, and guide the course of treatment.

A CT scan is also helpful to rule out disorders that may appear as diverticulitis such as appendicitis, IBD, or gynecological disorders (Feingold et al., 2014; Martin & Stocchi, 2011). Computed tomographic scan results suggesting moderate diverticulitis include the presence of diverticula with signs of inflammation of the pericolonic fat and thickening of the colonic wall of more than 4 mm. Severe diverticulitis is suggested when there is evidence of pericolonic abscess, free air, or extravasation of contrast (Biondo, Borao, Millan, Kreisler, & Jaurrieta, 2011). It is important to consider ordering serum electrolytes, complete blood count, and amylase, lipase, and liver function tests to assist in confirming the diagnosis and rule out differential diagnoses.

### Management of Diverticular Disease

Guidelines from gastroenterology societies on diverticular disease have been published, such as the American College of Gastroenterology in 1999 and the World Gastroenterology Organisation in 2007, but

have not had recent updates. The American Gastroenterological Association Guidelines for acute diverticulitis are currently being updated. There are recent studies challenging traditional management guidelines. As a result of the changing dogma, traditional and evolving concepts in the management of diverticular disease are presented.

### Asymptomatic Diverticulosis

Asymptomatic diverticulosis does not require any treatment. Most patients will remain asymptomatic and unaware of the presence of diverticula until found on colonoscopy. A diet high in fiber has long been suggested to promote bowel health (Stollman & Raskin, 1999). The role of fiber in managing diverticulosis still needs to be confirmed with high-quality randomized clinical trials (Boynton & Floch, 2013).

### Symptomatic Uncomplicated Diverticular Disease

The main goal of treatment of SUDD is to improve symptoms, prevent symptoms from reoccurring, and prevent complications. Traditional treatment involves bowel rest with a clear liquid diet that is advanced to a regular diet as symptoms improve (Stollman & Raskin, 1999; Tursi & Papagrigroriadis, 2009).

Although the pathophysiology of SUDD is not completely understood, chronic low-grade inflammation may play a role. It has been suggested that chronic inflammation in diverticular disease may be similar to that in IBD (Boynton & Floch, 2013). As a result of these findings, inflammation has become a new investigational target, and the use of mesalamine, an aminosalicilate, has been studied in the management of symptomatic disease and preventing recurrence of diverticulitis (Mosadeghi et al., 2015). In a systemic review and meta-analysis of the efficacy and treatment of SUDD with mesalamine, findings suggest mesalamine to be effective in the treatment of SUDD. Patients were reported to have fewer recurrences of acute diverticulitis. The authors concluded, however, that larger well-designed studies are needed to confirm the results (Jafri et al., 2013).

The use of mesalamine with a probiotic has also been investigated. Probiotics are thought to restore the normal intestinal flora that may be altered because of stasis and reduced intestinal time. Probiotics have been used in the management of various conditions such as diarrhea, constipation, bloating, IBS, and IBD (Maconi, Monteleone, Bezzio, Furfaro, & de Franchis, 2013). Probiotics with or without mesalamine in the treatment of SUDD remains unclear as results of studies have been inconclusive and underpowered (Mosadeghi et al., 2015).

There is also evidence that there is a presence of microscopic and subclinical infection in SUDD patients. As a result, studies have examined the use of rifaximin

as a nonabsorbable antibiotic effective against gram-positive and gram-negative bacteria. Rifaximin has been used to treat traveler's diarrhea, and small bowel bacterial overgrowth (Boynnton & Floch, 2013). Inconclusive evidence has been found in the efficacy of intermittent rifaximin to prevent the occurrence of acute diverticulitis and recurrence (Mosadeghi et al., 2015; Tursi & Danese, 2014).

