UpToDate® Official reprint from UpToDate®
www.uptodate.com ©2019 UpToDate, Inc. and/or its affiliates. All Rights Reserved.

Wolters Kluwer

Etiology of lower gastrointestinal bleeding in adults

Author: Lisa Strate, MD, MPH
Section Editor: J Thomas Lamont, MD
Deputy Editor: Shilpa Grover, MD, MPH, AGAF

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: Sep 2019. | **This topic last updated:** Jun 04, 2019.

INTRODUCTION

Lower gastrointestinal bleeding (LGIB) refers to blood loss of recent onset originating from a site distal to the ligament of Treitz [1]. It is usually suspected when patients complain of hematochezia (passage of maroon or bright red blood or blood clots per rectum). This is different from the clinical presentation of upper gastrointestinal (GI) bleeding, which includes hematemesis (vomiting of blood or coffee-ground-like material) and/or melena (black, tarry stools). Although helpful, the distinctions based upon stool color are not absolute since melena can be seen with GI bleeding from the right colon (or small intestine), and hematochezia can be seen with massive upper GI bleeding [2-4]. Therefore, it is imperative to exclude a massive upper GI bleed in hemodynamically unstable patients presenting with hematochezia [5]. A nasogastric tube lavage that yields blood or coffee-ground-like material confirms the diagnosis of upper GI bleeding; however, lavage may not be positive if bleeding has ceased or arises beyond a closed pylorus. (See "Approach to acute upper gastrointestinal bleeding in adults".)

The epidemiology of GI bleeding appears to be changing. Studies indicate that the incidence of hospitalizations for LGIB is similar to that of upper GI bleeding, largely due to a decrease in upper GI events [6-8].

This topic review will focus on the major causes of LGIB originating from the colon and briefly summarize the management of some of these disorders. Although the definition of LGIB includes small bowel sources, the clinical presentation, management, and outcomes of small bowel bleeding are generally distinct from bleeding from colon sources [9]. The diagnostic approach to patients with LGIB, suspected small bowel bleeding, and occult gastrointestinal bleeding are discussed separately. (See "Approach to acute lower gastrointestinal bleeding in adults" and "Evaluation of occult gastrointestinal bleeding" and "Evaluation of suspected small bowel bleeding (formerly obscure gastrointestinal bleeding)".)

ETIOLOGY

The causes of lower gastrointestinal bleeding (LGIB) may be grouped into several categories (table 1):

- Anatomic (diverticulosis)
- Vascular (angiodysplasia, ischemic, radiation-induced)
- Inflammatory (inflammatory bowel disease, infectious)
- Neoplastic

In most series, diverticulosis is the most common source of LGIB, accounting for approximately 15 to 55 percent of cases [10,11]. Angiodysplasia may be the most frequent cause in patients over the age of 65 years [12,13], though more recent data suggest that angiodysplasia may be a less common cause of LGIB than once thought [11]. (See "Angiodysplasia of the gastrointestinal tract".)

Hemorrhoids are the most common cause of rectal bleeding in patients under the age of 50 years [14]. However, hemorrhoidal bleeding is usually minor. Similarly, bloody diarrhea due to inflammatory causes can sometimes be distinguished from other causes of LGIB because of the clinical setting. In general, anatomic and vascular causes of bleeding present with painless, large-volume blood loss, whereas inflammatory sources are associated with diarrhea and abdominal pain [15].

In a review of several large studies that included 1559 patients with acute hematochezia, the following bleeding sources were identified [11]:

- Diverticulosis – 5 to 42 percent
- Ischemia – 6 to 18 percent
- Anorectal (hemorrhoids, anal fissures, rectal ulcers) – 6 to 16 percent
- Neoplasia (polyps and cancers) – 3 to 11 percent
- Angiodysplasia – 0 to 3 percent
- Postpolypectomy – 0 to 13 percent
- Inflammatory bowel disease – 2 to 4 percent
- Radiation colitis – 1 to 3 percent
- Other colitis (infectious, antibiotic associated, ischemic, colitis of unclear etiology) – 3 to 29 percent
- Small bowel/upper GI bleed – 3 to 13 percent
- Other causes – 1 to 9 percent
- Unknown cause – 6 to 23 percent

Diverticulosis — A diverticulum is a sac-like protrusion of the colonic wall. The prevalence of diverticular disease is age-dependent, increasing from less than 20 percent at age 40 to 60

percent by age 60. [16]. (See "Colonic diverticulosis and diverticular disease: Epidemiology, risk factors, and pathogenesis".)

