Colonic ischemia

INTRODUCTION — Intestinal ischemia is caused by a reduction in blood flow, which can be related to acute arterial occlusion (embolic, thrombotic), venous thrombosis, or hypoperfusion of the mesenteric vasculature causing nonocclusive ischemia.

Colonic ischemia is the most frequent form of intestinal ischemia, most often affecting the elderly [1]. Approximately 15 percent of patients with colonic ischemia develop gangrene, the consequences of which can be life-threatening, making rapid diagnosis and treatment imperative. The remainder develops nongangrenous ischemia, which is usually transient and resolves without sequelae [2]. However, some of these patients will have a more prolonged course or develop long-term complications, such as stricture or chronic ischemic colitis.

The diagnosis and treatment of patients can be challenging since colonic ischemia often occurs in patients who are debilitated and have multiple medical problems. The clinical features, diagnosis, and treatment of ischemia affecting the colon and rectum will be reviewed here. Acute and chronic intestinal ischemia of the small intestine are discussed separately. (See "Acute mesenteric ischemia" and "Chronic mesenteric ischemia").

BLOOD SUPPLY OF THE COLON — The circulation to the large intestine and rectum is derived from the superior mesenteric artery (SMA), inferior mesenteric artery (IMA), and internal iliac arteries (figure 1). The colorectal circulation is relatively constant except for rare individual anatomic variations [3]. The mesenteric veins parallel the arterial circulation, draining into the portal venous system (figure 2). An extensive collateral circulation (figure 3) protects the intestines from transient periods of inadequate perfusion [4, 5]. However, the "watershed" areas of the colon, which have limited collateral blood flow, such as the splenic flexure and rectosigmoid junction, are at risk for ischemia particularly related to hypoperfusion (figure 4) [6-8]. The anatomy of the intestinal circulation is discussed in detail elsewhere. (See "Acute mesenteric ischemia", section on 'Intestinal circulation'.)

PATHOPHYSIOLOGY — Colonic ischemia is usually the result of a sudden, but usually transient, reduction in blood flow, the effects of which are particularly prominent at the "watershed" regions of the colon. Prolonged severe ischemia causes necrosis of the villous layer, which can lead to transmural infarction within 8 to 16 hours [9]. Perfusion to the colon can be compromised by changes in the systemic circulation or by anatomic or functional changes in the local mesenteric vasculature.

Three main mechanisms are responsible for intestinal ischemia. (See "Acute mesenteric ischemia", section on 'Response to acute ischemia'.)

- Nonocclusive colonic ischemia – Nonocclusive ischemia is the predominant mechanism (95 percent of cases) causing colonic ischemia. Nonocclusive colonic ischemia most commonly affects the "watershed" areas of the colon that have limited collateralization, such as the splenic flexure and rectosigmoid junction (figure 4) [6, 7]. In a study of more than 1000 patients, the left colon was involved in approximately 75 percent of patients, with about one-quarter of lesions affecting the splenic flexure [8]. The rectum was involved in only 5 percent of patients, which can be explained because of collateralization of the inferior mesenteric artery with the systemic circulation through the hemorrhoidal vessels. Low flow states can also reduce perfusion to cause ischemia to the distal ileum and right colon related to their greater distance from the aorta. (See "Nonocclusive mesenteric ischemia").

- Embolic and thrombotic arterial occlusion – Colonic ischemia can be due to spontaneous emboli from a proximal source to the mesenteric vessels or emboli that result from aortic instrumentation. Colonic ischemia can also be related to inferior mesenteric artery ligation during aortic repair, typically in the setting of prior colon resection that has altered the normal arterial anatomy of the colon. In many patients with colonic ischemia, a specific occluding lesion of a major artery to the colon cannot be identified on arteriography, particularly if there is no concomitant small bowel ischemia (ie, superior mesenteric artery occlusion). A case-control study involving 60 patients with segmental colonic ischemia suggested that a potential cardiac source of embolism was present in up to one-third of patients [10].

- Mesenteric vein thrombosis – Mesenteric vein thrombosis rarely involves the colon; when present, it almost always involves the distal small intestine/proximal colon [11]. Phlebosclerotic colitis is a rare form of ischemic colitis that results from venous obstruction caused by fibrotic sclerosis and calcification of the walls of the mesenteric veins [12].

The colon is relatively vulnerable to hypoperfusion since it receives less blood flow compared with the rest of the gastrointestinal tract. In addition, the microvascular plexus of the colon is less developed and is embedded in a relatively thick wall compared with the small bowel.

The injury to the colon following an ischemic event is due to hypoxia and the sequelae of reperfusion. The hypoxic component causes detectable injury in the superficial part of the mucosa within one hour. The reperfusion component of intestinal injury is mainly seen following partial ischemia. It is initiated by an increased release of oxygen free radicals, other toxic byproducts of ischemic injury, and neutrophil activation [13]. Reperfusion injury can lead to multigain organ failure.

