Management of Ischemic Colitis

Christopher Washington, MD¹ Joseph C. Carmichael, MD¹

¹Department of Surgery, Division of Colon and Rectal Surgery, University of California, Irvine, Orange, California

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Address for correspondence and reprint requests Joseph C. Carmichael, MD, Department of Surgery, Division of Colon and Rectal Surgery, University of California, Irvine, 333 City Blvd. West, Ste. 850, Orange, CA 92868 (e-mail: jcarmich@uci.edu).

Abstract

Keywords

- ischemic colitis
- gastrointestinal bleeding
- colonoscopy
- colectomy

Ischemic colitis is a commonly misunderstood clinical condition. Although the colon is the most common region of ischemia in the gastrointestinal tract, many surgeons have difficulty with diagnosis and treatment of ischemic colitis. The process can occur from either occlusive vascular disease or nonocclusive disease, and can be gangrenous or nongangrenous. Differentiating gangrenous from nongangrenous disease can be a difficult clinical challenge as both sets of patients generally present with abdominal pain and bloody diarrhea. Although the majority of patients have transient ischemia with nongangrenous colitis that can be successfully managed nonoperatively, prompt recognition and surgical intervention is critical in patients with gangrenous colitis. In this article, the diagnosis and treatment of ischemic colitis is reviewed with emphasis on a systematic, evidence-based approach to management.

Objectives: On completion of this article, the reader should be able to summarize the management of ischemic colitis.

Ischemic colitis is the most common form of gastrointestinal (GI) ischemia, accounting for 50 to 60% of all cases¹ and occurring with an incidence of 4.5 to 44 cases per 100,000 person years.² It accounts for 1 in 2000 hospital admissions.³ The causes of ischemic colitis are numerous, but all lead to diminished perfusion of the colon, which in turn leads to mucosal injury or even full-thickness necrosis.

For over a century, gangrene of the colon has been well described in the medical literature after ligation of a major colic artery. However, the potential transient nature of the disease without gross vascular occlusion was not described until 1963, when Boley et al⁴ identified five patients with “reversible vascular occlusion of the colon.” Around the same time, Marston et al introduced the term “ischemic colitis.” Marston further classified the disease into three forms: gangrenous, stricturing, and transient.⁵ Although these classifications continue to hold true today, the clinical acute disease is divided into only two groups: full thickness with gangrene and partial thickness, limited to the mucosa and submucosa. Since these first descriptions, ischemic colitis has become a well-recognized condition caused by decreased or interrupted blood flow to the colon. Most cases of ischemia are transient, do not lead to full thickness necrosis, and can be managed conservatively.

Establishing the correct diagnosis and providing appropriate and timely therapy continues to be a challenge. As the disease is often transient and presents with self-limiting and vague abdominal and intestinal symptoms, the exact etiology is rarely identified. Risk factors include age greater than 65, history of irritable bowel disease, chronic obstructive pulmonary disease, constipation, thrombophilia, and systemic arterial disease. Persons of female gender have been found to be at higher risk,^{2,6} although this has not been constant across all published reports.

Anatomy

Knowledge of the vascular anatomy of the colon is important to understand the development of ischemic colitis. The colon receives its blood supply from the superior mesenteric artery (SMA) and the inferior mesenteric artery (IMA). The rectum receives its blood supply from the paired internal iliac arteries and the inferior mesenteric artery (→ **Fig. 1**).

The SMA gives rise to the middle colic, right colic, and ileocolic arteries, which supply the proximal two-thirds of the transverse colon, ascending colon, and cecum. Variations in anatomy occur with ~20% of the population having an absent middle colic artery.^{7,8} The IMA branches into the left colic, sigmoid, and superior rectal (hemorrhoidal) arteries supplying the distal transverse colon, descending colon, sigmoid colon, and proximal aspect of the rectum,

Figure 1 Vascular supply of the colon. Source: Reproduced with permission of Lippincott Williams & Wilkins. From Corman ML.⁷⁰

respectively. The paired internal iliac arteries give rise to the middle and inferior rectal (hemorrhoidal) arteries that supply the mid and distal rectum, and are in continuity with the superior rectal (hemorrhoidal) arteries and the blood supply from the IMA.

In addition to these primary arterial branches, there is a rich collateral circulation. The marginal artery of Drummond extends along the periphery of the colonic mesentery, giving rise to the vasa recta and linking the inferior mesenteric artery to the superior mesenteric artery.⁹ In ~70% of patients with peripheral vascular disease, the marginal artery of Drummond comprises the main collateral and only significant source of arterial blood to the distal colon.⁸ The arc of Riolo, though highly variable in the population, directly connects the superior mesenteric and inferior mesenteric systems. These collateral vessels help to ensure adequate perfusion should one of the main branches become occluded. In persons with chronic occlusion of one of the main vascular branches, the arc of Riolo or marginal artery of Drummond may enlarge to compensate for the decreased flow.