The high prevalence of diverticulosis explains why it is the most common cause of LGIB, even though bleeding is a rare complication of this common disease. Diverticular bleeding typically occurs in the absence of diverticulitis [17], and the risk of bleeding is not further increased if diverticulitis is present [18].

As a diverticulum forms, 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 1) [17]. On histological examination, these vessels demonstrate eccentric intimal thickening and thinning of the media, presumably due to chronic injury along their luminal aspect. These changes may result in segmental weakness of the artery, predisposing to rupture into the lumen. (See "Colonic diverticular bleeding".)

In western countries, 75 percent of diverticula occur on the left side of the colon and, when right-sided diverticula do occur, they are usually associated with left-sided diverticula [19-21]. However, the right colon is the source of diverticular bleeding in 50 to 90 percent of patients [17,22,23]. The anatomic relationship between diverticula and the vasa recta is similar in both the right and left colon, but right-sided diverticula have wider necks and domes. This could expose the vasa recta to injury over a greater length, which may explain the higher incidence of right-sided hemorrhage [17].

Diverticular bleeding may be massive and life-threatening since diverticula often form at the site of arterial vascular penetration. The bleeding is usually painless except for mild abdominal discomfort and cramping due to colonic spasm from intraluminal blood. Diverticular bleeding is self-limited in 70 to 80 percent of cases. However, the long-term rebleeding rate approaches 40 percent after the initial bleeding episode in those who do not undergo surgery [24-27], and appears to be higher in patients found to have definitive diverticular bleeding at colonoscopy [28].

Risk factors for diverticular bleeding include aspirin and nonsteroidal anti-inflammatory drug (NSAID) use, advanced age, obesity, physical inactivity, hypertension, ischemic heart disease, chronic renal insufficiency, and hyperlipidemia [29-39]. Aspirin and NSAIDs may increase the risk of LGIB by a variety of mechanisms, including local erosive topical damage and platelet dysfunction [40].

The management of diverticular bleeding is discussed separately. (See "Colonic diverticular bleeding".)

Angiodysplasia — Angiodysplasia refers to dilated, tortuous submucosal vessels. The walls of these blood vessels are composed of endothelial cells that lack smooth muscle (picture 2 and picture 3). Angiodysplasia appears endoscopically as peripherally expanding dilated capillaries with a central origin typically measuring between 0.1 and 1.0 cm in diameter (picture 4). They are

not visualized by barium enema or at autopsy (since blood volume is removed). (See "Angiodysplasia of the gastrointestinal tract".)

Lower gastrointestinal angiodysplasia is uncommon in the general population. A study of 964 asymptomatic patients undergoing screening colonoscopy found that less than 1 percent had angiodysplasia [41]. No cases of bleeding were identified during three years of follow-up, suggesting that treatment of asymptomatic lesions is not necessary. Nevertheless, similar to diverticulosis, the incidence of angiodysplasia increases with age, likely due to degeneration of the vascular walls [12]. In addition, several conditions have been associated with angiodysplasia [42,43], including aortic stenosis, von Willebrand disease, and chronic renal failure. (See "Angiodysplasia of the gastrointestinal tract", section on 'Conditions associated with angiodysplasia'.)

Angiodysplasia may occur throughout the colon, although bleeding most often originates from the cecum or ascending colon [44]. Similar to diverticular disease, bleeding from angiodysplasia tends to be episodic and self-limited. Blood loss can be overt, presenting with painless hematochezia or melena, but is more often occult, manifested by Hemoccult positive stools and iron deficiency anemia [44,45]. (See "Evaluation of occult gastrointestinal bleeding" and "Evaluation of suspected small bowel bleeding (formerly obscure gastrointestinal bleeding)".)

Bleeding from angiodysplasia is venous in origin (in contrast to arterial bleeding with diverticula) and therefore tends to be less massive than diverticular bleeding. Endoscopic coagulation (with bipolar probe or heater probes), injection sclerotherapy, and argon laser coagulation can all achieve definitive hemostasis in patients with angiodysplasia, but rebleeding may occur [42,46]. (See "Angiodysplasia of the gastrointestinal tract".)

Colitis — Infectious and ischemic colitis and inflammatory bowel disease can all present initially with hematochezia. Mucosal inflammation (colitis) is the common response to acute injury, resulting in activation of the immune system and inflammatory cascade.

The clinical presentation and endoscopic appearance of the various types of colitis can be indistinguishable. Patients can present with abdominal pain, hematochezia (with or without diarrhea), fever, and dehydration. Blood loss tends to be mild. Endoscopically, colitis appears as edema, friability, erythema, and ulceration (picture 5). Histologically, there is evidence of nonspecific acute and chronic inflammation, fibrin exudates, crypt abscesses, and ulceration.

