Colonic diverticulosis and diverticular disease: Epidemiology, risk factors, and pathogenesis

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INTRODUCTION

Diverticular disease of the colon is an important cause of hospital admissions and a significant contributor to health care costs in Western and industrialized societies [1,2].

This topic will review the epidemiology, risk factors, and the pathogenesis of diverticulosis and diverticular disease. The clinical manifestations, diagnosis, and treatment of diverticulitis, diverticular bleeding, and segmental colitis associated with diverticula (diverticular colitis) are discussed in detail, separately. (See "Clinical manifestations and diagnosis of acute colonic diverticulitis in adults" and "Acute colonic diverticulitis: Medical management" and "Colonic diverticular bleeding" and "Segmental colitis associated with diverticulosis".)

TERMINOLOGY

- Diverticulosis is defined by the presence of diverticula, which are sac-like protrusions of the colonic mucosa through weak points in the muscular wall (figure 1). Diverticulosis may be asymptomatic or symptomatic.

- Diverticular disease is defined as clinically significant and symptomatic diverticulosis due to diverticulitis or its complications, diverticular bleeding, segmental colitis associated with diverticula, or symptomatic uncomplicated diverticular disease.

DIVERTICULOSIS

Epidemiology

Prevalence — The prevalence of diverticulosis is age-dependent, and the prevalence and distribution within the colon varies by geography and race [3]. In the United States, the prevalence of diverticulosis increases from fewer than 20 percent at age 40 to 60 percent by age 60 (image 1) [4]. The number and size of diverticula also increase with age, suggesting that diverticulosis is a progressive process [4]. The prevalence of diverticulosis appears to be higher in the left colon in countries in the West. In a study of 624 individuals undergoing first-time screening colonoscopy of whom 260 (42 percent) had diverticulosis, 72 percent had diverticula in the sigmoid colon, 10 percent in the descending, 6 percent in the transverse, 11 percent in the ascending, and 1 percent in the cecum. Black Americans were more likely to have diverticula in the proximal colon than White Americans. Data from more than 270,000 colonoscopy procedures indicate that females are less likely to have diverticulosis than males, particularly in younger age groups [5]. Non-Hispanic White Americans were more likely to have diverticulosis than non-Hispanic Black Americans and Asian/Pacific Islanders. Non-Hispanic Black Americans were at increased odds of having any proximal diverticulosis and Asian/Pacific Islanders were at increased odds of having only proximal diverticulosis, which are often congenital in nature.

Contemporary studies of the prevalence of diverticulosis in other countries are limited. In one series that included 302 patients, the prevalence of diverticulosis in Nigeria was 9.4 percent [6]. Prevalence rates in studies performed in Southeast Asia range from 12 percent in Korea, to 25 percent in Japan, and 29 percent in Thailand [6,7]. In Asia, diverticulosis is predominantly **right-sided** [8].

Time trends — The prevalence of diverticulosis has increased both in the Western hemisphere and in countries that have adopted a more Western lifestyle. As an example, Japan has experienced an increase in the prevalence of right-sided diverticulosis similar to the increase in left-sided diverticula in westernized countries [9,10].

Risk factors — In well-characterized, cross-sectional cohorts of patients undergoing screening colonoscopy, older age, male sex, smoking, and elevated body mass index have been identified as risk factors for diverticulosis [4,11]. White race was also a risk factor for diverticulosis [5]. Contrary to popular belief, low dietary fiber intake and constipation were not associated with increased risk of diverticulosis found on colonoscopy [3,11]. Dietary red meat and fat, alcohol consumption, and physical inactivity also do not appear to contribute to the risk of diverticulosis.

Pathogenesis — The pathogenesis of diverticulosis and its complications have not been clearly elucidated. Several theories exist that are supported largely by indirect evidence. These theories are evolving over time and build on advancements in genetics and the gut microbiome. Diverticula develop at well-defined points of weakness, which correspond to where the vasa recta penetrate the circular muscle layer of the colon (figure 1) [12]. In the West, most colonic diverticula are acquired and are "false" or pulsion diverticulum, in which mucosa and submucosa herniate through the muscle layer, covered only by serosa. In Asia, congenital diverticula are common, tend to be in the right colon, and include all layers of the colon wall.

Abnormal colonic motility is hypothesized to be an important predisposing factor in the development of diverticula [13,14]. Patients with diverticulosis have exaggerated segmentation contractions in which segmental muscular contractions separate the lumen into chambers (image 2). This may result in an increase in intraluminal pressure that predisposes to herniation of mucosa and submucosa. However, studies have also shown that constipation and a low fiber diet are not associated with asymptomatic diverticulosis. The neural basis for the abnormal motility observed in patients with diverticulosis remains unclear, although one report found that a central event appeared to be upregulation of smooth muscle M3 receptors [13].

There are also structural changes in collagen that are similar to, but greater in magnitude than, those that occur because of aging [15]. Structural changes in the wall may also be responsible for the appearance of diverticula at an early age in connective tissue disorders such as Ehlers-Danlos and Marfan's syndromes and in autosomal dominant polycystic kidney disease [16]. Genome-wide association studies have identified susceptibility loci in areas with genes associated with intestinal motility and enriched for expression in connective tissue cell types [17]. (See "Clinical manifestations and diagnosis of Ehlers-Danlos syndromes" and "Genetics, clinical features, and diagnosis of Marfan syndrome and related disorders" and "Autosomal dominant polycystic kidney disease (ADPKD): Extrarenal manifestations".)

DIVERTICULITIS

Diverticulitis is defined as inflammation of one or several adjacent diverticula. Diverticulitis may be uncomplicated or complicated by a diverticular abscess, fistula, bowel obstruction, or free perforation. Smoldering diverticulitis is an uncommon presentation of diverticulitis defined as diverticulitis that persists or relapses shortly after treatment. (See "Clinical manifestations and diagnosis of acute colonic diverticulitis in adults".)