Areas of the colon most sensitive to decreased blood flow and more likely to suffer from ischemia are the splenic flexure (Griffiths point) and sigmoid colon (Sudek's point). These "watershed" areas are where the circulation from the superior mesenteric artery and inferior mesenteric artery meet, and where the inferior mesenteric artery and rectal arteries meet. Watershed areas in particular rely on the marginal artery of Drummond to provide collateral flow. In anatomic studies, it has been shown that ~50% of the population has a poorly developed marginal artery, and ~5% have an incomplete artery leading to increased risk for ischemic injury.¹⁰ There is also evidence that the colic arteries become more tortuous with advanced age.¹¹ Overall, the watershed anatomy of the colon provides an excellent reason why the left

colon and splenic flexure is involved in 75% of ischemic colitis cases¹² and why the rectum (with its rich dual blood supply) is involved in only 5% of cases.

The venous drainage of the colon parallels the arterial supply. The superior mesenteric vein drains the small intestine, right, and transverse colon; the inferior mesenteric vein drains the left colon and the rectum. The inferior mesenteric vein connects with the splenic vein, which joins the superior mesenteric vein to form the portal vein.

Etiology

The causes of intestinal ischemia are numerous and can be divided into two groups: occlusive disease and nonocclusive disease (►Table 1). Both lead to a reduction in mesenteric blood flow to the colon leading to ischemia. The severity of the ischemia will determine whether transmural necrosis (gangrenous colitis) occurs or if only the mucosa and/or submucosa is involved (nongangrenous colitis). Segmental, nongangrenous ischemic colitis accounts for 80 to 85% of cases and often resolves with medical management.³ Gangrene occurs in the remaining cases and always requires surgical intervention.

Frequently no clear cause for the ischemia is identified, making the diagnosis difficult. Angiography is rarely helpful in the workup of ischemic colitis, as large vessel occlusion is almost never identified. The most likely cause in these instances is localized, nonocclusive ischemia in association with small vessel disease. Patients with ischemic colitis tend to have multiple risk factors for vascular disease such as persons over 65 years of age, persons with cardiac arrhythmia, and thrombophilia. For unclear reasons, patients with irritable bowel syndrome (IBS) have a threefold increased risk for ischemic colitis. Patients with chronic obstructive pulmonary disease (COPD) have a two- to fourfold increased risk compared with the general population.² Constipation has also been identified as a risk factor for the development of ischemic colitis. This is believed to be due to increased intraluminal pressures causing compression of the blood vessels and resultant decreased mucosal vessel flow.¹³ Concurrent use of laxatives can exacerbate the problem, potentially leading to perforation. Females are more likely to suffer from ischemic colitis than are males.^{2,6} Other common risk factors included hypertension, renal failure or nephropathy, and diabetes.^{14,15}

As noted above, thrombophilia appears to play a role in colonic ischemia and abnormal test results have been reported in anywhere from 28 to 74% of patients diagnosed with ischemic colitis.¹² However, it is still undetermined how much of a role this plays in the development of the disease. Antiphospholipid antibodies and Factor V Leiden mutations are found 10 times more frequently in patients with ischemic colitis.¹⁶

When colonic ischemia is found in younger patients, there has been association with vasculitis,¹⁷ drugs and medications,^{18–23} sickle cell disease,²⁴ extreme exercise,^{25,26} and even taking an airplane flight.²⁷ The right colon is more frequently involved for unknown reasons.

Table 1 Causes of Ischemic Colitis

Occlusive Disease	Nonocclusive Disease
Arterial	Hypoperfusion
Thrombosis/emboli	Cardiac failure
Arterial emboli (cardiac)	Septic shock
Cholesterol emboli	Hemorrhagic shock
Small vessel disease	Hemodialysis
Atherosclerosis	Hypovolemia
Diabetes	Anaphylaxis
Vasculitis	Pancreatitis
Rheumatoid arthritis	
Radiation	Iatrogenic
Amyloidosis	Drugs
Trauma	Alosetron
	Antihypertensives
Surgical	Digitalis
Aortoiliac reconstruction	Diuretics
Cardiopulmonary bypass	Cocaine
Colectomy	Estrogens
Endoscopy	Danazol
Renal transplant	Nonsteroidal antiinflammatory drugs
Colonoscopy	Tegaserod
Barium enema	Vasopressors
	Pseudoephedrine
Venous	Paclitaxel and carboplatin
Mesenteric venous thrombosis	Sumatriptan
Hypercoagulable state	Simvastatin
Sickle cell disease	Interferon-ribavirin
Pancreatitis	
Portal hypertension	Colonic obstruction
Lymphocytic phlebitis	Volvulus
	Colon cancer
	Constipation
	Pseudo-obstruction
	Long-distance running

Pathophysiology

Colonic ischemia is usually the result of a sudden, temporary, reduction in blood flow that is insufficient to meet the metabolic demands of the region of colon. The initial and most intense ischemic changes are always in the colonic mucosa. Ischemic change will subsequently extend from the mucosa to the serosa. The mucosal change will be most evident on the antimesenteric mucosal lining of the colon. During periods of hypotension, blood flow is redirected to the

brain at the expense of the splanchnic circulation. During these episodes, patients are more susceptible to ischemic insults. Mucosal injury will develop in ~20 minutes to 1 hour, whereas transmural infarction occurs within 8 to 16 hours.²⁸ Additional insult occurs when blood flow is reestablished and reperfusion injury occurs. Reperfusion injury is associated with the release of reactive oxygen species, which cause lipid peroxidation within cell membranes, resulting in cell necrosis.^{29,30}