Establishing a specific diagnosis is paramount in the treatment of acute colitis since therapy depends upon the underlying disease process. The diagnosis requires an interpretation of the histologic and gross findings within its clinical context.

Infectious colitis — There are many infectious causes of colitis. A routine stool culture will identify Salmonella, Campylobacter, and Shigella, the three most common causes of bacterial diarrhea in the United States. As noted above, bleeding due to infectious causes can sometimes

be distinguished from other causes of LGIB because of the clinical setting. (See "Approach to the adult with acute diarrhea in resource-rich settings".)

Ischemic colitis — Older adults are most likely to experience ischemia-related colitis because of underlying risk factors such as relative hypotension, heart failure, and arrhythmias. However, often there is no clear precipitating event, and young patients can present with ischemic colitis, particularly those with a hypercoagulable state [47]. Patients classically have associated abdominal pain, although its absence does not preclude the diagnosis. Ischemic colitis tends to be continuous, left-sided (figure 1), and associated with mucosal friability, findings that resemble ulcerative colitis (picture 6). Distinguishing features may include a clear demarcation between involved and normal mucosa, rectal sparing, and a single longitudinal ulcer [48]. Bleeding is self-limited, and most cases (85 to 90 percent) resolve with correction of the underlying cause and volume repletion [49]. This is in contrast to acute mesenteric ischemia of the small intestine, which is often a life-threatening surgical emergency. Nonetheless, patients with LGIB from ischemic colitis tend to have worse outcomes than patients with other sources of bleeding [49]. (See "Colonic ischemia".)

Inflammatory bowel disease — Inflammatory bowel disease refers to both Crohn disease and ulcerative colitis. Hematochezia is a more common initial presentation with the latter and tends to occur in the setting of active inflammation. (See "Clinical manifestations, diagnosis and prognosis of Crohn disease in adults" and "Clinical manifestations, diagnosis, and prognosis of ulcerative colitis in adults".)

Differentiating Crohn disease from ulcerative colitis during an episode of acute LGIB is not imperative since the acute management is similar for both conditions. However, it is important not to misdiagnosis inflammatory bowel disease as ischemic or infectious colitis since the therapy is different [50]. In older adults in particular, it can be difficult to differentiate ischemic colitis from inflammatory bowel disease [51]. (See "Overview of the medical management of mild (low risk) Crohn disease in adults" and "Management of the hospitalized adult patient with severe ulcerative colitis".)

Colon cancer — Colon cancer is a less common but serious cause of hematochezia. It is responsible for approximately 10 percent of cases of rectal bleeding in patients over age 50 years, but is rare in younger individuals [52]. Bleeding occurs as the result of overlying erosion or ulceration. The bleeding tends to be low-grade and recurrent. Bright red blood suggests left-sided lesions; right-sided lesions can present with maroon blood or melena. (See "Clinical presentation, diagnosis, and staging of colorectal cancer".)

Endoscopic therapy of colon cancer presenting with rectal bleeding is limited. There is a significant risk of inducing more bleeding or causing a perforation with endoscopic therapy due to the friability and size of the lesions. The endoscopist should biopsy suspicious masses, look for synchronous

lesions, and exclude additional causes of bleeding. Endoscopically applied TC-325 (Hemospray) is a promising agent for bleeding from diffuse, friable sources such as colon cancer [53].

Rectal bleeding is present in some women with ovarian cancer but is rarely the presenting symptom. (See "Epithelial carcinoma of the ovary, fallopian tube, and peritoneum: Clinical features and diagnosis", section on 'Clinical presentation'.)

Miscellaneous anorectal disorders — Hemorrhoids are dilated submucosal veins in the anus that are located above (internal) or below (external) the dentate line. They are usually asymptomatic but can present with hematochezia, thrombosis, strangulation, or pruritus. Hematochezia results from rupture of internal hemorrhoids, which are supplied by the superior and middle hemorrhoidal arteries. Hemorrhoidal bleeding is almost always painless. Bright red blood typically coats the stool at the end of defecation. Blood may also drip into the toilet or stain toilet paper. Occasionally, bleeding can be copious and distressing for the patient. However, significant lower GI bleeding from hemorrhoids is uncommon (ie, bleeding that causes hemodynamic instability or results in anemia). The risk of serious bleeding is increased in patients with a coagulopathy or on anticoagulant therapy [54]. While hemorrhoids are a common cause of bleeding in patients under the age of 50 years [14], LGIB in adults generally requires endoscopic evaluation to exclude a more serious source. (See "Hemorrhoids: Clinical manifestations and diagnosis".)