The degree to which acquired or hereditary hypercoagulable states contribute to the pathogenesis of colonic ischemia is not well understood. One report compared the prevalence of a variety of hypercoagulable states among 36 patients with colonic ischemia, 18 patients with diverticulitis, and 52 healthy controls [14]. The prevalence of antiphospholipid antibodies was significantly higher among patients with colonic ischemia compared with the patients with diverticulitis and controls (19.4 versus 0 and 1.9 percent, respectively). Factor V Leiden mutations were also found more frequently in patients with colonic ischemia (22.2 versus 0 and 3.8 percent, respectively). One or several prothrombotic abnormalities were present in 26 patients (72 percent). Another study focused on 19 patients with nonocclusive colonic ischemia who were younger than 55 years old [15]. Compared with a group of 52 matched healthy controls, patients with colonic ischemia were significantly more likely to have polymorphisms in factor V Leiden and plasminogen activator inhibitor that may predispose to thrombophilia.

The high prevalence of thrombophilic abnormalities in this study must be reconciled with the observations that patients generally present with colonic ischemia in later life (mean age 65) and that recurrence of colonic ischemia is uncommon [16]. This suggests that other factors must be involved in precipitating an ischemic event (a "two hit" hypothesis). It is also possible that specific types of thrombophilic disorders may predispose to particular forms of colonic ischemia such as chronic ischemic colitis and stricture formation.

It is unclear which, if any, patients with colonic ischemia should undergo evaluation for hypercoagulability based upon the limited data that are currently available. Younger patients and those with recurrent colonic ischemia may be reasonable candidates. (See "Evaluation of the patient with established venous thrombosis").
Colonic ischemia typically occurs in well-defined clinical settings in patients with risk factors for mesenteric ischemia. However, colonic ischemia can also develop insidiously without identifiable risk factors. The main risk factors for colonic ischemia are discussed below [17]. Risk factors associated with mesenteric ischemia, in general, are discussed elsewhere and are given in the table (table 1) [16,18].

- Aortoiliac instrumentation/surgery — Aortic surgery, such as repair of abdominal aortic aneurysm, particularly ruptured aneurysm, other forms of aortoiliac reconstruction including endovascular therapies, and aortic catheterization, can lead to colonic ischemia [16,19-22]. During endovascular surgery, ischemia almost always affects the distal left colon and is related to loss of collateral flow due to inferior mesenteric artery ligation, iliac artery ligation, embolic events, vascular compression with surgical instruments, or hypotension. (See "Open surgical repair of abdominal aortic aneurysm", section on 'Bowel ischemia'.)

- Cardiopulmonary bypass — Colonic ischemia after cardiopulmonary bypass occurs in less than 0.2 percent of patients but is a lethal complication with a mortality rate of up to 23 percent [20,27]. Risk factors include older age, end-stage renal disease, valve operation, emergent bypass surgery, and severe postoperative low cardiac output [28,29]. In addition to the low flow state of bypass perfusion, the procedure exposes the patient's blood to foreign surfaces, which may lead to hypercoagulability, microemboli, alterations in cells and proteins, release of vasoactive substances, and activation of the complement cascade [30]. Long bypass times, use of inotropic agents, and an intraaortic balloon pump are associated with increased severity of colonic ischemia [31].

- Myocardial infarction — Colonic ischemia was described in 14 of 100 patients who underwent a colonoscopy within a mean of 15 days after a myocardial infarction [32]. The indications for colonoscopy were overt or occult bleeding in most patients. One report suggested that ischemic colitis developing in the setting of myocardial infarction was associated with more complications and a worse in-hospital prognosis compared with other causes of ischemic colitis [33].

- Hemodialysis — Colonic ischemia in the setting of hemodialysis is typically nonocclusive and is due to underlying atherosclerosis, diabetes, and hemodialysis-induced hypotension [34-37]. (See "Gastrointestinal disease in dialysis patients".)

- Acquired or hereditary thrombophilia — The prevalence of thrombophilic abnormalities in patients with colonic ischemia is increased [14,15]. (See 'Pathophysiology' above.)

- Drugs (table 1) [17,38]

- Extreme exercise — Extreme exercise (as occurs in marathon running or triathlon competition) has been associated with intestinal ischemia. The ischemia is probably triggered by shunting of blood flow away from the splanchnic circulation accompanied by dehydration, hyperthermia, and electrolyte abnormalities including hyponatremia and hypokalemia.

**CLINICAL MANIFESTATIONS** — The clinical manifestations of colonic ischemia vary depending upon the clinical setting and onset, duration, and extent of the ischemia. It may be difficult to identify symptoms in patients who are unconscious, such as those in an intensive care unit [39]. A systematic review suggested rectal bleeding, peritonitis, renal failure, and right-sided colitis as the most significant predictors of disease severity [40].