Incidence — Approximately 4 percent of patients with diverticulosis develop diverticulitis [18]. The incidence of diverticulitis increases with age. The mean age at admission for acute

diverticulitis is 63 years [19]. While the incidence of acute diverticulitis is lower in younger individuals, approximately 16 percent of admissions for acute diverticulitis are in patients under 45 years of age [20]. Diverticulitis is predominantly left-sided in western countries, with right-sided diverticulitis being present in only 1.5 percent of cases [21]. In the United States, diverticulitis is more common in non-Hispanic Whites than in non-Hispanic Blacks, and is lowest in Asians [22].

The incidence of diverticulitis is increasing. A nationwide inpatient study of hospitalizations in the United States showed an increase in admissions for acute diverticulitis of 26 percent from 1998 to 2005 [19], although in another nationwide study, prevalence appeared to plateau after 2008 [22]. The largest increase was in patients aged 18 to 44 years (82 percent). Elective operations for diverticulitis also increased by 29 percent with the largest increase in patients aged 18 to 44 years (73 percent). Under age 50 years, diverticulitis is more common in men; there is a slight female preponderance between the ages of 50 and 70, and a marked female preponderance over age 70 [22-26].

Risk factors

Lifestyle factors — Several lifestyle factors have been associated with risk of diverticulitis including diet, lack of physical activity, body mass index, and smoking. A prospective cohort study evaluated the association between lifestyle factors and the risk of incident diverticulitis in over 51,000 males aged 40 to 75 years at study baseline [27]. The risk of diverticulitis decreased incrementally with the number of low-risk lifestyle factors (fewer than four servings of red meat/week, at least 23 g/day of dietary fiber, BMI 18.5 to 25, at least two hours/week of vigorous physical activity, and never-smoker). Males who adhered to all five low-risk factors were 73 percent less likely to develop diverticulitis, and 50 percent of the population attributable risk of diverticulitis was due to these lifestyle factors.

Diet — Dietary factors that increase the risk of diverticulitis include high intake of red meat alone and a Western dietary pattern (high in red meat, fat, and refined grains). In a large, prospective study of men, those in the highest quintile of Western dietary pattern score were 55 percent more likely to develop incident diverticulitis than those in the lowest quintile after adjustment for multiple confounders [28]. Similarly, males who consumed the most red meat had a 58 percent higher risk of diverticulitis. The risk plateaued after six servings per week and substitution of poultry or fish for red meat decreased risk [29]. In contrast, high intake of dietary fiber, and a prudent dietary pattern (high in fruits, vegetables, and whole grains) and a vegetarian diet have been associated with decreased risk of incident diverticulitis [28,30-32]. The inflammatory potential of diet and circulating levels of inflammatory markers have also been associated with an increase in risk of subsequent diverticulitis, and changes in the gut

microbiome are seen in individuals who have had diverticulitis [33-35]. Together these findings suggest that diet acts through the gut microbiome and chronic inflammation to contribute to diverticulitis risk.

Nut, corn, and popcorn consumption are **not** associated with an increase in risk of diverticulitis. In a large prospective cohort study that included 47,228 males, there was an inverse association between nut and popcorn consumption and risk of incident diverticulitis in highest to lowest consumption comparisons (hazard ratio [HR] nuts 0.8, 95% CI 0.63-1.01; HR popcorn 0.72, 95% CI 0.56-0.92) [36]. No association was found between consumption of corn and diverticulitis or between nut, popcorn, or corn consumption and diverticular bleeding or uncomplicated diverticulosis. (See "Acute colonic diverticulitis: Medical management", section on 'Recurrent diverticulitis'.)

Physical activity — Physical activity, particularly vigorous activity, decreases the risk of diverticulitis [37-39]. In a prospective study of males, those in the highest category of physical activity had an adjusted HR of 0.75 for diverticulitis when compared with those in the lowest category; for vigorous activity the HR was 0.66 and jogging/running specifically was associated with a decreased risk [37].

Obesity — Obesity is a strong and consistent risk factor for diverticulitis. Large, prospective cohorts studies as well as case-control studies have found that body mass index (BMI) is positively associated with risk of diverticulitis [38,40,41], with a relative risk ranging from 1.3 to 4.4 for a BMI in the highest category (eg, >30 kg/m^2) compared with the lowest. Central obesity and weight gain have also been shown to be risk factors for diverticulitis [40,41].

Smoking and alcohol — Smoking increases the risk of diverticulitis, particularly for perforated diverticulitis [42-44]. Heavy alcohol use is also associated with diverticulitis [45]. However, it is uncertain whether smaller amounts of alcohol use increase the risk of diverticulitis.

Medications

Immunosuppression — Corticosteroids increase the risk of diverticulitis and particularly of complicated diverticulitis. In a large case-control study, corticosteroids were associated with a threefold increased risk of diverticular perforation (odds ratio [OR] 2.74, 95% CI 1.63-4.61) [46]. Patients who are immunocompromised for other reasons, such as after organ transplant or while receiving chemotherapy, are also at risk of complications, but the risks are less well-defined due to small sample sizes and the heterogeneity in immunosuppressive agents in available studies [47,48].

Other — Nonsteroidal anti-inflammatory drugs, menopausal hormone therapy, and opiates are associated with an increased risk of diverticulitis [46,49-51]. In a large case-control study, higher levels of vitamin D were associated with a reduced risk of hospitalization for diverticulitis [52]. The risk of hospitalization for diverticulitis decreased with increasing vitamin D levels (adjusted relative risk [RR] highest versus lowest quintile of 25 hydroxy vitamin D levels 0.49, 95% CI 0.38-0.62). Another study found that ultraviolet (UV) light exposure was negatively associated with risk of diverticulitis presumed to be related to vitamin D levels given that UV exposure is the major contributor to vitamin D status [53]. Studies evaluating the risk of diverticular perforation and use of statins have been conflicting [46,54].

Family history/genetics — Genetic susceptibility may also contribute to the risk of diverticulitis. Two population-based twin studies in Scandinavia found the risk of diverticulitis in a twin whose co-twin had diverticulitis was higher in monozygotic twins (14.5, 95% CI 8.9-23) than dizygotic twins (5.5, 95% CI, 3.3-8.6). Non-twin siblings of an individual with diverticulitis were three times more likely to have diverticulitis than the general population (2.92, 95% CI 2.50-3.39 [55,56]). These studies estimated that about 50 percent of the tendency to develop diverticulitis was due to inherited factors. Genome-wide association studies have identified loci that associate with diverticular disease. Genes in these loci are related to immunity, the extracellular matrix, cell adhesion, membrane transport, and intestinal motility [17].