A variety of other lesions in the anorectum may be associated with bleeding. These include solitary rectal ulcers, anal fissures, rectal varices, and Dieulafoy's lesions. (See "Solitary rectal ulcer syndrome" and "Anal fissure: Clinical manifestations, diagnosis, prevention" and "Prediction of variceal hemorrhage in patients with cirrhosis", section on 'Location of varices' and "Causes of upper gastrointestinal bleeding in adults", section on 'Dieulafoy's lesion'.)

Radiation telangiectasia or proctitis — Radiation therapy of abdominal and pelvic cancers (such as cervical or prostate carcinoma) can lead to lower gastrointestinal bleeding as either an early or late complication of radiation damage. Risk factors for radiation-induced damage include immobilization of the bowel in the rectosigmoid area, arteriosclerosis, and concomitant chemotherapy.

The timing of rectal bleeding relative to the administration of radiation therapy can narrow the differential diagnosis. Acute radiation injury occurs within six weeks of therapy. Symptoms include diarrhea and rectal urgency or tenesmus, and, uncommonly, bleeding. Chronic radiation proctosigmoiditis has a more delayed onset. The first signs often occur at approximately 9 to 14 months following radiation exposure, but may develop after more than two years in some patients and rarely up to 30 years after exposure. (See "Clinical manifestations, diagnosis, and treatment of radiation proctitis".)

Ulceration or cancer recurrence can also be seen as late complications following radiation therapy. These entities must be excluded in patients with rectal bleeding.

Following biopsy or polypectomy — Bleeding following endoscopic biopsy or polypectomy is usually self-limited, although active arterial bleeding can occur acutely [55]. Acute bleeding is due to involvement of an underlying artery or inadequate coagulation of the polyp stalk. Delayed bleeding can occur as late as three weeks following endoscopic polypectomy, presumably due to sloughing of the coagulated eschar. (See "Management and prevention of bleeding after colonoscopy with polypectomy".)

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: Gastrointestinal bleeding in adults".)

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: Ulcerative colitis in adults (The Basics)" and "Patient education: Crohn disease in adults (The Basics)" and "Patient education: Angiodysplasia of the GI tract (The Basics)")
- Beyond the Basics topics (see "Patient education: Ulcerative colitis (Beyond the Basics)" and "Patient education: Crohn disease (Beyond the Basics)")

SUMMARY

- Lower gastrointestinal bleeding (LGIB) refers to blood loss of recent onset originating from a site distal to the ligament of Treitz, which results in hemodynamic instability, anemia, or the need for blood transfusion.
- The causes of LGIB may be grouped into several categories: anatomic (diverticulosis), vascular (angiodysplasia, ischemic, radiation-induced), inflammatory (inflammatory bowel disease, infectious), and neoplastic (table 1).
- In most series, diverticulosis accounts for approximately 15 to 55 percent of LGIB. (See 'Diverticulosis' above.)

Use of UpToDate is subject to the Subscription and License Agreement.

REFERENCES

1. Zuccaro G Jr. Management of the adult patient with acute lower gastrointestinal bleeding. American College of Gastroenterology. Practice Parameters Committee. Am J Gastroenterol 1998; 93:1202.
2. Jensen DM, Machicado GA. Diagnosis and treatment of severe hematochezia. The role of urgent colonoscopy after purge. Gastroenterology 1988; 95:1569.
3. Zuckerman GR, Trellis DR, Sherman TM, Clouse RE. An objective measure of stool color for differentiating upper from lower gastrointestinal bleeding. Dig Dis Sci 1995; 40:1614.
4. Wilcox CM, Alexander LN, Cotsonis G. A prospective characterization of upper gastrointestinal hemorrhage presenting with hematochezia. Am J Gastroenterol 1997; 92:231.
5. Laine L, Shah A. Randomized trial of urgent vs. elective colonoscopy in patients hospitalized with lower GI bleeding. Am J Gastroenterol 2010; 105:2636.
6. Laine L, Yang H, Chang SC, Datto C. Trends for incidence of hospitalization and death due to GI complications in the United States from 2001 to 2009. Am J Gastroenterol 2012; 107:1190.
7. Lanas A, García-Rodríguez LA, Polo-Tomás M, et al. Time trends and impact of upper and lower gastrointestinal bleeding and perforation in clinical practice. Am J Gastroenterol 2009; 104:1633.
8. Peery AF, Crockett SD, Murphy CC, et al. Burden and Cost of Gastrointestinal, Liver, and Pancreatic Diseases in the United States: Update 2018. Gastroenterology 2019; 156:254.