**Acute colonic ischemia**

**Symptoms and signs** — Patients with acute colonic ischemia usually present with rapid onset of mild abdominal pain and tenderness over the affected bowel, most often involving the left side. Mild to moderate amounts of rectal bleeding or bloody diarrhea usually develop within 24 hours of the onset of abdominal pain, though bleeding without prior abdominal pain also occurs frequently. Bleeding may be more common with ischemia of the left compared with the right colon (83.8 versus 36.4 percent in one study [41]). About 15 percent of patients have abdominal pain without evidence of bleeding. Compared with ischemia affecting the small intestine, the pain that accompanies colonic ischemia usually is not as severe, is felt laterally rather than periumbilically, and often is associated with hematochezia. (See 'Large vs. small bowel ischemia' below.)

As a general rule, three progressive clinical stages have been described [42,43]:

- Hyperactive phase: Soon after occlusion or hypoperfusion, severe pain dominates with frequent passage of bloody, loose stools. Blood loss is usually mild without the need for transfusion.

- Paralytic phase: The pain usually diminishes, becomes more continuous, and diffuses. The abdomen becomes more tender and distended without bowel sounds.

- Shock phase: Massive fluid, protein, and electrolytes start to leak through a damaged, gangrenous mucosa. Severe dehydration with shock and metabolic acidosis may develop, requiring rapid surgical intervention. Fortunately, this most severe form affects only 10 to 20 percent of patients.

Passage of bloody stools, unexplained failure to improve, lactic acidosis, fever, leukocytosis, or thrombocytopenia should raise suspicion for the diagnosis following aortoiliac instrumentation or surgery [44,45].

Colonic infarction develops as a consequence of severe hypoperfusion leading to transmural necrosis of the bowel wall, which can progress to sepsis, peritonitis, free intraabdominal air, or extensive gangrene. In a review of 364 patients, peritoneal signs were present in only 7.4 percent of patients [41].

**Laboratory studies** — There are no specific laboratory markers for ischemia, although an increased serum lactate, LDH, CPK, or amylase may indicate advanced tissue damage. Decreased hemoglobin levels may reflect intestinal blood loss. White blood count above 20,000 µL and metabolic acidosis in a patient with signs and symptoms of acute colitis are highly suggestive of intestinal ischemia with infarction. In a multicenter review, albumin levels below 2.8 g/L were present on admission in 23.2 percent, and more common in those with gangrenous changes [41].

**Abdominal imaging**

**Plain films** — A plain abdominal x-ray is frequently nonspecific. Distension or pneumatisos (image 1) are typically seen only in advanced ischemia. In one series of 23 cases, specific signs such as thumbprinting (indicating submucosal edema) and hemorrhage could be identified in only 30 percent of patients with mesenteric infarction [46]. However, when present, radiographic findings suggesting ischemia may portend a worse prognosis, as illustrated in one study in which patients with a normal abdominal x-ray appeared to have a lower mortality compared with those with abnormal findings (29 versus 78 percent) [47].

**Abdominal CT** — Computed tomography (CT) of the abdomen with intravenous contrast is usually the first diagnostic test obtained in patients presenting with features of colonic ischemia. CT findings are generally nonspecific and scans may initially be normal [48-50]. The typical finding of thickening of the bowel wall in a segmental pattern (image 2) is not specific for ischemia but can be seen in infectious colitis such as from Clostridium difficile or Crohn's colitis. Other findings that may be seen on CT scan include a "target" or "double halo" appearance caused by hyperdensity of the mucosa and muscularis with submucosal edema, irregular bowel contours, mesenteric inflammation with stranding of the fat, or free peritoneal fluid. Pneumatosis colli (image 2), gas in the mesenteric or portal veins, or pneumoperitoneum indicating perforation may be seen in the more advanced stages but are also not specific to ischemic colitis.
Hepatic portal venous gas (HPVG) is a rare radiographic finding that has been associated with extended bowel necrosis and fatal outcome. In a patient with additional clinical and radiographic evidence of necrotic bowel, HPVG predicts a high risk of mortality (>50 percent) [51]. However, HPVG can also be detected by CT in more benign situations (eg, after surgical or endoscopic manipulation). Urgent exploratory laparotomy is mandatory only in a patient in whom intestinal ischemia or infarction is suspected on the basis of both radiologic and clinical findings.

**Chronic ischemic colitis** — Patients with episodes of chronic recurrent colonic ischemia can present with recurrent bacteremia, persistent sepsis, symptomatic colonic strictures, bloody diarrhea, weight loss from protein losing enteropathy, or recurrent abdominal pain [52]. The protracted time course and less severe symptoms distinguish these patients from those with acute colonic ischemia.

Patients with chronic ischemic colitis who are misdiagnosed as having inflammatory bowel disease will respond poorly to immunosuppressive therapy and have an increased risk of perforation on steroids.

Phlebsclerotic colitis is a rare form of ischemic colitis that results from venous obstruction caused by fibrotic sclerosis and calcification of the walls of the mesenteric veins [19]. It usually involves the right colon. Linear calcifications in the region of the right colon can be seen on plain abdominal films, while CT scan may reveal colonic wall thickening associated with mesenteric venous calcifications. Symptoms usually resolve spontaneously.