Pathogenesis — The underlying cause of diverticulitis is unknown. It was previously believed that obstruction of diverticula (eg, by fecaliths) increased diverticular pressure and caused perforation. However, such obstruction is now thought to be rare (picture 1) [57]. One theory posits that erosion of the diverticular wall by increased intraluminal pressure or inspissated food particles leads to inflammation, focal necrosis, and micro or macro perforation. This process is similar to what occurs in appendicitis. However, a study of nut, corn, and popcorn consumption, once felt to be the particles responsible for diverticular injury, found no increased risk of diverticulitis in individuals who consume the highest amount of these foods as compared with those who consumed them infrequently [36]. (See "Clinical manifestations and diagnosis of acute colonic diverticulitis in adults" and "Acute colonic diverticulitis: Medical management".)

Newer theories suggest that changes in the microbiome and chronic inflammation are important factors in the development of diverticulitis and there is growing evidence to support them. Diseases known to be associated with chronic systemic inflammation, including diabetes and cardiovascular disease, share many risk factors with diverticulitis. Circulating inflammatory markers including C reactive protein (CRP) and interleukin (IL)-6 are elevated in patients who go on to develop diverticulitis when compared with those who do not [33]. In addition, the

composition and function of the gut microbiome in patients with diverticulitis is different than those who have not had diverticulitis [34,58,59].

DIVERTICULAR BLEEDING

Diverticular bleeding is characterized by painless hematochezia due to luminal rupture of the vasa recta associated with a diverticulum. (See "Colonic diverticular bleeding".)

Incidence — The incidence of diverticular bleeding increases dramatically with age and is highest among non-Hispanic Blacks [22]. Among patients with diverticulosis, bleeding occurs in approximately 0.5 per 1000 person-years [60]. In a study of 1514 asymptomatic patients with diverticulosis in Japan, the cumulative incidence of bleeding was 0.2 percent at 12 months, 2.2 percent at 60 months, and 9.5 percent at 120 months [60]. These estimates may be higher than in Western countries where similar estimates are not available. In both Asian and Western populations, the right colon is the source of colonic diverticular bleeding in the majority of patients. In a multicenter study of 10,342 patients in Japan, of patients with definitive diverticular bleeding on computed tomography (CT) angiography, 63 percent were located proximal to the splenic flexure [61]. A prospective study of 83 United States patients with definitive diverticular bleeding identified on colonoscopy also found that 63 percent were located proximal to the splenic flexure [62]. A possible explanation for this is that right-sided diverticula have wider necks and domes, exposing a greater length of vasa recta to injury. Another contributing factor may be the thinner wall of the right colon [63].

Risk factors — Nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin, and antiplatelet medications have been consistently identified as risk factors for diverticular bleeding [49,60,64]. In a meta-analysis of six studies, the relative risk for diverticular bleeding in NSAID users was 2.24 (95% CI 1. 63-3.09) and for aspirin users was 1.73 (95% CI 1.31-2.30) [65]. The combination of aspirin or antiplatelet drugs with anticoagulants increases the risk of lower gastrointestinal bleeding compared to the use of single agents alone. Given that diverticular bleeding is the most common cause of lower gastrointestinal bleeding, this drug combination likely also increases the risk of diverticular bleeding. However, it is less clear if anticoagulant medications alone increase the risk of diverticular bleeding [66,67].

Several studies have found that individuals with diverticulosis in the left and right colon are at an increased risk of bleeding [60,66,68]. Hypertension has also been associated with diverticular bleeding in a number of studies from Japan [68-70]. Less is known about diet and lifestyle risk factors for diverticular bleeding than for diverticulitis. In a large prospective cohort study, males with a BMI ≥30 had a threefold higher risk of bleeding as compared with males with a BMI <21

(RR 3.19, 95% CI 1.45-7.0) [40]. In the same cohort, vigorous physical activity was associated with a decreased risk of diverticular bleeding (RR 0.66, 95% CI 0.51-0.86 [37]). In small case control studies, smoking and alcohol have not been associated with diverticular bleeding [68,70,71]. Nut, corn, and popcorn consumption are **not** associated with an increase of diverticular bleeding [36].

Pathogenesis — As a diverticulum herniates, the penetrating vessel responsible for the wall weakness at that point becomes draped over the dome of the diverticulum, separated from the bowel lumen only by mucosa (picture 2) [12]. It is hypothesized that over time, the vasa recta is exposed to injury along its luminal aspect, leading to eccentric intimal thickening and thinning of the media. Hypertension is a risk factor for diverticular bleeding and may also contribute to the vascular injury [69]. These changes may result in segmental weakness of the artery, predisposing to rupture into the lumen. Diverticular bleeding typically occurs in the absence of diverticulitis [12]. (See "Colonic diverticular bleeding", section on 'Clinical manifestations'.)

OTHER DIVERTICULAR DISEASES

Symptomatic uncomplicated diverticular disease and segmental colitis associated with diverticulosis are less well-defined manifestations of diverticular disease.

Symptomatic uncomplicated diverticular disease — Symptomatic uncomplicated diverticular disease (SUDD) is characterized by persistent abdominal pain attributed to diverticula in the absence of macroscopically overt colitis or diverticulitis. The prevalence of SUDD is unknown.

Altered colonic motility may be one of the underlying causes of abdominal pain and constipation in patients with SUDD. In one study, patients with SUDD displayed an increase in duration of rhythmic, low-frequency contractile activity particularly in segments of the colon with diverticula [72]. In another study, patients with diverticulosis were demonstrated to have a significantly reduced density of interstitial cells of Cajal as compared with controls, suggesting that abnormal colonic motility may be the underlying cause of symptoms [73]. SUDD is difficult to differentiate from smoldering diverticulitis and irritable bowel syndrome. Usually, CT scan in patients with smoldering disease reveals ongoing inflammation. Although the data are sparse, in the authors' practice, fecal calprotectin is often elevated in patients with smoldering diverticulitis. Irritable bowel syndrome is common following an episode of diverticulitis perhaps due to visceral hypersensitivity. However, it is controversial whether SUDD is a separate entity from irritable bowel syndrome, particularly in those without prior diverticulitis [74].