9. Prakash C, Zuckerman GR. Acute small bowel bleeding: a distinct entity with significantly different economic implications compared with GI bleeding from other locations. Gastrointest Endosc 2003; 58:330.

10. Zuckerman GR, Prakash C. Acute lower intestinal bleeding. Part II: etiology, therapy, and outcomes. Gastrointest Endosc 1999; 49:228.

11. Strate LL. Lower GI bleeding: epidemiology and diagnosis. Gastroenterol Clin North Am 2005; 34:643.

12. Boley SJ, Sammartano R, Adams A, et al. On the nature and etiology of vascular ectasias of the colon. Degenerative lesions of aging. Gastroenterology 1977; 72:650.

13. Boley SJ, DiBiase A, Brandt LJ, Sammartano RJ. Lower intestinal bleeding in the elderly. Am J Surg 1979; 137:57.

14. Korkis AM, McDougall CJ. Rectal bleeding in patients less than 50 years of age. Dig Dis Sci 1995; 40:1520.

15. Strate LL, Orav EJ, Syngal S. Early predictors of severity in acute lower intestinal tract bleeding. Arch Intern Med 2003; 163:838.

16. Peery AF, Barrett PR, Park D, et al. A high-fiber diet does not protect against asymptomatic diverticulosis. Gastroenterology 2012; 142:266.

17. Meyers MA, Alonso DR, Gray GF, Baer JW. Pathogenesis of bleeding colonic diverticulosis. Gastroenterology 1976; 71:577.

18. Ramanath HK, Hinshaw JR. Management and mismanagement of bleeding colonic diverticula. Arch Surg 1971; 103:311.

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

20. HORNER JL. Natural history of diverticulosis of the colon. Am J Dig Dis 1958; 3:343.

21. Parks TG. Post-mortem studies on the colon with special reference to diverticular disease. Proc R Soc Med 1968; 61:932.

22. Gostout CJ, Wang KK, Ahlquist DA, et al. Acute gastrointestinal bleeding. Experience of a specialized management team. J Clin Gastroenterol 1992; 14:260.

23. Casarella WJ, Kanter IE, Seaman WB. Right-sided colonic diverticula as a cause of acute rectal hemorrhage. N Engl J Med 1972; 286:450.

24. McGuire HH Jr. Bleeding colonic diverticula. A reappraisal of natural history and management. Ann Surg 1994; 220:653.

25. Longstreth GF. Epidemiology and outcome of patients hospitalized with acute lower gastrointestinal hemorrhage: a population-based study. Am J Gastroenterol 1997; 92:419.

26. Liebau H. [Blood pressure response to vasoactive substances and blood pressure regulatory mechanisms in pheochromocytoma]. Arch Klin Med 1968; 215:219.

27. Nagata N, Niikura R, Aoki T, et al. Impact of discontinuing non-steroidal antiinflammatory drugs on long-term recurrence in colonic diverticular bleeding. World J Gastroenterol 2015; 21:1292.

28. Aytac E, Stocchi L, Gorgun E, Ozuner G. Risk of recurrence and long-term outcomes after colonic diverticular bleeding. Int J Colorectal Dis 2014; 29:373.

29. Bjarnason I, Macpherson AJ. Intestinal toxicity of non-steroidal anti-inflammatory drugs. Pharmacol Ther 1994; 62:145.

30. Wilcox CM, Alexander LN, Cotsonis GA, Clark WS. Nonsteroidal antiinflammatory drugs are associated with both upper and lower gastrointestinal bleeding. Dig Dis Sci 1997; 42:990.

31. 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.

32. Chang CH, Lin JW, Chen HC, et al. Non-steroidal anti-inflammatory drugs and risk of lower gastrointestinal adverse events: a nationwide study in Taiwan. Gut 2011; 60:1372.

33. Strate LL, Liu YL, Aldoori WH, Giovannucci EL. Physical activity decreases diverticular complications. Am J Gastroenterol 2009; 104:1221.

34. 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.

35. 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.

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

37. Suzuki K, Uchiyama S, Imajyo K, et al. Risk factors for colonic diverticular hemorrhage: Japanese multicenter study. Digestion 2012; 85:261.

38. Okamoto T, Watabe H, Yamada A, et al. The association between arteriosclerosis related diseases and diverticular bleeding. Int J Colorectal Dis 2012; 27:1161.

39. Nagata N, Niikura R, Aoki T, et al. Lower GI bleeding risk of nonsteroidal anti-inflammatory drugs and antiplatelet drug use alone and the effect of combined therapy. Gastrointest Endosc 2014; 80:1124.