In healthy individuals, the splanchnic circulation receives 10 to 35% of the cardiac output. In a canine model, blood flow to the intestine had to be reduced by 50% to induce ischemia, showing that the intestine is quite resistant to periods of poor perfusion.³¹ The local autoregulation of the splanchnic vasculature allows for maintenance of intestinal perfusion and oxygen extraction until mesenteric arterial pressure falls below 40 to 45 mm Hg.³² However, given the high capillary density of the bowel and the increased permeability, any increase in the capillary pressure can lead to significant fluid loss resulting in edema of the bowel. Additionally, as permeability increases and ischemia progresses, bacterial translocation will occur leading to systemic absorption of endoluminal toxins and potential progression to multiorgan failure.^{33,34}

Clinical Presentation

Whether the inciting event was from occlusive or nonocclusive disease, ischemic colitis presents with acute-onset abdominal cramping and abdominal pain. The development of pain is very often associated with the urge to defecate and may be followed by the development of hematochezia within 24 hours. The occasional patient may present with painless diarrhea and/or hematochezia; however, ischemic colitis accounts for only 3 to 9% of all cases of acute lower GI bleeding. Bleeding may be bright red in left colon involvement, or maroon and mixed with the stool in right colon disease. Other symptoms include diarrhea (68%), abdominal distention (63%), and nausea/vomiting (38%).³⁵

Clinical examination often reveals abdominal tenderness over the region of the ischemic segment. This tenderness is often mild and may be very subtle. Peritoneal signs, such as rebound pain and guarding, correspond to more advanced disease with likely transmural necrosis. These patients will commonly have systemic signs of SIRS (systemic inflammatory response syndrome); sepsis with tachycardia and leukocytosis being the most common two findings. Fortunately, this is a rare occurrence and most patients do not have signs of peritonitis or hemodynamic instability on presentation.

There is often no identifiable event; however, a history of recent cardiac or vascular surgery, major systemic illness, or myocardial event may be present. The differential diagnosis is broad and includes, but is not limited to, diverticulitis, colon cancer, mesenteric artery insufficiency, mesenteric venous thrombosis, inflammatory bowel disease, infectious colitis, peptic ulcer disease, bowel obstruction, and pancreatitis. ► **Table 1** lists the various possible etiologies of ischemic colitis.

In the chronic setting, ischemic colitis may present with abdominal distention and obstipation. These obstructive symptoms are usually the result of an ischemic stricture.

Diagnosis

Given the vague clinical presentation, broad differential diagnosis, and difficulty in finding an inciting event, the diagnosis of ischemic colitis is often delayed while exploring the more common etiologies. Diagnostic evaluation of these patients must include stool cultures to evaluate for infectious colitis, *Clostridium difficile* antigen to assess for *Clostridium difficile* colitis, computed tomography (CT) scan to assess for diverticulitis, and a combination of CT imaging and endoscopy to look for inflammatory bowel disease or colonic carcinoma. In many cases, endoscopic biopsy may be instrumental in differentiating ischemic colitis from ulcerative or infectious colitis.³⁶ The combination of history, physical examination findings, laboratory data, radiographic imaging, endoscopy, and biopsy allow for the accurate diagnosis. In addition, routine-screening electrocardiogram (ECG), transthoracic echocardiogram, and even Holter monitoring has been suggested due to the high potential for cardiac emboli.³⁷ In younger patients, a cause is often more easily identified due to a higher association with medications, illicit drugs, sickle cell disease, thrombophilia, or vasculitis.^{1,35,38}

Laboratory Studies

Routine laboratory studies are nonspecific for colonic ischemia and are generally unhelpful.³⁹ Leukocytosis is a frequent finding, especially in advanced disease. Findings such as an elevated lactate, metabolic acidosis, and significant base deficit may be present in cases of severe ischemia and/or necrosis, but this is generally a late sign. Recently, studies involving serum levels of the stereoisomer D-lactate have shown promise in identifying a more sensitive marker of colonic ischemia. D-Lactate is produced only by bacteria, especially in the colon, as a normal product of bacterial fermentation. During ischemia, as the normal mucosal barrier is damaged and permeability increases, D-lactate enters the circulation; because the liver is unable to metabolize D-lactate, a rise in the serum concentration occurs.^{40,41} These experimental laboratory studies have yet to enter standard clinical practice.

Imaging

X-Rays

Plain radiographs are rarely helpful in the diagnosis of ischemic colitis. Unless the disease has progressed to transmural necrosis and perforation causing pneumoperitoneum, the findings may be subtle or absent. Thumbprinting is a classic finding for mucosal edema, and though not specific for ischemic colitis, it may be helpful in directing management. Pneumatosis intestinalis may also be identified if mucosal damage has occurred with passage of gas into the bowel wall. Although this finding is concerning, it is not specific to ischemic colitis and warrants further evaluation. An initial

set of plain radiographs is helpful to ensure that there has been no visceral perforation with evidence of pneumoperitoneum, as this would mandate more-aggressive management.