### Acute Uncomplicated Diverticulitis

Mild symptoms of acute uncomplicated diverticulitis without peritoneal signs can be managed as an outpatient. Outpatient management is not suitable for patients who are vomiting/unable to tolerate oral intake, have significant comorbidities including insulin-dependent diabetes mellitus, are on immunosuppressive medication, unable to self-care, have a lack of adequate family or social support, or find home treatment unacceptable (Feingold et al., 2014; Jackson & Hammond, 2014). The clinical presentation of the patient and the providers' clinical impression should determine the need for hospitalization.

According to the World Gastroenterology Organization (2007) practice guidelines for diverticular disease, a low-residue diet and antibiotics such as amoxicillin plus clavulanic acid or sulfamethoxazole-trimethoprim or metronidazole with a quinolone for 7–10 days are acceptable choices for management. However, recent studies have questioned the use of antibiotics in the treatment of acute diverticulitis. A Cochrane review of antibiotic use in uncomplicated diverticulitis found that in spite of published guidelines, the best published data does not support the use of antibiotics (Shabanzadeh & Wille-Jørgensen, 2012). It has been suggested that antibiotic use was found to have no effect on complications or the need for surgery or recurrence rate (Morris, Regenbogen, Hardiman, & Hendren, 2014).

The recommendations of antibiotic therapy are based on tradition and expert opinions and not on evidence derived from controlled trials (de Korte et al., 2011). Two retrospective studies with limited design did not demonstrate any benefit of antibiotic use (de Korte et al., 2011). Antibiotic overuse has produced an increasing problem of resistance and, therefore, requires the need for diligent use (Chabok, Pahlman, Hjern, Haapaniemi, & Smedh, 2012). Further evidence for the role of antibiotics in uncomplicated diverticulitis management is necessary as practice guidelines have not addressed newer findings (Morris et al., 2014). Finally, data on the usefulness of probiotics in preventing diverticulitis long term are lacking (Maconi et al., 2013).

Patient teaching should include instruction to call the office for increasing pain, fever, or the inability to

tolerate liquids. Improvement in symptoms should occur within 2 to 3 days. Diet may also be slowly advanced if improvement is noted. If no improvement occurs within that time, hospitalization may be necessary. Traditionally, after an acute episode and complete resolution of the inflammatory process, usually 4 to 6 weeks, a colonoscopy is considered to confirm the diagnosis and rule out colonic neoplasia. Colonoscopy is not recommended during the acute phase because of a greater risk of perforation (Peery & Sandler, 2013; Stollman & Raskin, 1999).

Recent literature suggests that it is not necessary to have a colonoscopy following radiologically confirmed uncomplicated diverticulitis. Colonoscopy is suggested only for radiologically confirmed complicated diverticulitis due to a greater risk of malignancy. Tursi (2015) concluded that it is strongly advisable to have a colonoscopy following diverticulitis to exclude other diseases that share similarities. Peery and Sandler (2013) added that a colonoscopy should be performed to rule out colon cancer after an initial occurrence of diverticulitis; however, if a recent colonoscopy was done before the patient developed diverticulitis, the benefit is unknown.

### Recurrent Diverticulitis

Elective sigmoid resection was once commonly done following a second episode of diverticulitis regardless of complications. More recent guidelines suggest that a decision for resection following recurrent diverticulitis should be based on a case-by-case basis with the patients' age, comorbidities, the presence of chronic or lingering symptoms, and the frequency and severity of attacks (Feingold et al., 2014).

### Complicated Diverticulitis

For complicated diverticulitis, elective colectomy should typically be considered after the patient recovers from an episode of complicated diverticulitis. Instead of age, the decision for operative intervention should be based on the clinical scenario, physiologic status of the patient, physical examination, and response to ongoing therapy. Historically, routine elective resection for those younger than 50 years was advised as it was thought that younger patients had greater risk of recurrence of diverticulitis or complications of disease. Recent studies have shown that there is no difference in the risk of recurrence or complications between younger and older patients (Feingold et al., 2014).