40. Foutch PG. Diverticular bleeding: are nonsteroidal anti-inflammatory drugs risk factors for hemorrhage and can colonoscopy predict outcome for patients? Am J Gastroenterol 1995; 90:1779.

41. Foutch PG, Rex DK, Lieberman DA. Prevalence and natural history of colonic angiodysplasia among healthy asymptomatic people. Am J Gastroenterol 1995; 90:564.

42. Foutch PG. Angiodysplasia of the gastrointestinal tract. Am J Gastroenterol 1993; 88:807.

43. Sami SS, Al-Araji SA, Ragunath K. Review article: gastrointestinal angiodysplasia - pathogenesis, diagnosis and management. Aliment Pharmacol Ther 2014; 39:15.

44. Diggs NG, Holub JL, Lieberman DA, et al. Factors that contribute to blood loss in patients with colonic angiodysplasia from a population-based study. Clin Gastroenterol Hepatol 2011; 9:415.

45. Cappell MS, Gupta A. Changing epidemiology of gastrointestinal angiodysplasia with increasing recognition of clinically milder cases: angiodysplasia tend to produce mild chronic gastrointestinal bleeding in a study of 47 consecutive patients admitted from 1980-1989. Am J Gastroenterol 1992; 87:201.

46. Gupta N, Longo WE, Vernava AM 3rd. Angiodysplasia of the lower gastrointestinal tract: an entity readily diagnosed by colonoscopy and primarily managed nonoperatively. Dis Colon Rectum 1995; 38:979.

47. Theodoropoulou A, Sfiridaki A, Oustamanolakis P, et al. Genetic risk factors in young patients with ischemic colitis. Clin Gastroenterol Hepatol 2008; 6:907.

48. Zuckerman GR, Prakash C, Merriman RB, et al. The colon single-stripe sign and its relationship to ischemic colitis. Am J Gastroenterol 2003; 98:2018.

49. Chavalitdhamrong D, Jensen DM, Kovacs TO, et al. Ischemic colitis as a cause of severe hematochezia: risk factors and outcomes compared with other colon diagnoses. Gastrointest Endosc 2011; 74:852.

50. Dignan CR, Greenson JK. Can ischemic colitis be differentiated from C difficile colitis in biopsy specimens? Am J Surg Pathol 1997; 21:706.

51. Fernández E, Linares A, Alonso JL, et al. Colonoscopic findings in patients with lower gastrointestinal bleeding send to a hospital for their study. Value of clinical data in predicting

normal or pathological findings. Rev Esp Enferm Dig 1996; 88:16.

52. Macrae FA, St John DJ. Relationship between patterns of bleeding and Hemoccult sensitivity in patients with colorectal cancers or adenomas. Gastroenterology 1982; 82:891.

53. Soulellis CA, Carpentier S, Chen YI, et al. Lower GI hemorrhage controlled with endoscopically applied TC-325 (with videos). Gastrointest Endosc 2013; 77:504.

54. Ozdil B, Akkiz H, Sandikci M, et al. Massive lower gastrointestinal hemorrhage secondary to rectal hemorrhoids in elderly patients receiving anticoagulant therapy: case series. Dig Dis Sci 2010; 55:2693.

55. Rogers BH. Endoscopic diagnosis and therapy of mucosal vascular abnormalities of the gastrointestinal tract occurring in elderly patients and associated with cardiac, vascular, and pulmonary disease. Gastrointest Endosc 1980; 26:134.