It has been hypothesized that visceral hypersensitivity plays an important role in the development of symptoms in patients with diverticulosis who do not have overt diverticulitis. A study compared colonic visceral pain perception in response to luminal distention in patients with SUDD, asymptomatic diverticulosis, and healthy controls. In this study, patients with SUDD but not asymptomatic diverticulosis and healthy controls demonstrated a heightened pain perception both in the sigmoid colon with diverticula and in the unaffected rectum. The mechanism of hypersensitivity in patients with SUDD may relate to increased neuropeptides and alterations in enteric innervation following an episode of diverticulitis [75].

Segmental colitis associated with diverticulosis — Segmental colitis associated with diverticulosis (SCAD) or diverticular colitis is characterized by chronic inflammation involving regions of the left colon that contain diverticulosis. It spares the rectum, which is not susceptible to diverticulosis. Histologically, inflammation involves the interdiverticular mucosa but not the diverticular orifices. It is a rare manifestation of diverticulosis that overlaps significantly with inflammatory bowel disease. The epidemiology, clinical manifestations, diagnosis, and treatment of SCAD are discussed in detail separately. (See "Segmental colitis associated with diverticulosis".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Colonic diverticular disease".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a

variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Diverticulitis (The Basics)")
- Beyond the Basics topics (see "Patient education: Diverticular disease (Beyond the Basics)")

SUMMARY

- **Terminology** – A diverticulum is a sac-like protrusion of the colonic wall. Diverticulosis merely describes the presence of diverticula. These may not be symptomatic or complicated. Diverticular disease is defined as clinically significant and symptomatic diverticulosis. Diverticular symptoms may be due to diverticulitis or diverticular bleeding. Symptomatic uncomplicated diverticular disease, and segmental colitis associated with diverticulosis are less well-defined manifestations of diverticular disease. (See 'Terminology' above.)
- **Epidemiology** – The prevalence of diverticulosis increases with age from less than 20 percent at age 40 to 60 percent by age 60. In the Western hemisphere, diverticulosis is predominantly left-sided. In contrast, in Asia, the prevalence of diverticulosis is lower and diverticulosis is predominantly right-sided. Among patients with diverticulosis, bleeding occurs in approximately 0.5 per 1000 person-years of follow-up, with the right colon being the predominant location of bleeding. Approximately 4 percent of patients with diverticulosis develop diverticulitis.
- **Risk factors**
  - Risk factors for diverticulosis include male sex, White race, smoking, and obesity. Importantly, constipation and low dietary fiber intake do not appear to be associated with an increased risk of diverticulosis.
  - Diet and lifestyle factors are associated with an increased risk of diverticulitis including a dietary pattern that is high in red meat and low in fiber, physical inactivity, and obesity. Avoiding these risk factors as well as smoking may reduce the risk of diverticulitis by as much as 75 percent. Use of certain medications also increases the risk for diverticulitis including aspirin, nonsteroidal anti-inflammatory drugs, opiate analgesics, and immunosuppressive agents. There is no association between nut, corn, and popcorn consumption and the risk of diverticulosis. Constipation is also not a risk factor. (See 'Risk factors' above.)

- Diverticular bleeding is more common in patients who use aspirin and nonsteroidal anti-inflammatory drugs, are obese and physically inactive, and/or have diverticulosis in both the right and left colon.

- **Pathogenesis** – The pathogenesis of diverticulosis and its complications have not been clearly elucidated. Several theories exist that are supported largely by indirect evidence. These theories are evolving over time and currently build on advancements in genetics and the gut microbiome.

  - Diverticula occur at points of weakness in the bowel wall where blood vessels penetrate (figure 1). The development of diverticula is probably multifactorial, involving both abnormalities in motility and connective tissues of the bowel wall. (See 'Diverticulosis' above.)

  - Segmental weakness of the artery in the diverticular wall predisposes to rupture into the lumen, resulting in a diverticular bleed (picture 2). In contrast, the underlying cause of diverticulitis is micro- or macroscopic perforation of the diverticulum itself. The inflammation is frequently mild, and a small perforation is walled off by pericolic fat and mesentery. This may lead to a localized abscess or, if adjacent organs are involved, a fistula or obstruction. Poor containment of the inflamed diverticulum or abscess results in free perforation and peritonitis. (See 'Pathogenesis' above.)

  - Alterations in the gut microbiome and chronic inflammation have been implicated in the pathogenesis of diverticulitis. Visceral hypersensitivity may contribute to abdominal symptoms in individuals following resolution of acute diverticulitis. (See 'Pathogenesis' above.)

  - Altered colonic motility and visceral hypersensitivity may be responsible for the symptoms of symptomatic uncomplicated diverticular disease. (See 'Symptomatic uncomplicated diverticular disease' above.)