The use of contrast enemas had previously been the diagnostic modality of choice, but has been replaced, for the most part, by CT imaging and colonoscopy. Contrast enemas may suggest colonic ischemia by showing thumbprinting, pseudopolyps, sacculization, tubular narrowing, or a ragged, saw-toothed irregularity of the mucosa, but the findings are nonspecific and diagnosis can only be made with serial enemas demonstrating resolution of the disease.^{4,8,42} The pressure exerted on the colon wall may also exacerbate the ischemic injury by colonic distension or potentially cause perforation. It also may interfere with subsequent angiography, computed tomography, or endoscopy due to residual contrast agent.⁴³ Contrast enemas continue to be helpful in the management of ischemic strictures when the length and location of the stricture can be clearly identified.

Computed Tomography

CT is the most helpful in the initial assessment of the patient with abdominal pain. It can exclude other causes of abdominal pain, suggest a location and source of ischemia, and identify complications associated with more-advanced disease.

In the nongangrenous form of the disease, the CT will frequently show bowel wall thickening, thumbprinting, and pericolic stranding with or without ascites. The double halo or target sign may be present.⁴⁴ Segmental bowel wall thickening is seen in most cases with an average wall thickness of 8 mm.⁴⁵ After reperfusion, there may be evidence of submucosal edema or hemorrhage. Emboli or thrombus causing complete arterial occlusion is occasionally seen with corresponding thin, unenhancing colonic wall due to complete lack of reperfusion. Pneumatosis coli, or air within the mesenteric or portal venous system, is a more ominous finding associated with bowel infarction; however, it can be associated with COPD, infectious colitis, steroid treatment, or after radiation or chemotherapy, or associated with acquired immunodeficiency syndrome (AIDS).⁴⁴ Unfortunately, CT findings do not correlate with, nor do they predict, the development of bowel infarction.⁴⁵

Angiography

Angiography is rarely helpful in the diagnosis of ischemic colitis, as most cases of ischemic colitis involve transient small vessel hypoperfusion. In cases where embolus is suspected or suggested by CT or acute mesenteric ischemia is considered, then angiography may be useful. This is especially true in cases of isolated right-sided colitis when the diagnosis of acute mesenteric ischemia must be excluded.¹²

Ultrasound

Color Doppler sonography has recently been shown to be a very sensitive predictor of colonic ischemia. Identification of colonic wall thickening and altered pericolic fat, along with

absent or barely visible color Doppler flow make the suggestion of ischemic disease.⁴⁶ Serial examinations may provide additional information regarding the progression of disease; however, sonography is limited by overlying bowel gas, the experience of the operator, and poor sensitivity for low-flow vessel disease.^{47,48}

Nuclear Medicine

The role of In-111 label leukocyte scans has been suggested as a noninvasive modality to help localize the extent of disease and aid in the type and extent of surgical resection⁴⁹ however, there has been rare use in clinical applications.

Endoscopy

The use of colonoscopy is essential in establishing the diagnosis, and has become the gold standard for confirmation of ischemic colitis. The first part of the colon to become affected by ischemia is the mucosa on the antimesenteric side. Findings on colonoscopy may include edema, ulceration and easy friability, involvement of watershed areas, asymmetric pattern of involvement with the antimesenteric side becoming ischemic first, and possibly the development of pseudomembranes related to mucosal sloughing. Within the first 48 hours, the colonic mucosa will appear pale and edematous with areas of petechial hemorrhage or superficial ulceration. A single linear ulcer running longitudinally along the antimesenteric colonic wall (colonic single stripe sign) may be seen and is associated with ischemic colitis.⁵⁰ ▶ **Figure 2** shows the endoscopic appearance of the classic single stripe sign. Hemorrhagic nodules may also be seen if the ischemia has progressed to the submucosa with resultant bleeding. After 48 hours, sloughing occurs, the purple submucosal hemorrhages dissipate, and ulcerations develop. In more severe ischemia with transmural infarction, the mucosa may appear gray-green or even black.

Colonoscopy should be performed with limited insufflation to avoid over distension of the colon, which could lead to further ischemia or even perforation. Whenever possible, CO₂ insufflation rather than air should be used, as CO₂ is more

Figure 2 Classic endoscopic view of antimesenteric ischemic “stripe.”

rapidly absorbed and acts as a vasodilator, potentially improving colonic perfusion. In addition to the visual inspection by endoscopy, it also allows for biopsy to establish tissue diagnosis. An early histologic finding of ischemia is focal crypt dropout. Advanced ischemia will reveal epithelial loss with the presence of acute and chronic inflammatory cells and submucosal congestion.