Topic 2546 Version 22.0

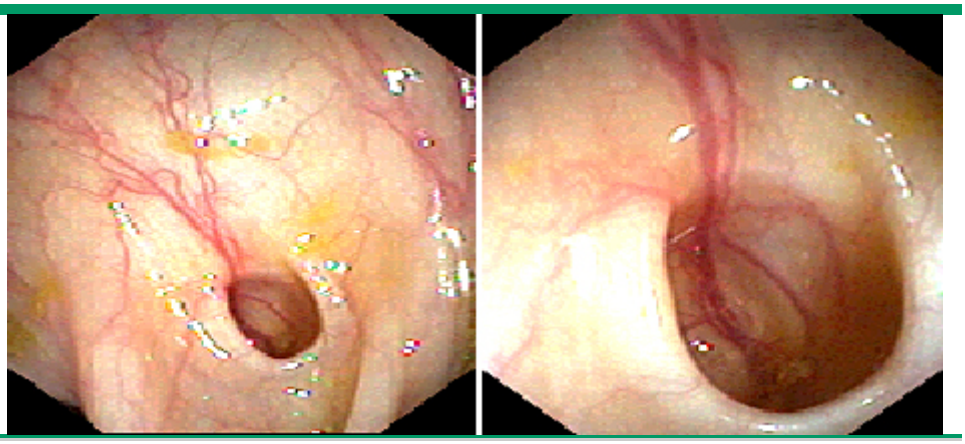
GRAPHICS

Common causes of lower gastrointestinal bleeding

Anatomic
Diverticulosis
Vascular
Angiodysplasia
Hemorrhoids
Ischemic
Post biopsy or polypectomy
Radiation-induced telangiectasia
Inflammatory
Infectious
Inflammatory bowel disease
Ulcer
Neoplastic
Polyp
Carcinoma

Graphic 58308 Version 4.0

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

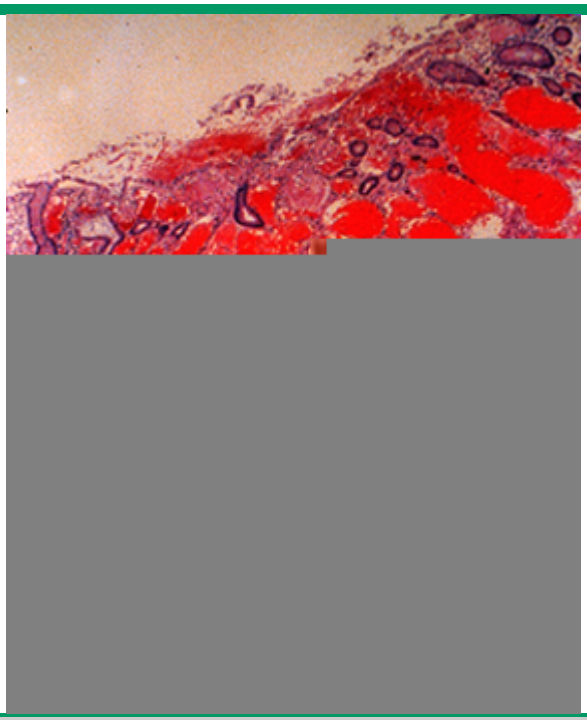
Angiodysplasia of the colon

Gross specimen of the colon with angiodysplasia shows a focal mucosal hemorrhagic area.

Courtesy of Robert Odze, MD.

Graphic 61483 Version 2.0

Angiodysplasia of the colon

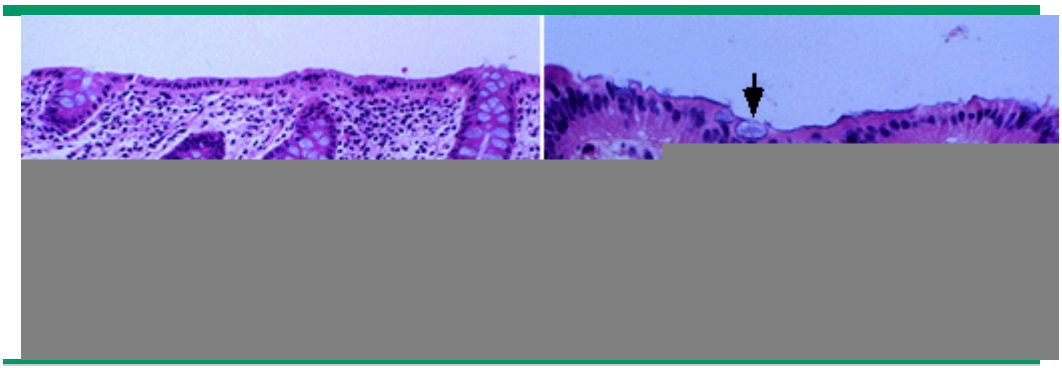


Low power view of a colonic biopsy in angiodysplasia shows mucosal and submucosal vascular dilatation and congestion.

Courtesy of Robert Odze, MD.

Graphic 74301 Version 2.0

Normal colon



Low (left) and high (right) power views of a biopsy of a normal colon. Low power reveals straight crypts and mild lamina propria mononuclear cell infiltration. High power shows the surface enterocytes with interspersed goblet cells (arrows).