Chronic ischemic colitis in long-distance runners presents with pain in the lower abdomen, diarrhea, and mild bleeding. Treatment with rehydration and correction of metabolic abnormalities is usually sufficient since most victims are young athletes with intact cardiovascular function [53]. (See "Gastrointestinal disorders in athletes".)

**DIAGNOSIS** — A diagnosis of colonic ischemia is usually suspected based upon history, physical examination, and clinical setting. For patients who present with fulminant gangrenous colitis leading to peritonitis and/or colon perforation, a definitive diagnosis will necessarily be made in the operating room. (See ‘Abdominal exploration’ below.)

Suspicion for colonic ischemia should be increased, particularly in elderly patients, with any of the risk factors discussed above, and lower abdominal pain and hematochezia. In a multicenter study from Spain that included 364 patients with confirmed ischemic colitis, the condition was initially suspected in only about 25 percent of patients [41]. In one study, the presence of four or more risk factors (ie, older than 60, hemodialysis, hypertension, hypoalbuminemia, diabetes mellitus, constipation-inducing medications) was 100 percent predictive of colonic ischemia [54].

Among patients who do not have indications for urgent abdominal exploration, lower endoscopy (sigmoidoscopy or colonoscopy) and arteriography remain the best methods to definitively identify colonic ischemia, and differentiate it from other etiologies of ischemia. Although abdominal imaging is typically performed first, findings are nonspecific but may differentiate it from other nonischemic causes of abdominal pain, or may suggest advanced ischemia (eg, necrosis, perforation) that indicates the need for colon resection. If the diagnosis remains in question, abdominal exploration may be needed. (See ‘Abdominal CT’ above and ‘Abdominal exploration’ below.)

**Lower endoscopy** — Lower endoscopy, typically colonoscopy, can confirm a diagnosis of ischemic colitis. When indicated, lower endoscopy should be performed with minimal air insufflation to avoid excessive distention that could lead to colon perforation. (See ‘Overview of colonoscopy in adults’.)

Sigmoidoscopy is limited in its ability to reliably diagnose ischemic colon [55]. It is most commonly used in the postoperative period [56]. Some vascular surgeons recommend serial sigmoidoscopic examinations in patients in whom the bowel was considered to be at risk following aortic surgery [56]. However, no study has demonstrated that this approach is associated with improved survival [22,44,56].

Colonoscopy is sensitive for detecting mucosal lesions, permits biopsy of suspicious areas, and does not interfere with subsequent arteriography. Colonoscopic findings in the acute setting frequently include pale mucosa with petechial bleeding. Blush hemorrhagic nodules may be seen representing submucosal bleeding; these are the equivalent to "thumbnails" detected on radiological studies. More severe disease is marked by cyanotic mucosa and hemorrhagic ulcerations (picture 1). Occasional patients have pseudomembranous colitis with yellowish round plaques or confluent membranes not related to C. difficile infection [57]. Ischemia, rather than inflammatory bowel disease, is suggested by segmental distribution, abrupt transition between injured and noninjured mucosa, rectal sparing, and rapid resolution on serial endoscopy or CT scan. A single linear ulcer running along the longitudinal axis of the colon (the "single-stripe sign") may also favor an ischemic cause of colitis [58].

Biopsies taken from affected areas may show nonspecific changes such as hemorrhage, crypt destruction, capillary thrombosis, granulation tissue with crypt abscesses, and pseudopolyps, which may mimic Crohn's disease [59,60]. In the chronic phase of ischemic colitis, mucosal atrophy and areas of granulation tissue may be found. Biopsy of a post-ischemic stricture is marked by extensive transmural fibrosis and mucosal atrophy.

**Arteriography** — Arteriography is rarely helpful in the diagnosis of colonic ischemia. Arteriography is not always readily available, and patients with nonocclusive colonic ischemia are often markedly dehydrated and acidic. These patients also frequently suffer from comorbid diseases, such as cardiac and renal dysfunction, which can increase the risk of complications related to intravenous contrast injection. Thus, these patients generally need careful resuscitation before arteriography can be performed, and as a result, in many cases, colonic blood flow will have normalized.

In the absence of instrumentation or aortic surgery, the major mesenteric vessels and vascular arcades are usually patent. Ischemic changes are typically limited to the arteriolar vessels, and, when seen, may be diagnostic, but these changes are rarely seen.

However, arteriography may be needed if the clinical examination cannot exclude small bowel ischemia, and lower endoscopy is not revealing. (See ‘Large versus small bowel ischemia’ below and ‘Acute mesenteric ischemia’, section on ‘Diagnosis’,)

**DIFFERENTIAL DIAGNOSIS**

**Large versus small bowel ischemia** — Several features distinguish acute colonic ischemia from acute mesenteric ischemia involving the small bowel (table 2) [7]. Chronic ischemic colitis is rarely confused with chronic mesenteric ischemia. (See ‘Acute mesenteric ischemia’, section on ‘Clinical manifestations’,)

- Severe pain is more likely for acute ischemia involving the small bowel compared with the colon, in which extreme pain is usually not as prominent a feature.
- The onset of pain is sudden when ischemia is caused by embolic disease. In contrast, the pain may occur more insidiously (hours to days) in patients with thrombotic causes, vasculitis, or nonocclusive ischemia.
- In patients with small bowel obstruction leading to ischemia, pain often precedes vomiting.