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REFERENCES

1. Everhart JE, Ruhl CE. Burden of digestive diseases in the United States part II: lower gastrointestinal diseases. Gastroenterology 2009; 136:741.
2. Shaheen NJ, Hansen RA, Morgan DR, et al. The burden of gastrointestinal and liver diseases, 2006. Am J Gastroenterol 2006; 101:2128.
3. Peery AF, Barrett PR, Park D, et al. A high-fiber diet does not protect against asymptomatic diverticulosis. Gastroenterology 2012; 142:266.
4. Peery AF, Keku TO, Martin CF, et al. Distribution and Characteristics of Colonic Diverticula in a United States Screening Population. Clin Gastroenterol Hepatol 2016; 14:980.
5. Peery AF, Keku TO, Galanko JA, Sandler RS. Sex and Race Disparities in Diverticulosis Prevalence. Clin Gastroenterol Hepatol 2020; 18:1980.
6. Alatise OI, Arigbabu AO, Agbakwuru EA, et al. Spectrum of colonoscopy findings in Ile-Ife Nigeria. Niger Postgrad Med J 2012; 19:219.
7. Yamamichi N, Shimamoto T, Takahashi Y, et al. Trend and risk factors of diverticulosis in Japan: age, gender, and lifestyle/metabolic-related factors may cooperatively affect on the colorectal diverticula formation. PLoS One 2015; 10:e0123688.
8. Reichert MC, Lammert F. The genetic epidemiology of diverticulosis and diverticular disease: Emerging evidence. United European Gastroenterol J 2015; 3:409.
9. Sugihara K, Muto T, Morioka Y, et al. Diverticular disease of the colon in Japan. A review of 615 cases. Dis Colon Rectum 1984; 27:531.
10. Miura S, Kodaira S, Shatari T, et al. Recent trends in diverticulosis of the right colon in Japan: retrospective review in a regional hospital. Dis Colon Rectum 2000; 43:1383.
11. Peery AF, Sandler RS, Ahnen DJ, et al. Constipation and a low-fiber diet are not associated with diverticulosis. Clin Gastroenterol Hepatol 2013; 11:1622.
12. Meyers MA, Alonso DR, Gray GF, Baer JW. Pathogenesis of bleeding colonic diverticulosis. Gastroenterology 1976; 71:577.
13. Golder M, Burleigh DE, Belai A, et al. Smooth muscle cholinergic denervation hypersensitivity in diverticular disease. Lancet 2003; 361:1945.
14. Böttner M, Barrenschee M, Hellwig I, et al. The enteric serotonergic system is altered in patients with diverticular disease. Gut 2013; 62:1753.
15. Wess L, Eastwood MA, Wess TJ, et al. Cross linking of collagen is increased in colonic diverticulosis. Gut 1995; 37:91.
16. Scheff RT, Zuckerman G, Harter H, et al. Diverticular disease in patients with chronic renal failure due to polycystic kidney disease. Ann Intern Med 1980; 92:202.

17. Maguire LH, Handelman SK, Du X, et al. Genome-wide association analyses identify 39 new susceptibility loci for diverticular disease. Nat Genet 2018; 50:1359.
18. Shahedi K, Fuller G, Bolus R, et al. Long-term risk of acute diverticulitis among patients with incidental diverticulosis found during colonoscopy. Clin Gastroenterol Hepatol 2013; 11:1609.
19. Etzioni DA, Mack TM, Beart RW Jr, Kaiser AM. Diverticulitis in the United States: 1998-2005: changing patterns of disease and treatment. Ann Surg 2009; 249:210.
20. Nguyen GC, Sam J, Anand N. Epidemiological trends and geographic variation in hospital admissions for diverticulitis in the United States. World J Gastroenterol 2011; 17:1600.
21. Fischer MG, Farkas AM. Diverticulitis of the cecum and ascending colon. Dis Colon Rectum 1984; 27:454.
22. Wheat CL, Strate LL. Trends in Hospitalization for Diverticulitis and Diverticular Bleeding in the United States From 2000 to 2010. Clin Gastroenterol Hepatol 2016; 14:96.
23. Rodkey GV, Welch CE. Changing patterns in the surgical treatment of diverticular disease. Ann Surg 1984; 200:466.
24. Acosta JA, Grebenc ML, Doberneck RC, et al. Colonic diverticular disease in patients 40 years old or younger. Am Surg 1992; 58:605.
25. Schauer PR, Ramos R, Ghiatas AA, Sirinek KR. Virulent diverticular disease in young obese men. Am J Surg 1992; 164:443.
26. Francis NK, Sylla P, Abou-Khalil M, et al. EAES and SAGES 2018 consensus conference on acute diverticulitis management: evidence-based recommendations for clinical practice. Surg Endosc 2019; 33:2726.
27. Liu PH, Cao Y, Keeley BR, et al. Adherence to a Healthy Lifestyle is Associated With a Lower Risk of Diverticulitis among Men. Am J Gastroenterol 2017; 112:1868.
28. Strate LL, Keeley BR, Cao Y, et al. Western Dietary Pattern Increases, and Prudent Dietary Pattern Decreases, Risk of Incident Diverticulitis in a Prospective Cohort Study. Gastroenterology 2017; 152:1023.
29. Cao Y, Strate LL, Keeley BR, et al. Meat intake and risk of diverticulitis among men. Gut 2018; 67:466.
30. Crowe FL, Appleby PN, Allen NE, Key TJ. Diet and risk of diverticular disease in Oxford cohort of European Prospective Investigation into Cancer and Nutrition (EPIC): prospective study of British vegetarians and non-vegetarians. BMJ 2011; 343:d4131.
31. Ma W, Nguyen LH, Song M, et al. Intake of Dietary Fiber, Fruits, and Vegetables and Risk of Diverticulitis. Am J Gastroenterol 2019; 114:1531.

32. Crowe FL, Balkwill A, Cairns BJ, et al. Source of dietary fibre and diverticular disease incidence: a prospective study of UK women. Gut 2014; 63:1450.
33. Ma W, Jovani M, Nguyen LH, et al. Association Between Inflammatory Diets, Circulating Markers of Inflammation, and Risk of Diverticulitis. Clin Gastroenterol Hepatol 2020; 18:2279.
34. Daniels L, Budding AE, de Korte N, et al. Fecal microbiome analysis as a diagnostic test for diverticulitis. Eur J Clin Microbiol Infect Dis 2014; 33:1927.
35. Mj O, Turner GA, A S, et al. Distinct changes in the colonic microbiome associated with acute diverticulitis. Colorectal Dis 2022; 24:1591.
36. Strate LL, Liu YL, Syngal S, et al. Nut, corn, and popcorn consumption and the incidence of diverticular disease. JAMA 2008; 300:907.
37. Strate LL, Liu YL, Aldoori WH, Giovannucci EL. Physical activity decreases diverticular complications. Am J Gastroenterol 2009; 104:1221.
38. Hjern F, Wolk A, Håkansson N. Obesity, physical inactivity, and colonic diverticular disease requiring hospitalization in women: a prospective cohort study. Am J Gastroenterol 2012; 107:296.
39. Williams PT. Incident diverticular disease is inversely related to vigorous physical activity. Med Sci Sports Exerc 2009; 41:1042.
40. Strate LL, Liu YL, Aldoori WH, et al. Obesity increases the risks of diverticulitis and diverticular bleeding. Gastroenterology 2009; 136:115.
41. Ma W, Jovani M, Liu PH, et al. Association Between Obesity and Weight Change and Risk of Diverticulitis in Women. Gastroenterology 2018; 155:58.
42. Hjern F, Wolk A, Håkansson N. Smoking and the risk of diverticular disease in women. Br J Surg 2011; 98:997.
43. Humes DJ, Ludvigsson JF, Jarvholm B. Smoking and the Risk of Hospitalization for Symptomatic Diverticular Disease: A Population-Based Cohort Study from Sweden. Dis Colon Rectum 2016; 59:110.
44. Aune D, Sen A, Leitzmann MF, et al. Tobacco smoking and the risk of diverticular disease - a systematic review and meta-analysis of prospective studies. Colorectal Dis 2017; 19:621.
45. Tønnesen H, Engholm G, Moller H. Association between alcoholism and diverticulitis. Br J Surg 1999; 86:1067.
46. Humes DJ, Fleming KM, Spiller RC, West J. Concurrent drug use and the risk of perforated colonic diverticular disease: a population-based case-control study. Gut 2011; 60:219.