Courtesy of Robert Odze, MD

Graphic 81083 Version 1.0

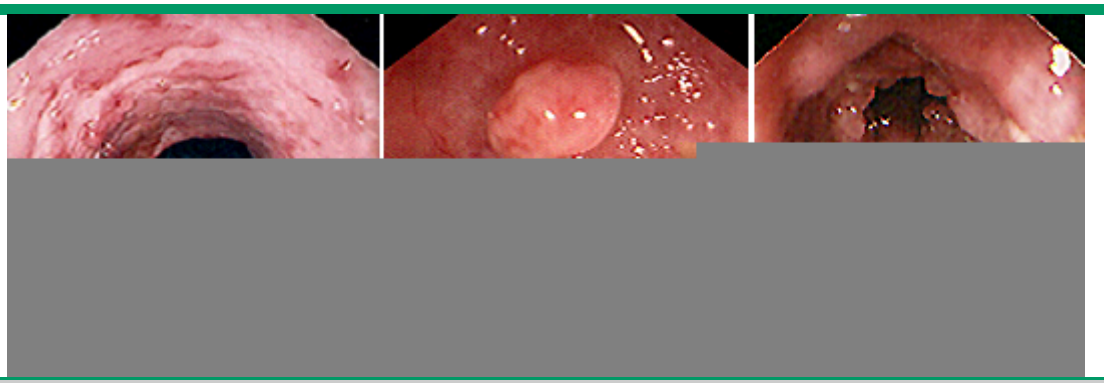
Endoscopic image of colonic angiodysplasia

Angiodysplasia appears endoscopically as peripherally expanding dilated capillaries with a central origin measuring between 0.1 to 1.0 cm in diameter.

Courtesy of Rome Jutabha, MD.

Graphic 50137 Version 4.0

Ulcerative colitis



Endoscopic appearance of ulcerative colitis. Extensive ulceration of the mucosa is the most common endoscopic finding (panel A). The surface is irregular, friable, and erythematous, with loss of the normal vascular markings. Pseudopolyps may form as a reaction to inflammation (panel B); these can become quite extensive (panel C).

Courtesy of James B McGee, MD.

Graphic 81755 Version 2.0

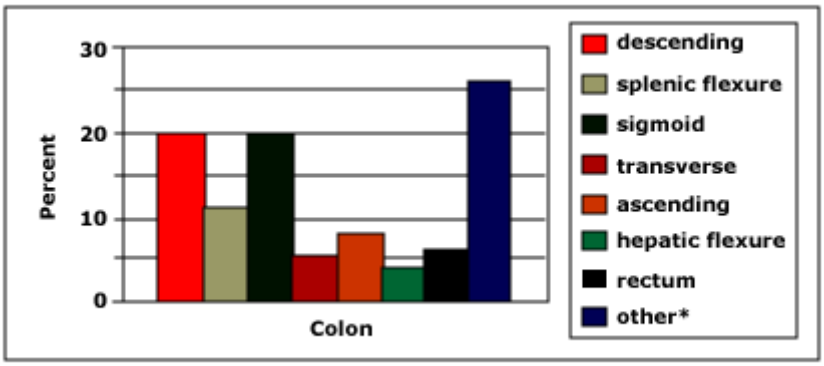
Normal sigmoid colon

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

Distribution of colon ischemia

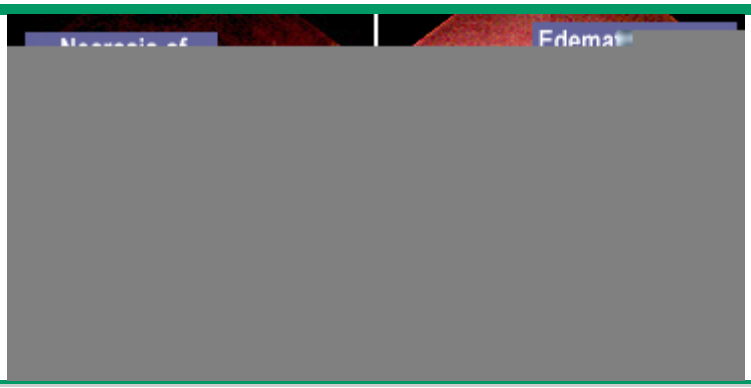


* Other areas refer to combination of different regions.

Data from: Reinus JF, Brandt LJ, Boley SJ. Ischemic diseases of the bowel. Gastroenterol Clin North Am 1990; 19:319.

Graphic 65067 Version 5.0

Ischemic colitis on colonoscopy



Endoscopy of ischemic colitis may reveal continuous necrosis and mucosal friability that resembles ulcerative colitis (left panel); discrete ulcers with surrounding edema may also be seen (right panel).

Courtesy of James B McGee, MD.

Graphic 59803 Version 2.0

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

Lisa Strate, MD, MPH Nothing to disclose **J Thomas Lamont, MD** Nothing to disclose **Shilpa Grover, MD, MPH, AGAF** Nothing to disclose

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

Conflict of interest policy