### Case Study—Management

Mrs. Anderson reported a past history of diverticula; therefore, following traditional management, a CT scan was ordered. The patient obtains the CT scan the same

day and the practitioner is notified of the results—diverticulitis of the sigmoid colon. Mrs. Anderson is able to initiate treatment as an outpatient as she lives with her husband and is able to tolerate oral intake well. The practitioner follows traditional management and orders antibiotic therapy and a low-residue diet. After a follow-up telephone call to Mrs. Anderson the next day, she reports that her fever has subsided and her lower abdominal pain is improving. She is referred to a gastroenterology provider for follow-up.

## Conclusion

Diverticular disease is a complex disorder that can be challenging to diagnose and manage. Recent studies have changed the paradigm of diverticular disease and questioned traditional dogma. However, further research studies are necessary to determine the most effective prevention strategies and management of diverticular disease. It is essential for practitioners to be aware of the signs and symptoms of diverticular disease and the latest research on the prevention, diagnosis, and management for the best patient outcomes. 🌟

## REFERENCES

Aldoori, W. H., Giovannucci, E. L., Rimm, E. B., Ascherio, A., Stampfer, M. J., Colditz, G. A., ... Willett, W. C. (1995). Prospective study of physical activity and the risk of symptomatic diverticular disease in men. *Gut*, *36*, 276–282.

Aldoori, W. H., Giovannucci, E. L., Rockett, H. R. H., Sampson, L., Rimm, E. B., & Willett, W. C. (1998). A prospective study of dietary fiber types and symptomatic diverticular disease in men. *The Journal of Nutrition*, *128*(4), 714–719.

Biondo, J., Borao, L., Millan, M., Kreisler, E., & Jaurieta, E. (2011). Current status of the treatment of acute colonic diverticulitis: A systematic review. *Colorectal Disease*, *14*(1), e1–e11. doi:10.1111/j.1463-1318.2011.02766.x

Boynton, W., & Floch, M. (2013). New strategies for the management of diverticular disease: Insights for the clinician. *Therapeutic Advances in Gastroenterology*, *6*(3), 205–213. doi:10.1177/1756283x13478679

Chabok, A., Pahlman, L., Hjern, F., Haapaniemi, S., & Smedh, K. (2012). Randomized clinical trial of antibiotics in acute uncomplicated diverticulitis. *British Journal of Surgery*, *99*(4), 532–539.

Crowe, F., Appleby, P. N., Allen, N. E., & Key, T. J. (2011). Diet and risk of diverticular disease In Oxford cohort of European Prospective Investigation into Cancer and Nutrition (EPIC): Prospective of British vegetarians and non-vegetarians. *British Medical Journal*, *343*(d4131). Retrieved from doi:10.1136/bmj.d4131

de Korte, N., Ünlü, C., Boermeester, M. A., Cuesta, M. A., Vrouenreats, B. C., & Stockmann, H. B. (2011). Use of antibiotics in uncomplicated diverticulitis. *British Journal of Surgery*, *98*, 761–767.

Etzioni, D. A., Mack, T. M., Beart, R. W., & Kaiser, A. M. (2009). Diverticulitis in the United States: 1998–2005: Changing patterns of disease and treatment. *Annals of Surgery*, *249*(2), 210–217.

Feingold, D., Steele, S. R., Lee, S., Kaiser, A., Boushey, R., Buie, W.D., & Rafferty, J. F. (2014). Practice parameters for the treatment of sigmoid diverticulitis. *Diseases of the Colon & Rectum*, *57*(3), 284–294. doi:10.1097/DCR.000000000000075

Fong, S. S., Tan, E. Y., Foo, A., Sim, R., & Cheong, D. M. O. (2011). The changing trend of diverticular in a developing Nation. *Colorectal Disease*, *13*(3), 312–316. doi:10.1111/j.1463-1318.2009.02121.x

Gaglia, A., & Probert, C. S. (2015). Diverticular disease. *Medicine*, *43*(6), 320–323.