47. Samdani T, Pieracci FM, Eachempati SR, et al. Colonic diverticulitis in chemotherapy patients: should operative indications change? A retrospective cohort study. Int J Surg 2014; 12:1489.

48. Biondo S, Borao JL, Kreisler E, et al. Recurrence and virulence of colonic diverticulitis in immunocompromised patients. Am J Surg 2012; 204:172.

49. Strate LL, Liu YL, Huang ES, et al. Use of aspirin or nonsteroidal anti-inflammatory drugs increases risk for diverticulitis and diverticular bleeding. Gastroenterology 2011; 140:1427.

50. Aldoori WH, Giovannucci EL, Rimm EB, et al. Use of acetaminophen and nonsteroidal anti-inflammatory drugs: a prospective study and the risk of symptomatic diverticular disease in men. Arch Fam Med 1998; 7:255.

51. Jovani M, Ma W, Joshi AD, et al. Menopausal Hormone Therapy and Risk of Diverticulitis. Am J Gastroenterol 2019; 114:315.

52. Maguire LH, Song M, Strate LE, et al. Higher serum levels of vitamin D are associated with a reduced risk of diverticulitis. Clin Gastroenterol Hepatol 2013; 11:1631.

53. Maguire LH, Song M, Strate LL, et al. Association of geographic and seasonal variation with diverticulitis admissions. JAMA Surg 2015; 150:74.

54. Skajaa N, Schønfeldt Troelsen F, Pedersen L, et al. Statins and risk of diverticular disease: Nested case-control study. Pharmacoepidemiol Drug Saf 2021; 30:770.

55. Granlund J, Svensson T, Olén O, et al. The genetic influence on diverticular disease--a twin study. Aliment Pharmacol Ther 2012; 35:1103.

56. Strate LL, Erichsen R, Baron JA, et al. Heritability and familial aggregation of diverticular disease: a population-based study of twins and siblings. Gastroenterology 2013; 144:736.

57. Rege RV, Nahrwold DL. Diverticular disease. Curr Probl Surg 1989; 26:133.

58. Barbara G, Scaioli E, Barbaro MR, et al. Gut microbiota, metabolome and immune signatures in patients with uncomplicated diverticular disease. Gut 2017; 66:1252.

59. Gueimonde M, Ouwehand A, Huhtinen H, et al. Qualitative and quantitative analyses of the bifidobacterial microbiota in the colonic mucosa of patients with colorectal cancer, diverticulitis and inflammatory bowel disease. World J Gastroenterol 2007; 13:3985.

60. Niikura R, Nagata N, Shimbo T, et al. Natural history of bleeding risk in colonic diverticulosis patients: a long-term colonoscopy-based cohort study. Aliment Pharmacol Ther 2015; 41:888.

61. Nagata N, Kobayashi K, Yamauchi A, et al. Identifying Bleeding Etiologies by Endoscopy Affected Outcomes in 10,342 Cases With Hematochezia: CODE BLUE-J Study. Am J Gastroenterol 2021; 116:2222.

62. Jensen DM, Machicado GA, Jutabha R, Kovacs TO. Urgent colonoscopy for the diagnosis and treatment of severe diverticular hemorrhage. N Engl J Med 2000; 342:78.

63. Imbembo AL, Bailey RW. Diverticular disease of the colon. In: Textbook of Surgery, 14th ed, Sabiston DC Jr (Ed), Churchill Livingstone, 1992. p.910.

64. Nagata N, Niikura R, Aoki T, et al. Colonic diverticular hemorrhage associated with the use of nonsteroidal anti-inflammatory drugs, low-dose aspirin, antiplatelet drugs, and dual therapy. J Gastroenterol Hepatol 2014; 29:1786.

65. Yuhara H, Corley DA, Nakahara F, et al. Aspirin and non-aspirin NSAIDs increase risk of colonic diverticular bleeding: a systematic review and meta-analysis. J Gastroenterol 2014; 49:992.

66. Taki M, Oshima T, Tozawa K, et al. Analysis of risk factors for colonic diverticular bleeding and recurrence. Medicine (Baltimore) 2017; 96:e8090.

67. Turpin M, Gregory P. Direct Oral Anticoagulant Use and Risk of Diverticular Hemorrhage: A Systematic Review of the Literature. Can J Gastroenterol Hepatol 2019; 2019:9851307.

68. Yamada A, Sugimoto T, Kondo S, et al. Assessment of the risk factors for colonic diverticular hemorrhage. Dis Colon Rectum 2008; 51:116.

69. Niikura R, Nagata N, Yamada A, et al. Recurrence of colonic diverticular bleeding and associated risk factors. Colorectal Dis 2012; 14:302.

70. Tsuruoka N, Iwakiri R, Hara M, et al. NSAIDs are a significant risk factor for colonic diverticular hemorrhage in elder patients: evaluation by a case-control study. J Gastroenterol Hepatol 2011; 26:1047.