**Other** — The differential diagnosis of colonic ischemia also includes infectious colitis, inflammatory bowel disease, diverticulitis, radiation enteritis, solitary rectal ulcer syndrome, and colon carcinoma. Stool cultures for Salmonella, Shigella, Campylobacter, Yersinia, E-coli O157:H7, and ova and parasites to exclude parasites should be considered except in patients who developed symptoms more than 72 hours after hospitalization in whom stool cultures are likely to be of low yield [61,62]. Clostridium difficile infection should be excluded in hospitalized patients exposed to antibiotics. This infection produces marked thickening of the colon on CT scan as well as very high total white blood cell counts, which
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resemble the findings of ischemic colitis. However, bloody stools are quite rare in C. difficile infection.

Infection with Klebsiella oxytoca has been associated with right-sided hemorrhagic colitis that can mimic ischemic colitis. This rare infection occurs in patients exposed to antibiotics, particularly penicillin derivatives. The organism produces a cytotoxin that is thought to be involved in the pathogenesis of colitis [63, 64]. Diagnosis is established by culture.

TREATMENT — Treatment of acute colonic ischemia depends upon its etiology, severity, and the clinical setting.

Supportive care — Supportive care is appropriate in the absence of colonic gangrene or perforation. Intravenous fluids should be given to ensure adequate colonic perfusion, and patients should be placed on bowel rest.

A nasogastric tube should be inserted if an ileus is present. Any medications (eg, vasopressors, digitalis) that can promote ischemia should be promptly discontinued, if feasible. Cardiac function and oxygenation should be optimized.

Most patients with nonocclusive ischemia improve within one or two days, and have complete clinical and radiological resolution within one to two weeks.

The patient should be monitored for persistent fever, leukocytosis, peritoneal irritation, protracted diarrhea, or gastrointestinal bleeding. If clinical deterioration is evident despite conservative therapy, abdominal exploration is indicated. (See 'Abdominal exploration' below.)

Severe ischemia can cause ulceration and inflammation, which, over time, may develop into a stricture, or chronic ischemic colitis. These lesions may be asymptomatic, but they should be followed to document healing or the development of persistent colitis or stricture, which can cause symptoms of partial bowel obstruction.

Antibiotics — Empiric broad spectrum antibiotics should be initiated in moderate to severe cases [65]. This recommendation is based mainly on older studies in which antibiotics reduced the severity and extent of experimental bowel damage when given prior to an ischemic event [66-68]. In addition, some studies suggest that antibiotics theoretically protect against bacterial translocation occurring from loss of mucosal integrity, and animal studies have suggested a potential survival advantage with antibiotics [69-71].

Antithrombotic therapy — Antithrombotic therapy is not indicated for most patients with colonic ischemia as the majority has nonocclusive ischemia.

However, anticoagulant therapy is indicated for patients who develop ischemia due to mesenteric venous thrombosis or due to cardiac embolism. Recanalization of the thrombosed vein has been described following long-term anticoagulant therapy [72, 73]. In addition to systemic anticoagulation, patients with mesenteric vein thrombosis should be evaluated for hypercoagulability. (See "Evaluation of the patient with established venous thrombosis").

Antiplatelet agents have not been well studied in this setting, and are generally not indicated in those without known peripheral vascular disease.

Abdominal exploration — Surgery is required in about 20 percent of cases [16]. Patients with colonic infarction and necrosis require urgent surgical intervention, which can be life-saving [68]. Other indications for abdominal exploration include endoscopic evidence of full-thickness irreversible necrosis of the colonic muscularis, or lesser degrees of ischemia in patients who do not respond appropriately to nonsurgical supportive care [16]. (See 'Supportive care' above.)

Prior to abdominal exploration (open or laparoscopic), bowel preparation should not be used as it can precipitate perforation or toxic dilatation of the colon.

For patients who do not have contraindications, laparoscopic exploration may be appropriate to confirm the diagnosis prior to open exploration [74]. A concern about laparoscopy is the effect of pneumoperitoneum on mesenteric blood flow [75]. The intraperitoneal pressure should be lowered (about 10 mmHg) in those suspected with suspected mesenteric ischemia. (See 'Complications of laparoscopic surgery', section on 'Related to pneumoperitoneum'.)

Once the abdomen is exposed (open or laparoscopic), the bowel should be systematically inspected from the ligament of Treitz to the peritoneal reflection overlying the rectum. The serosal surface of the intestines may appear normal in early or mild ischemia. With more advanced ischemia, dark peritoneal fluid may be present in the paracolic gutters or within the pelvis. Overtly ischemic bowel will appear edematous with patchy areas of serosal hemorrhage, or gangrenous changes with or without perforation.