Treatment

For the majority of patients without peritoneal signs, nonoperative management may be safely employed. As the underlying cause of ischemic colitis is hypoperfusion of the colon, it is important to remove any potential exacerbating factors and to optimize perfusion of the ischemic region. This entails use of intravenous (IV) fluid resuscitation, optimizing cardiac output, and use of supplemental oxygen. If the inciting cause can be removed, such as in the case of medications, this should be done immediately. Patients should be placed on bowel rest, and a nasogastric tube placed should signs of ileus appear. Parenteral nutrition should be considered in patients who need prolonged bowel rest or have a major contraindication to surgery.⁵¹ The use of broad-spectrum antibiotics to cover both aerobic and anaerobic coliform bacteria has been advocated, as the disease process does cause a disruption of the mucosal barrier and may lead to bacterial translocation.³ The use of bowel prep prior to colonoscopy or surgical intervention is contraindicated due to the risk of perforation or toxic dilation. There is no role for steroids in the treatment of ischemic colitis as they only serve in masking the development of peritoneal signs and may delay necessary surgical intervention.³⁶ Close monitoring should continue assessing for fever, increasing leukocytosis, development of acidosis, increased abdominal pain or tenderness. In patients without clinical improvement within 24 to 48 hours, repeat endoscopy or imaging is useful to reexamine the severity of the disease. In patients who develop worsening peritonitis, perforated viscous, uncontrolled bleeding, or failure to improve, surgical intervention is required and bowel resection is performed.

The extent of bowel resection must rely on preoperative imaging or endoscopy to determine the extent of disease. Because the ischemia is usually limited to the mucosa or submucosa, the serosa will often appear normal, giving an unreliable measure of the true extent of disease. Other modalities used intraoperatively to assess the viability of the bowel are Doppler ultrasonography, intraoperative colonoscopy, intraoperative photoplethysmography, oxygen electrodes, pulse oximetry of transcolonic oxygen saturation, and use of IV fluorescein.^{52–54} However, none of these modalities has entered into routine clinical practice. At the time of resection, the colon can be opened and the mucosa evaluated.

The decision for anastomosis is dependant on the clinical picture. For patients where there is concern for ongoing ischemia, leaving the bowel in discontinuity and returning for a second-look laparotomy has been advised⁵⁵ due to the need for additional resection in up to 25% of these patients.⁵⁶ When a definitive operation is performed for left-sided

ischemic colitis, published opinion advocates for an end colostomy and rectal stump (Hartmann procedure). In a stable patient with uncomplicated right colon ischemia with no evidence of perforation, resection with primary anastomosis is preferred.⁵⁷ Otherwise, resection with terminal ileostomy is performed. In patients who have developed colonic stricture after resolution of an acute ischemic episode or have developed a stricture from chronic ischemia, bowel resection is indicated. Alternatively, there are now reports of endoscopic dilation or stenting of short strictures.¹² Chronic colitis as a development of continued ischemia or unhealed areas of segmental colitis should also be considered for elective resection.

Special Cases

Aortoiliac Surgery

One of the first recognized causes of colonic ischemia was ligation of the inferior mesenteric artery (IMA) during abdominal aortic surgery. Studies have shown that the overall incidence is between 1% and 7% following elective aortic reconstruction and as high as 9% in cases of aortic rupture.^{58–61} However, these figures may not include asymptomatic patients who could only be identified with routine endoscopy. In a series of 160 patients who underwent routine sigmoidoscopy with biopsy after aortic aneurysm repair, 15% had histologically proven ischemia, but only a third of those were symptomatic.⁶² The distal left colon is almost exclusively involved due to ligation of the IMA, or intraoperative hypoperfusion in conjunction with disease of this artery. Routine reimplantation of the IMA after aortic repair has not been shown to alter the overall incidence of ischemic colitis; however, in older patients and patients with increased blood loss this technique may be helpful in avoidance of ischemia.⁶³ The risk of colonic ischemia is significantly lower after endovascular repair with a recorded incidence of 0.5%. Those patients who develop ischemic colitis have a mortality that is six times greater than their matched cohorts.⁶³

Often the first manifestation of ischemic colitis in these postoperative patients is grossly bloody or guaiac-positive diarrhea. Abdominal pain is frequently masked by the recent major operation and renders physical exam findings unreliable. Bleeding usually occurs within 2 days of surgery and must be investigated with prompt sigmoidoscopy to evaluate for evidence of ischemia. The stable patient without full-thickness necrosis can be managed nonoperatively with IV fluids, bowel rest, and antibiotics. If surgical intervention and bowel resection is indicated, primary anastomosis is contraindicated due to the risk of leakage and potential contamination of the aortic prosthesis.

Phlebosclerotic Colitis and Lymphocytic Colitis

Phlebosclerotic colitis is a very rare form of ischemic colitis from venous obstruction caused by fibrotic sclerosis and calcification. It preferentially involves the right colon and the etiology is unknown. On CT, colonic wall thickening with adjacent mesenteric venous calcifications are identified.⁶⁴ A similar entity, known as enterocolic lymphocytic phlebitis or

lymphocytic colitis, is characterized by prominent lymphocytic inflammation of the venous system. The involved vessels are found in the bowel wall and mesentery.⁶⁵ Symptoms generally resolve with supportive care; surgery is reserved for rare cases in which gangrene develops or a postischemic stricture causes intestinal obstruction.