Hall, J. F., Roberts, P. L., Ricciardi, R., Read, T., Scheirey, C., Wald, C., ... Schoetz, D. J. (2011). Long-term follow-up after an initial episode of diverticulitis: What are the predictors of recurrence? *Diseases of the Colon & Rectum*, *54*(3), 283–288. doi:10.1007/DCR.0b013e3182028576

Hughes, L. E. (1969). Postmortem survey of diverticular disease of the colon: Part 1 diverticulosis and diverticulitis. *Gut*, *10*(5), 336–344.

Jackson, J. D., & Hammond, T. (2014). Systematic review: Out-patient management of acute uncomplicated diverticulitis. *International Journal of Colorectal Disease*, *29*(7), 775–781. doi:10.1007/s00384-014-1900-4

Jafri, M., Sadiq, J. N., Kamran, H., Rana, S. A., Bhanvadia, A., & Gress, F. G. (2013). Efficacy of mesalamine in the treatment of symptomatic uncomplicated diverticular disease (SUDD): Systemic review and meta analysis. *Gastroenterology*, *114*(5), S783–S784. doi:http://dx.doi.org/10.1016/S0016-5085(13)62901-7

Lutwak, N., & Dill, C. (2013). Mild to moderate diverticulitis: What's new in diagnostic approach, treatment, and prevention of recurrence? *Consultant 360*, *21*(7). Retrieved from http://www.consultant360.com/articles/mild-moderate-diverticulitis-what-s-new-diagnostic-approach-treatment-and-prevention

Maconi, G., Monteleone, M., Bezzio, C., Furfaro, F., & de Franchis, R. (2013). Diverticular disease of the colon: Antibiotics or probiotics? *International Journal of Probiotics and Prebiotics*, *8*(1), 33–40.

Martin, S. T., & Stocchi, L. (2011). New and emerging treatments for the prevention of recurrent diverticulitis. *Clinical and Experimental Gastroenterology*, *4*, 203–212.

Matrana, M. R., & Margolin, D. A. (2009). Epidemiology and pathophysiology of diverticular disease. *Clinics in Colon and Rectal Surgery*, *22*(3), 141–146. doi:10.1055/s-0029-1236157

Mehta, T. A., & Probert, C. S. (2011). Diverticular disease. *Medicine*, *39*(5), 265–268.

Moorman, S. (2012). Help patients defy diverticular disease. *Journal of Christian Nursing*, *29*(2), 82–87. doi:10.1097/CNJ.0b013e318245cfd2

Morris, A. M., Regenbogen, S. E., Hardiman, K. M., & Hendren, S. (2014). Sigmoid diverticulitis: A systematic review. *Journal of American Medical Association*, *311*(3), 287–297.

Mosadeghi, S., Bhuket, T., & Stollman, N. (2015). Diverticular disease: Evolving concepts in classification, presentation, and management. *Current Opinion in Gastroenterology*, *31*(1), 50–55. doi:10.1097/MOG.0000000000000145

Painter, N. S., & Burkitt, D. P. (1971). Diverticular disease of the colon: A deficiency disease of Western civilization. *British Medical Journal*, *2*(5759), 450–454.

Peery, A. F., Barrett, P. R., Park, D., Rogers, A. J., Galanko, J. A., Martin, C. F., & Sandler, R. S. (2012a). A high-fiber diet does not protect against asymptomatic diverticulosis. *Gastroenterology*, *142*(2), 266–272. doi:10.1053/j.gastro.2011.10.035