Specific surgical management depends upon the location of the affected colon. When the need for colectomy is identified during laparoscopic exploration, laparoscopic colectomy can be performed by those facile with the technique.

- Right-sided colonic ischemia and necrosis is managed with right hemicolectomy and primary anastomosis. Right colectomy with end-ileostomy and distal mucocutaneous fistula may be needed if perforation is associated with gross soilage [76].
- Left-sided colonic ischemia is managed with sigmoid resection or left hemicolectomy with either proximal stoma and distal mucous fistula, or Hartmann's procedure depending upon the extent of the ischemia.
- The rare patient with a fulminating type of colonic ischemia involving most of the colon and rectum may require subtotal colectomy with terminal ileostomy.

Adequate surgical margins that are beyond macroscopically involved regions should be ensured. Primary anastomosis should be avoided in patients with severe colitis. Among patients with an aortic or iliac vascular graft, primary colonic anastomosis is also contraindicated in those who require bowel resection because any subsequent anastomotic leak would contaminate the graft [77].

Second look procedure — In most cases following exploration or colonic resection, repeat exploration, ie "second look" operation (open or laparoscopic), should be performed within 12 to 24 hours to assess the viability of the remaining bowel and integrity of any anastomoses [76, 78].

Leaving the abdomen open may be needed if abdominal closure will lead to increased intraabdominal pressure, and facilitates the second-look procedure. (See "Management of the open abdomen in adults").

Vascular intervention — Local infusion of vasodilators (such as papaverine) can attenuate vasospasm, but systemic side effects often limit its use in patients with nonocclusive colonic ischemia. Vasodilatory therapy is discussed in more detail elsewhere. (See "Nonocclusive mesenteric ischemia", section on 'Vasodilator infusion'.)

As a general rule, embolectomy, bypass graft, or endarterectomy are not options in cases of primary colonic ischemia, which is not related to large artery obstruction. However, in selected patients with early postoperative colonic ischemia following aortic surgery, delayed reimplantation of the inferior mesenteric artery or revascularization of the hypogastric artery may be an option depending upon the severity of ischemia and anatomic suitability.

POSTOPERATIVE CARE AND FOLLOW-UP — Following abdominal exploration or colon resection, the patient should be returned to an intensive care setting for hemodynamic support and monitoring.

Ostomy closure — For patients who have undergone colectomy requiring ileostomy or colostomy, ostomy closure should be delayed for four to six months, although up to two
thirds of patients never proceed to reversal because of comorbid conditions [16, 79]. In-hospital mortality related to elective ostomy reversal was 18 percent in one study, with 35 percent of patients requiring prolonged postoperative intensive care admission [80].

Repeat endoscopy — Severe ischemia causes ulceration and inflammation, which may develop into segmental ulcerating colitis or strictures. These lesions may be asymptomatic, but they should be followed to document healing or the development of persistent colitis or stricture.

MORBIDITY AND MORTALITY — The prognosis of patients with ischemic colitis depends upon the etiology, disease severity, distribution, and comorbidities [33, 81]. As a general rule, nongangrenous colonic ischemia is associated with a low mortality [16, 62].

A systematic review of 11 studies included 1049 patients with ischemic colitis [40]. Medical management was used in 80 percent of patients with a 6 percent fatality rate. These patients had only mucosal and submucosal injury, for which symptoms resolved with conservative measures and no long-term sequelae. Surgical intervention was associated with a 40 percent mortality rate. The difference in mortality reflected predominantly the severity of illness in those who required surgery. In other studies, perioperative mortality rates in those with intestinal gangrene are similarly high, between 50 and 75 percent [21, 83-86]. For those who cannot undergo surgical resection, the disease is nearly uniformly fatal.

In a retrospective review of 273 patients with colonic ischemia and isolated right colon ischemia had a worse outcome with a fivefold higher rate of surgery and a twofold higher mortality than those with ischemia involving other areas of the colon [87]. Similarly, in a study of 313 patients with biopsy-proven ischemia, patients with left colon ischemia were less likely to require surgery and had a shorter length of stay than any other pattern of ischemic colitis [81].

About 20 percent of patients develop chronic ischemic colitis from irreversible ischemic injury [59]. The presentation of patients with chronic ischemic colitis is discussed above. (See "Chronic ischemic colitis" above.)

Ischemic strictures that produce no symptoms should be observed. Some strictures will resolve in 12 to 24 months without specific therapy. If symptoms of partial obstruction develop, segmental resection is indicated [88]. Endoscopic dilatation or stenting may be alternatives for patients who are poor surgical candidates [89-90]. However, the effectiveness of these techniques has not been substantiated in this population of patients, but rather has been primarily used to palliate malignant obstruction. (See "Enteral stents for the management of malignant colorectal obstruction".)

Recurrent episodes of bacteremia or sepsis in patients with unhealed areas of segmental colitis are indications for elective segmental colon resection.