Prognosis

The overall prognosis for ischemic colitis is dependent upon the location of disease, comorbid conditions, and whether or not the patient requires surgery.¹⁴ The overall mortality is ~22%.⁶⁶ The severity of disease and ultimate mortality is higher in right-sided disease.¹⁵ The majority of patients will have improvement in their symptoms within 24 to 48 hours and complete clinical recovery within 1 to 2 weeks as the colonic mucosa regenerates and heals. The 20% of patients who will require surgery have an associated mortality rate of 10 to 65%, and can be as high as 75% in patients with pancolitis.^{3,35} In one series, 75% of patients requiring stomas for segmental resections were eventually able to have their stomas closed. In patients with total colonic involvement, only one-third had stoma closure.⁶⁷ Patients with more severe transient colitis may ultimately develop strictures as the colon heals. Follow-up colonoscopy or imaging should be performed to evaluate for stricture and resolution of the colitis. If clinically significant stricture does occur, then elective segmental resection with primary anastomosis is recommended.

Risk factors for poor outcomes include the development of ischemic colitis after aortoiliac surgery, the development of ischemic colitis in association with myocardial infarction,⁶⁸ isolated right colon involvement, chronic renal failure, or patients on hemodialysis.⁶⁹ Compared with other areas of ischemia, right colon involvement has been associated with longer hospitalizations, a greater need for surgery, and a higher mortality rate.

Conclusion

Ischemic colitis is the most common form of GI ischemia; the majority of cases resolve with nonsurgical management. Prompt recognition of full-thickness necrosis and gangrene is critical to good patient outcomes. The clinical scenario, and CT and endoscopic findings are essential in helping the clinician to identify patients with ischemic colitis who are at risk for full-thickness colonic necrosis.

References

- 1 Stamos MJ. Intestinal ischemia and infarction. In Mazier WP, Levien DH, Luchtefeld MA, Senagore AJ, eds. *Surgery of the Colon, Rectum and Anus*. Philadelphia: WB Saunders; 1995:685–718
- 2 Higgins PD, Davis KJ, Laine L. Systematic review: the epidemiology of ischaemic colitis. *Aliment Pharmacol Ther* 2004;19(7):729–738
- 3 Brandt LJ, Boley SJ. Colonic ischemia. *Surg Clin North Am* 1992; 72(1):203–229
- 4 Boley SJ, Schwartz S, Lash J, Sternhill V. Reversible vascular occlusion of the colon. *Surg Gynecol Obstet* 1963;116:53–60