- Peery, A. F., Dellon, E. S., Lund, J., Crockett, S.D., McGowan, C.E., Bulsiewicz, W. J., & Shaheen, N. J. (2012b). Burden of gastrointestinal disease in the United States: 2012 update. Burden of gastrointestinal disease in the United States: 2012 update. *Gastroenterology*, *143*(5), 1179–1187.
- Peery, A. F., & Sandler, R. S. (2013). Diverticular disease: Reconsidering conventional wisdom. *Perspectives in Clinical Gastroenterology and Hepatology*, *11*, 1532–1537.
- Pilgrim, S. M., Hart, A. R., & Speakman, C. T. M. (2013). Diverticular disease in younger patients—is it clinically more complicated and related to obesity? *Journal of Colorectal Disease*, *15*(10), 1205–1210. doi:10.1111/codi.12225
- Shabanzadeh, D. M., & Wille-Jørgensen, P. (2012). Antibiotics for uncomplicated diverticulitis. *Cochrane Database of Systemic Reviews*, *11*, CD009092. doi:10.1002/14651858.CD009092.pub2
- Shahedi, K., Fuller, G., Bolus, R., Snyder, B. J., Cohen, E. R., Vu, M., ... Spiegel, B. (2012). Progression from incidental diverticulosis to acute diverticulitis. *Gastroenterology*, *142*(5), S144. doi:http://dx.doi.org/10.1016/S0016-5085(12)60541-1
- Stollman, N. H., & Raskin, J. B. (1999). Practice guidelines: Diagnosis and management of diverticular disease of the colon in adults. *The American Journal of Gastroenterology*, *94*(11), 3110–3121. doi:10.1111/j.1572-0241.1999.01501.x
- Strate, L. L. (2012). Lifestyle factors and the course of diverticular disease. *Digestive Diseases*, *30*(1), 35–45. doi:10.1159/000335707
- Symeonidis, N., Psarras, K., Lalountas, M., Baltatzis, M., Micha, A., Pavlidis, E., & Sakantamis, A. (2011). Clinical features of colonic diverticular disease. *Techniques in Coloproctology*, *15*(Suppl. 1), S5–S8.
- Touzios, J. G., & Dozois, E. J. (2009). Diverticulosis and acute diverticulitis. *Gastroenterology Clinics of North America*, *38*(3), 513–525. doi:10.1016/j.gtc.2009.06.004
- Tursi, A. (2015). The role of colonoscopy in managing diverticular disease of the colon. *Journal of Gastrointestinal and Liver Diseases*, *24*(1), 85–93. doi:10.15403/jgld.2014.1121.tur
- Tursi, A., & Danese, S. (2014). Preventing diverticulitis recurrence by selecting the right therapy for a complex disease. *Gastroenterology*, *147*(4), 733–736. doi:http://dx.doi.org/10.1053/j.gastro.2014.08.022
- Tursi, A., & Papagrigoriadis, S. (2009). Review article: The current and evolving treatment of colonic diverticular disease. *Alimentary Pharmacology Therapeutics*, *30*(6), 532–546. doi:10.1111/j.1365-2036.2009.04072.x
- Weizman, A. V., & Nguyen, G. C. (2011). Diverticular disease: Epidemiology and management. *Canadian Journal of Gastroenterology*, *25*(7), 385–389.
- Wess, L., Eastwood, M. A., Wess, T. J., Busuttill, A., & Miller, A. (1995). Cross linking of collagen is increased in colonic diverticulosis. *Gut*, *37*(1), 91–94.
- West, A. B. (2006). The pathology of diverticulosis: Classical concepts and mucosal changes in diverticula. *Journal of Clinical Gastroenterology*, *40*(Suppl. 3), S126–S131.
- Williams, P. T. (2009). Incident diverticular disease is inversely related to vigorous physical activity. *Medicine and Science in Sports and Exercise*, *41*, 1042–1047.
- World Gastroenterology Organisation. (2007). *World Gastroenterology Organisation Practice Guidelines: Diverticular disease*. Retrieved from [http://www.worldgastroenterology.org/assets/downloads/en/pdf/guidelines/07\\_diverticular\\_disease.pdf](http://www.worldgastroenterology.org/assets/downloads/en/pdf/guidelines/07_diverticular_disease.pdf)
- Ye, E., Chacko, S., Chou, E., Kugizaki, M., & Liu, S. (2012). Greater whole grain intake is associated with lower risk of Type 2 diabetes, cardiovascular disease, and weight gain. *The Journal of Nutrition*, *142*(7), 1304–1313. doi:10.3945/jn.111.155325

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