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 topic (see "Patient information: Ischemic bowel disease (The Basics)"

SUMMARY AND RECOMMENDATIONS

• Ischemic colitis, which is defined as inadequate perfusion of the colon leading to colonic inflammation is the most frequent form of mesenteric ischemia, affecting mostly the elderly. The majority of patients have transient, nongangrenous ischemia, which resolves without sequelae. Some patients develop colonic necrosis and gangrene, which can be life-threatening. Long-term complications include persistent segmental colitis and the development of a stricture. (See "Introduction" above.)

• A number of conditions predispose the patient to colonic ischemia (table 1); however, colonic ischemia can also develop insidiously with no specific inciting cause identified. (See "Pathophysiology" above and "Risk factors" above.)

• The clinical manifestations of colonic ischemia vary depending upon the clinical setting and the extent and duration of the ischemia. Patients with acute colonic ischemia usually present with rapid onset of mild abdominal pain and tenderness over the affected bowel, most often involving the left colon. Mild to moderate amounts of rectal bleeding or bloody diarrhea usually develop within 24 hours of the onset of abdominal pain. About 20 percent of patients develop chronic ischemic colitis. (See "Clinical manifestations" above.)

• A diagnosis of colonic ischemia is usually suspected based upon history, physical examination, and clinical setting. Suspicion for colonic ischemia should be increased in patients with risk factors for colonic ischemia and lower abdominal pain and/or blood per rectum. Arteriography and abdominal exploration are rarely needed to establish the diagnosis, but may be useful when the diagnosis is unclear. (See "Diagnosis" above.)

• The differential diagnosis includes acute mesenteric ischemia affecting the small intestine, infectious colitis, inflammatory bowel disease, diverticulitis, radiation enteritis, solitary rectal ulcer syndrome, and colon carcinoma. Stool cultures for Salmonella, Shigella, Campylobacter, Yersinia, E-coli O157:H7, and assay for stool toxins of Clostridium difficile should be considered in the appropriate clinical situation. (See "Differential diagnosis" above.)

• Treatment of acute colonic ischemia depends upon its severity and the clinical setting. Hemodynamic support and monitoring in an intensive care unit is appropriate in the absence of colonic gangrene or perforation. Empiric broad spectrum antibiotics should be given for patients with moderate to severe disease. Medications that can promote ischemia should not be given. (See "Treatment" above.)

• The prognosis of patients with nonocclusive colonic ischemia depends upon the disease severity, distribution, and comorbidities. Most patients improve within one or two days, and have complete resolution within one to two weeks. More severe ischemia leads to chronic ischemic colitis or intestinal stricture. Patients with underlying comorbidities and those with right-sided ischemic colitis have a worse prognosis. (See "Morbidity and mortality" above.)

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The blood supply to the colon originates from the SMA and the IMA. The SMA arises approximately 1 cm below the celiac artery and runs inferiorly toward the cecum, terminating as the ileocolic artery. The SMA gives rise to the inferior pancreaticoduodenal artery, several jejunal and ileal branches, the middle colic artery, and the right colic artery. As a general rule, the middle colic artery arises from the proximal SMA and supplies the proximal to mid-transverse colon. However, it occasionally provides the predominant blood flow to the splenic flexure. The right colic artery arises either from a common trunk with, or just below, the middle colic artery, and supplies blood to the mid-distal ascending colon. The ileocolic artery supplies the distal ileum, cecum, and proximal ascending colon.

The IMA arises approximately 6 to 7 cm below the SMA. The IMA gives rise to the left colic artery, the sigmoid arteries, and the hemorrhoidal arteries. It is largely responsible for blood supply from the distal transverse colon to the rectum.

SMA: superior mesenteric artery; IMA: inferior mesenteric artery.
Venous drainage of the colon and rectum

The mesenteric veins parallel their corresponding arteries. The SMV drains the small intestine, cecum, ascending, and transverse colon via the jejunal, ileal, ileocolic, right colic, and middle colic veins. The IMV drains the descending colon through the left colic, the sigmoid through the sigmoid vein, and the rectum through the superior rectal vein. The IMV fuses with the splenic vein, which then joins the SMV to form the portal vein.

SMV: superior mesenteric vein; IMV: inferior mesenteric vein.

Graphic 81960 Version 3.0
An abundant collateral blood supply exists between the SMA and IMA, and the IMA and internal iliac arteries. The arcades of the SMA and IMA interconnect at the base and border of the mesentery. The connection at the base of the mesentery is called the arc of Riolan, whereas the connection along the mesenteric border is known as the marginal artery of Drummond. Ischemic damage to the rectum is rare since the rectum has a dual blood supply from the IMA and iliac arteries. Collateral flow between the IMA and iliac arteries occurs via the superior and middle/inferior rectal vessels. Despite the presence of collaterals, the colon circulation has two watershed areas that are vulnerable to ischemia during systemic hypotension: the narrow terminal branches of the SMA supply the splenic flexure, and the narrow terminal branches of the IMA supply the rectosigmoid junction.

SMA: superior mesenteric artery; IMA: inferior mesenteric artery.
Distribution of colon ischemia

* Other areas refer to combination of different regions.