- 5 Marston A, Pheils MT, Thomas ML, Morson BC. Ischaemic colitis. *Gut* 1966;7(1):1–15
- 6 Chang L, Kahler KH, Sarawate C, Quimbo R, Kralstein J. Assessment of potential risk factors associated with ischaemic colitis. *Neurogastroenterol Motil* 2008;20(1):36–42
- 7 Griffiths JD. Surgical anatomy of the blood supply of the distal colon. *Ann R Coll Surg Engl* 1956;19(4):241–256
- 8 Byrd BF Jr, Sawyers JL, Bomar RL, Klatte EC. Reversible vascular occlusion of the colon: recognition and management. *Ann Surg* 1968;167(6):901–908
- 9 Drummond H. Some points relating to the surgical anatomy of the arterial supply of the large intestine. *Proc R Soc Med* 1914; 7(Surg Sect):185–193
- 10 Sonneland J, Anson BJ, Beaton LE. Surgical anatomy of the arterial supply to the colon from the superior mesenteric artery based upon a study of 600 specimens. *Surg Gynecol Obstet* 1958;106 (4):385–398
- 11 Binns JC, Isaacson P. Age-related changes in the colonic blood supply: their relevance to ischaemic colitis. *Gut* 1978;19(5): 384–390
- 12 Elder K, Lashner BA, Al Solaiman F. Clinical approach to colonic ischemia. *Cleve Clin J Med* 2009;76(7):401–409
- 13 Beck IT. Possible mechanisms for ischemic colitis during alosetron therapy. *Gastroenterology* 2001;121(1):231–232
- 14 Korotinski S, Katz A, Malnick SD. Chronic ischaemic bowel diseases in the aged—go with the flow. *Age Ageing* 2005;34(1):10–16
- 15 O'Neill S, Elder K, Harrison SJ, Yalamarthi S. Predictors of severity in ischaemic colitis. *Int J Colorectal Dis* 2012;27(2):187–191
- 16 Koutroubakis IE, Sfiridaki A, Theodoropoulou A, Kouroumalis EA. Role of acquired and hereditary thrombotic risk factors in colon ischemia of ambulatory patients. *Gastroenterology* 2001;121(3): 561–565
- 17 Ho MS, Teh LB, Goh HS. Ischaemic colitis in systemic lupus erythematosus—report of a case and review of the literature. *Ann Acad Med Singapore* 1987;16(3):501–503
- 18 Gurbuz AK, Gurbuz B, Salas L, Rosenshein NB, Donowitz M, Giardiello FM. Premarin-induced ischemic colitis. *J Clin Gastroenterol* 1994;19(2):108–111
- 19 Miyata T, Tamechika Y, Torisu M. Ischemic colitis in a 33-year-old woman on danazol treatment for endometriosis. *Am J Gastroenterol* 1988;83(12):1420–1423
- 20 Wright A, Benfield GF, Felix-Davies D. Ischaemic colitis and immune complexes during gold therapy for rheumatoid arthritis. *Ann Rheum Dis* 1984;43(3):495–497
- 21 Carratù R, Parisi P, Agozzino A. Segmental ischemic colitis associated with nonsteroidal antiinflammatory drugs. *J Clin Gastroenterol* 1993;16(1):31–34
- 22 Larrey D, Lainey E, Blanc P, et al. Acute colitis associated with prolonged administration of neuroleptics. *J Clin Gastroenterol* 1992;14(1):64–67
- 23 Brown DN, Rosenholtz MJ, Marshall JB. Ischemic colitis related to cocaine abuse. *Am J Gastroenterol* 1994;89(9):1558–1561
- 24 Gage TP, Gagnier JM. Ischemic colitis complicating sickle cell crisis. *Gastroenterology* 1983;84(1):171–174
- 25 Beaumont AC, Teare JP. Subtotal colectomy following marathon running in a female patient. *J R Soc Med* 1991;84(7):439–440
- 26 Heer M, Repond F, Hany A, Sulser H, Kehl O, Jäger K. Acute ischaemic colitis in a female long distance runner. *Gut* 1987; 28(7):896–899
- 27 Butcher JH, Davis AJ, Page A, Green B, Shepherd HA. Transient ischaemic colitis following an aeroplane flight: two case reports and review of the literature. *Gut* 2002;51(5):746–747
- 28 Haglund U, Bulkley GB, Granger DN. On the pathophysiology of intestinal ischemic injury. Clinical review. *Acta Chir Scand* 1987;153(5-6):321–324
- 29 Kolkman JJ, Mensink PB. Non-occlusive mesenteric ischaemia: a common disorder in gastroenterology and intensive care. *Best Pract Res Clin Gastroenterol* 2003;17(3):457–473
- 30 Yasuhara H. Acute mesenteric ischemia: the challenge of gastroenterology. *Surg Today* 2005;35(3):185–195
- 31 Bulkley GB, Kvietys PR, Parks DA, Perry MA, Granger DN. Relationship of blood flow and oxygen consumption to ischemic injury in the canine small intestine. *Gastroenterology* 1985; 89(4):852–857
- 32 Haglund U, Bergqvist D. Intestinal ischemia—the basics. *Langenbecks Arch Surg* 1999;384(3):233–238
- 33 Zhi-Yong S, Dong YL, Wang XH. Bacterial translocation and multiple system organ failure in bowel ischemia and reperfusion. *J Trauma* 1992;32(2):148–153
- 34 Reilly PM, Bulkley GB. Vasoactive mediators and splanchnic perfusion. *Crit Care Med* 1993;21(2, Suppl):S55–S68
- 35 Longo WE, Ballantyne GH, Gusberg RJ. Ischemic colitis: patterns and prognosis. *Dis Colon Rectum* 1992;35(8):726–730
- 36 Stamatakis M, Douzinas E, Stefanaki C, et al. Ischemic colitis: surging waves of update. *Tohoku J Exp Med* 2009;218(2): 83–92
- 37 Hourmand-Ollivier I, Bouin M, Saloux E, et al. Cardiac sources of embolism should be routinely screened in ischemic colitis. *Am J Gastroenterol* 2003;98(7):1573–1577
- 38 Theodoropoulou A, Koutroubakis IE. Ischemic colitis: clinical practice in diagnosis and treatment. *World J Gastroenterol* 2008;14(48):7302–7308
- 39 Kurland B, Brandt LJ, Delany HM. Diagnostic tests for intestinal ischemia. *Surg Clin North Am* 1992;72(1):85–105
- 40 Poeze M, Froom AHM, Greve JWM, Ramsay G. D-lactate as an early marker of intestinal ischaemia after ruptured abdominal aortic aneurysm repair. *Br J Surg* 1998;85(9):1221–1224
- 41 Murray MJ, Gonze MD, Nowak LR, Cobb CF, Serum D. Serum D (-)-lactate levels as an aid to diagnosing acute intestinal ischemia. *Am J Surg* 1994;167(6):575–578
- 42 Davis JE. Reversible vascular occlusion of the colon. *Ann Surg* 1970;171(5):789–795
- 43 MacDonald PH. Ischaemic colitis. *Best Pract Res Clin Gastroenterol* 2002;16(1):51–61
- 44 Thoeni RF, Cello JP. CT imaging of colitis. *Radiology* 2006;240 (3):623–638
- 45 Balthazar EJ, Yen BC, Gordon RB. Ischemic colitis: CT evaluation of 54 cases. *Radiology* 1999;211(2):381–388
- 46 Ripollés T, Simó L, Martínez-Pérez MJ, Pastor MR, Igual A, López A. Sonographic findings in ischemic colitis in 58 patients. *AJR Am J Roentgenol* 2005;184(3):777–785
- 47 Roobottom CA, Dubbins PA. Significant disease of the celiac and superior mesenteric arteries in asymptomatic patients: predictive value of Doppler sonography. *AJR Am J Roentgenol* 1993;161 (5):985–988
- 48 Hollerweger A. Colonic diseases: the value of US examination. *Eur J Radiol* 2007;64(2):239–249
- 49 Vijayakumar V, Bekerman C, Blend MJ. Preoperative prediction of extent and severity of ischemic colitis by imaging with In-111 labeled leukocytes. *Clin Nucl Med* 1991;16(2):98–102
- 50 Zuckerman GR, Prakash C, Merriman RB, Sawhney MS, De-Schryver-Kecskemeti K, Clouse RE. The colon single-stripe sign and its relationship to ischemic colitis. *Am J Gastroenterol* 2003; 98(9):2018–2022
- 51 Brandt LJ, Boley SJ; American Gastrointestinal Association. AGA technical review on intestinal ischemia. *Gastroenterology* 2000; 118(5):954–968
- 52 Bergman RT, Gloviczki P, Welch TJ, et al. The role of intravenous fluorescein in the detection of colon ischemia during aortic reconstruction. *Ann Vasc Surg* 1992;6(1):74–79
- 53 Maupin GE, Rimar SD, Villalba M. Ischemic colitis following abdominal aortic reconstruction for ruptured aneurysm. A 10-year experience. *Am Surg* 1989;55(6):378–380
- 54 Bulkley GB, Zuidema GD, Hamilton SR, O'Mara CS, Klacsmann PG, Horn SD. Intraoperative determination of small intestinal viability following ischemic injury: a prospective, controlled trial of two