71. Jansen A, Harenberg S, Grenda U, Elsing C. Risk factors for colonic diverticular bleeding: a Westernized community based hospital study. World J Gastroenterol 2009; 15:457.

72. Bassotti G, Sietchiping-Nzepa F, De Roberto G, et al. Colonic regular contractile frequency patterns in irritable bowel syndrome: the 'spastic colon' revisited. Eur J Gastroenterol Hepatol 2004; 16:613.

73. Bassotti G, Battaglia E, Bellone G, et al. Interstitial cells of Cajal, enteric nerves, and glial cells in colonic diverticular disease. J Clin Pathol 2005; 58:973.

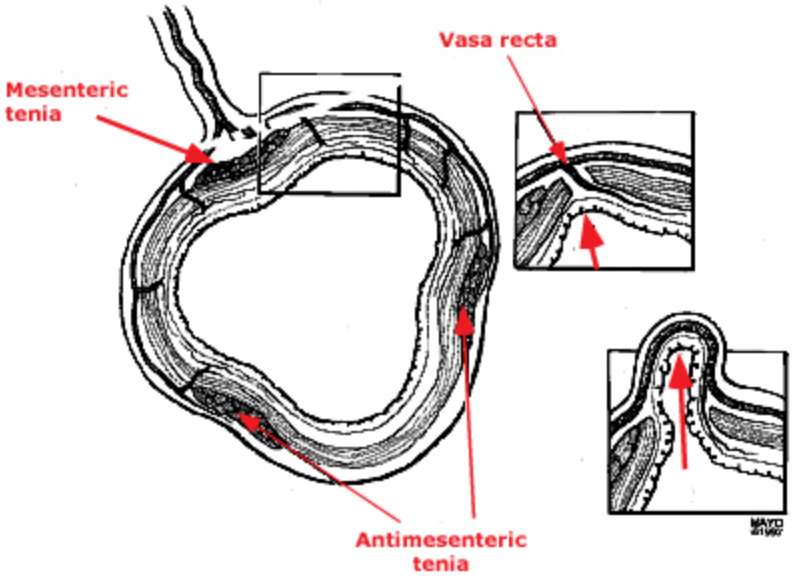
74. Peery AF, Keku TO, Addamo C, et al. Colonic Diverticula Are Not Associated With Mucosal Inflammation or Chronic Gastrointestinal Symptoms. Clin Gastroenterol Hepatol 2018; 16:884.

75. Simpson J, Sundler F, Humes DJ, et al. Post inflammatory damage to the enteric nervous system in diverticular disease and its relationship to symptoms. Neurogastroenterol Motil 2009; 21:847.

Topic 1379 Version 29.0

GRAPHICS

Location of colonic diverticula within the bowel wall



Diverticula develop at four well-defined points around the circumference of the colon, the sites at which the vasa recta penetrate the circular muscle layer. These vessels enter the wall on each side of the mesenteric taenia and on the mesenteric border of the two antimesenteric taeniae. The insets represent the development of a diverticulum at one such point of weakness.

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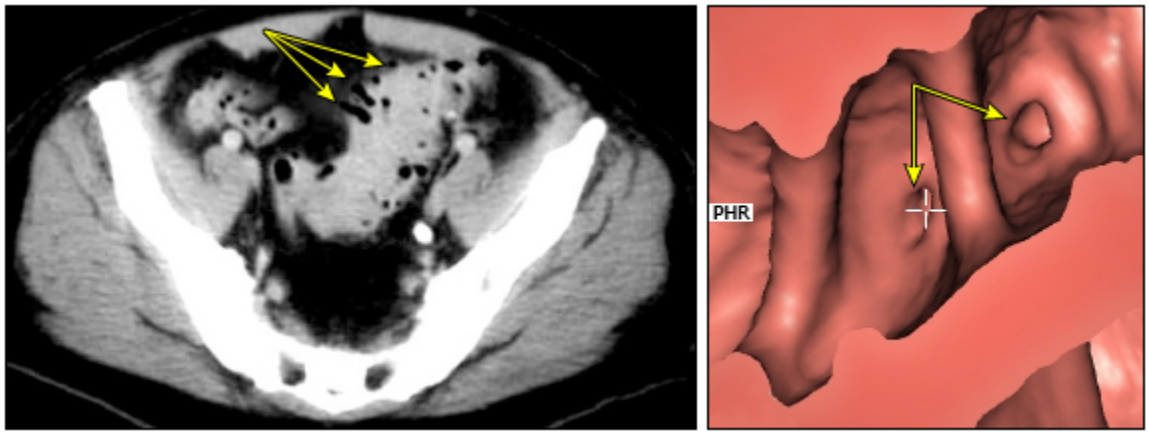
Graphic 62942 Version 4.0