### Etiology of colonic ischemia

<table>
<thead>
<tr>
<th>Mesenteric venous thrombosis</th>
<th>Iatrogenic</th>
</tr>
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<tr>
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<td>Surgical</td>
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<td>Lymphocytic phlebitis</td>
<td>Aortoiliac reconstruction</td>
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<td>Portal hypertension</td>
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<td>Renal transplant</td>
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<td><strong>Small vessel disease</strong></td>
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<td>Barium enema</td>
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<tr>
<td>Vasculitis</td>
<td>Drugs[1]</td>
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<tr>
<td>Polyarteritis nodosa</td>
<td>Alosetron</td>
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<td>Lupus erythematosus</td>
<td>Digitalis</td>
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<tr>
<td>Takayasu arteritis</td>
<td>Diuretics</td>
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<tr>
<td>Granulomatosis with polyangiitis (Wegener's)</td>
<td>Cocaine</td>
</tr>
<tr>
<td>Anticentromere antibodies</td>
<td>Estrogens</td>
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<tr>
<td>Buerger's disease</td>
<td>Danazol</td>
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<tr>
<td>Antiphospholipid antibodies</td>
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<td>Amyloidosis</td>
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<tr>
<td>Rheumatoid arthritis</td>
<td>Vasoactive substances</td>
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<td>Radiation</td>
<td>Paclitaxel and carboplatin</td>
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<td><strong>Shock</strong></td>
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<td>Cardiac failure</td>
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<tr>
<td>Pancreatitis</td>
<td>Others</td>
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<tr>
<td>Anaphylaxis</td>
<td>Long distance running</td>
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<tr>
<td><strong>Mechanical obstruction</strong></td>
<td>Dialysis</td>
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<td>Strangulated hernia</td>
<td>Neurogenic</td>
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<td>Colon cancer</td>
<td>Spontaneous in young adults</td>
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<td>Adhesion</td>
<td>Infections (CMV, E. coli O157:H7)</td>
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<tr>
<td>Rectal prolapse</td>
<td>Airplane flight</td>
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<tr>
<td>Fecal impaction or pseudobstruction</td>
<td></td>
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<tr>
<td><strong>Blood dyscrasia</strong></td>
<td>Major vascular occlusion</td>
</tr>
<tr>
<td>Hypercoagulable state</td>
<td>Mesenteric artery thrombosis</td>
</tr>
<tr>
<td>Sickle cell disease</td>
<td>Cholesterol emboli</td>
</tr>
</tbody>
</table>

NSAIDs: nonsteroidal antiinflammatory drugs; CMV: cytomegalovirus; E. coli: Escherichia coli; IMA: inferior mesenteric artery.

Reference:
Pneumatosis intestinalis of the colon on plain film

A plain film of the abdomen demonstrates air within the wall of the ascending colon (arrow). This can be seen in ischemic bowel, but may also occur after colonoscopy or surgical anastomoses, and in patients taking corticosteroids or with HIV infection.

Courtesy of Jonathan Kruskal, MD.

Graphic 81253 Version 3.0
Colonic ischemia on computed tomography

The CT scan of the abdomen is from a 72-year-old female who presented with acute abdominal pain. Image A is an axial projection through the cecum and image B shows the cecum and ascending colon reformatted in the coronal plane. The images show an accumulation of air within the wall of the cecum (orange, large arrows), a finding that is characteristic of pneumatosis coli. Associated radiologic findings include: the lack of formed stool in the colon reflecting loss of mucosal function, thickening of the peritoneum and lateral coronal fascia (blue, arrowhead), and ascites (white, small arrow). No portal venous air was evident. In the appropriate clinical setting, the findings are highly suggestive of acute ischemia of the large bowel.

Graphic 86279 Version 1.0
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.

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.
### Clinical features of acute colonic and small bowel ischemia

<table>
<thead>
<tr>
<th>Acute colonic ischemia</th>
<th>Acute mesenteric ischemia involving small bowel</th>
</tr>
</thead>
<tbody>
<tr>
<td>90 percent of patients over age 60 years</td>
<td>Age varies with etiology of ischemia</td>
</tr>
<tr>
<td>Acute precipitating cause is rare</td>
<td>Acute precipitating cause is typical</td>
</tr>
<tr>
<td>Patients do not appear severely ill</td>
<td>Patients appear severely ill</td>
</tr>
<tr>
<td>Mild abdominal pain, tenderness present</td>
<td>Pain is usually severe, tenderness is not prominent early</td>
</tr>
<tr>
<td>Rectal bleeding, bloody diarrhea typical</td>
<td>Bleeding uncommon until very late</td>
</tr>
<tr>
<td>Colonoscopy is procedure of choice</td>
<td>MRA or MDCT angiography may be considered as the initial diagnostic test, angiography is recommended if there is strong clinical suspicion</td>
</tr>
</tbody>
</table>

MDCT: multidetector row computed tomography; MRA: magnetic resonance angiography.


Graphic 62738 Version 5.0
Disclosures

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