- adjuvant methods (Doppler and fluorescein) compared with standard clinical judgment. *Ann Surg* 1981;193(5):628–637
- 55 Hanisch E, Schmandra TC, Encke A. Surgical strategies—anastomosis or stoma, a second look—when and why? *Langenbecks Arch Surg* 1999;384(3):239–242
 - 56 Schneider TA, Longo WE, Ure T, Vernava AM III. Mesenteric ischemia. Acute arterial syndromes. *Dis Colon Rectum* 1994;37(11):1163–1174
 - 57 Gandhi SK, Hanson MM, Vernava AM, Kaminski DL, Longo WE. Ischemic colitis. *Dis Colon Rectum* 1996;39(6):1171–1174
 - 58 Launer DP, Miscal BG, Beil AR Jr. Colorectal infarction following resection of abdominal aortic aneurysms. *Dis Colon Rectum* 1978;21(8):613–617
 - 59 Ottinger LW, Darling RC, Nathan MJ, Linton RR. Left colon ischemia complicating aorto-iliac reconstruction. Causes, diagnosis, management, and prevention. *Arch Surg* 1972;105(6):841–846
 - 60 Zelenock GB, Strodel WE, Knol JA, et al. A prospective study of clinically and endoscopically documented colonic ischemia in 100 patients undergoing aortic reconstructive surgery with aggressive colonic and direct pelvic revascularization, compared with historic controls. *Surgery* 1989;106(4):771–779, discussion 779–780
 - 61 Hagihara PF, Ernst CB, Griffen WO Jr. Incidence of ischemic colitis following abdominal aortic reconstruction. *Surg Gynecol Obstet* 1979;149(4):571–573
 - 62 Senekowitsch C, Assadian A, Assadian O, Hartleb H, Ptakovsky H, Hagmüller GW. Replanting the inferior mesentery artery during infrarenal aortic aneurysm repair: influence on postoperative colon ischemia. *J Vasc Surg* 2006;43(4):689–694
 - 63 Perry RJ, Martin MJ, Eckert MJ, Sohn VY, Steele SR. Colonic ischemia complicating open vs endovascular abdominal aortic aneurysm repair. *J Vasc Surg* 2008;48(2):272–277
 - 64 Jan YT, Yang FS. Phleboscrotic colitis. *J Am Coll Surg* 2008;207(5):785
 - 65 Wright CL, Cacala S. Enterocolic lymphocytic phlebitis with lymphocytic colitis, lymphocytic appendicitis, and lymphocytic enteritis. *Am J Surg Pathol* 2004;28(4):542–547
 - 66 Díaz Nieto R, Varcada M, Ogunbiyi OA, Winslet MC. Systematic review on the treatment of ischaemic colitis. *Colorectal Dis* 2011;13(7):744–747
 - 67 Longo WE, Ward D, Vernava AM III, Kaminski DL. Outcome of patients with total colonic ischemia. *Dis Colon Rectum* 1997;40(12):1448–1454
 - 68 Cappell MS, Mahajan D, Kurupath V. Characterization of ischemic colitis associated with myocardial infarction: an analysis of 23 patients. *Am J Med* 2006;119(6):527, e1–e9
 - 69 Flobert C, Cellier C, Berger A, et al. Right colonic involvement is associated with severe forms of ischemic colitis and occurs frequently in patients with chronic renal failure requiring hemodialysis. *Am J Gastroenterol* 2000;95(1):195–198
 - 70 Corman ML. Anatomy of the colon. In: *Colon and Rectal Surgery*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2005: 1–29