Diverticulosis of sigmoid colon with CT and VC

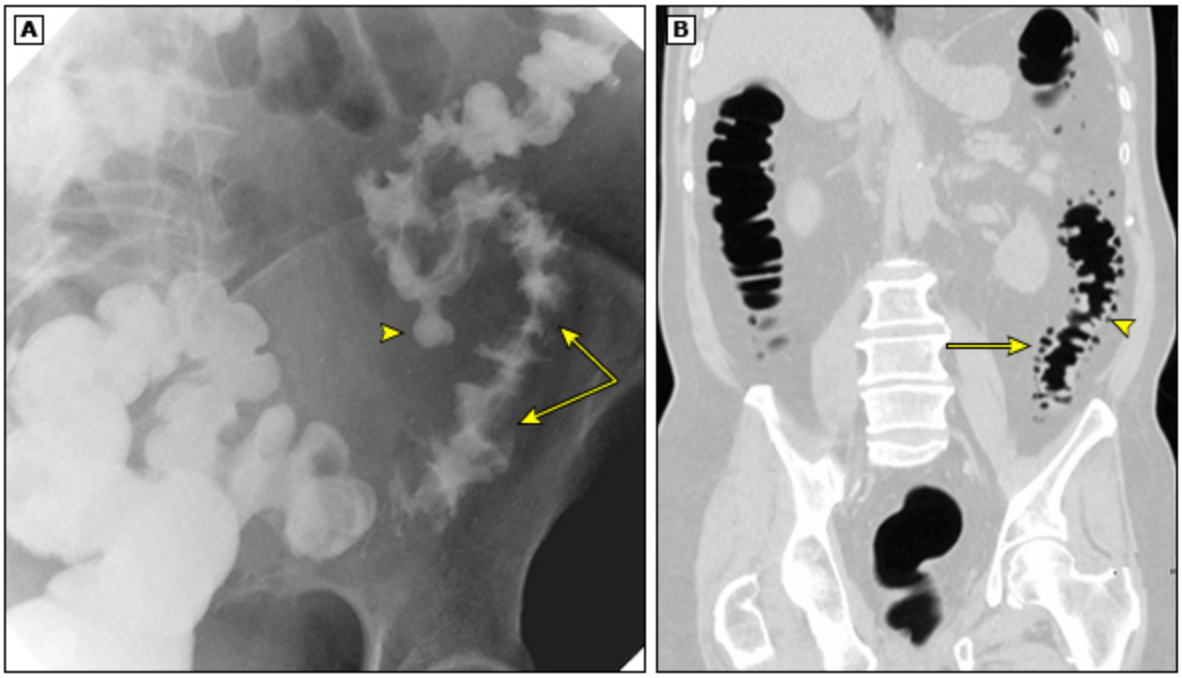


A CT scan through the pelvis (A) shows multiple air-filled diverticula (arrows) of the sigmoid colon. Image B is a virtual colonoscopy of the sigmoid colon showing the necks of sigmoid colon diverticula (arrows) from a luminal perspective.

CT: computed tomography; VC: virtual colonoscopy.

Graphic 91161 Version 2.0

Diverticulosis muscle thickening and narrowing on BE and CT colonography

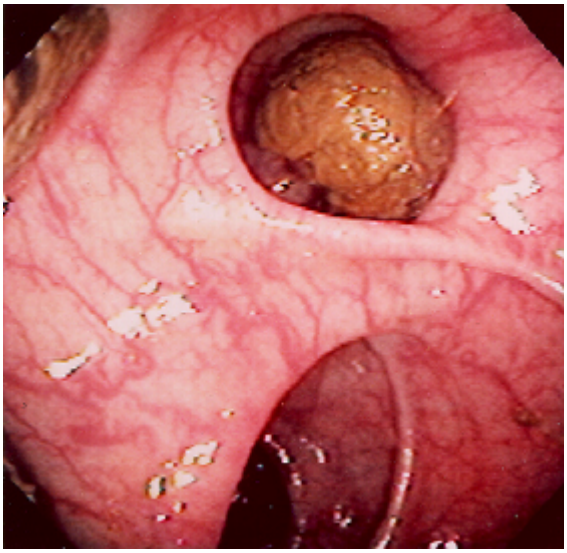


A single contrast barium enema (A) shows diverticulosis (arrowhead) with circular muscle thickening and luminal narrowing of the distal descending colon (double arrow). Source images for a CT virtual colonoscopy reconstructed in the coronal plane show extensive diverticulosis of the descending colon (arrow) with circular muscle thickening and luminal narrowing (arrowhead).

BE: barium enema; CT: computed tomography.

Graphic 91201 Version 2.0

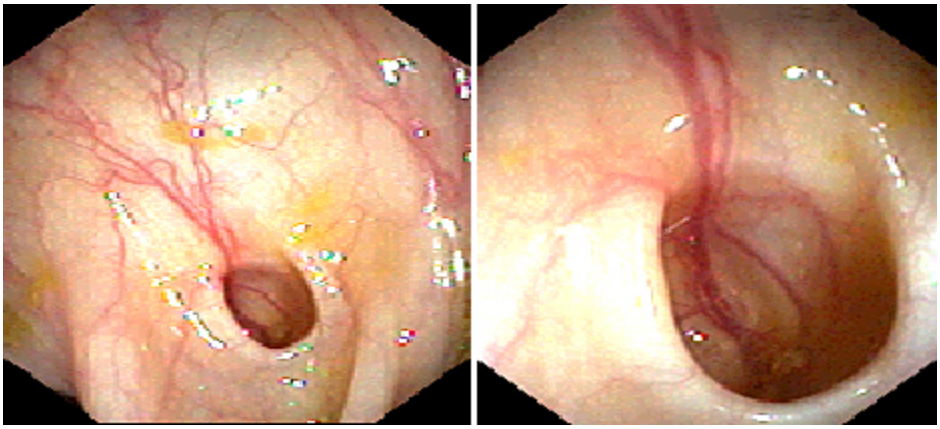
Diverticulum with nonobstructing fecalith



A nonobstructing fecalith is seen within a large diverticulum.

Graphic 80763 Version 3.0

Blood vessel within a colonic diverticulum

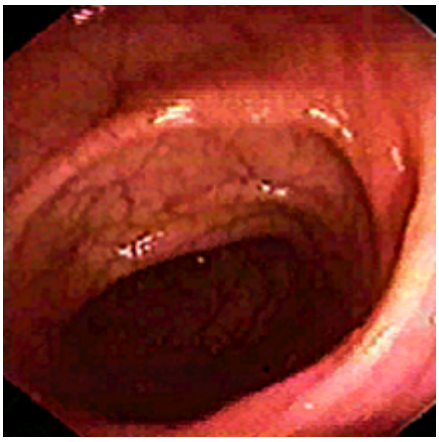


Endoscopy showing a blood vessel within a diverticulum. The blood vessel is separated from the bowel lumen only by mucosa. Over time, the vessel wall is exposed to injury along its luminal aspect, possibly leading to segmental weakness which predisposes to rupture into the lumen.

Courtesy of James B McGee, MD.

Graphic 52254 Version 3.0

Normal sigmoid colon



Endoscopic appearance of the normal sigmoid colonic mucosa. The fine vasculature is easily visible, and the surface is shiny and smooth. The folds are of normal thickness.

Courtesy of James B McGee, MD.

Graphic 55563 Version 1.0

Contributor Disclosures

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