Overview of intestinal ischemia in adults

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INTRODUCTION

Intestinal ischemia, which can affect the small or large intestine, can be caused by any process that reduces intestinal blood flow, such as arterial occlusion, venous occlusion, or arterial vasospasm. For patients with acute symptoms, a rapid diagnosis is imperative since the clinical consequences can be catastrophic, including sepsis, bowel infarction, and death.

An overview of intestinal ischemia and differentiation of the various forms of intestinal ischemia are reviewed here. Specific etiologies of intestinal ischemia are reviewed in more detail elsewhere.

- (See "Acute mesenteric arterial occlusion".)
- (See "Chronic mesenteric ischemia".)
- (See "Nonocclusive mesenteric ischemia".)
- (See "Mesenteric venous thrombosis in adults".)
- (See "Colonic ischemia".)

CLASSIFICATION

Intestinal ischemia can be classified according to the time course of onset and quality of symptoms, the degree to which blood flow is compromised, and the segment of bowel that is affected. Ischemia affecting the small intestine is generally referred to as mesenteric ischemia, while ischemia affecting the large intestine is referred to as colonic ischemia. A broader term, splanchnic (visceral) ischemia, encompasses ischemia affecting the intestine, as well as other abdominal organs such as the liver, spleen, or kidneys.

- Acute mesenteric ischemia refers to the sudden onset of small intestinal hypoperfusion, which can be due to occlusive or nonocclusive obstruction of the arterial blood supply or obstruction of venous outflow. Occlusive arterial obstruction is due to an acute embolism or thrombosis and most commonly affects the superior mesenteric artery (SMA). Venous thrombosis is due to obstruction of the intestinal outflow tract, including the superior and inferior mesenteric veins and the splenic and portal veins. Nonocclusive mesenteric ischemia is a result of a low-flow state and is most commonly due to vasoconstriction from low cardiac output or the use of vasopressors.

- Chronic mesenteric ischemia usually develops in patients with mesenteric atherosclerosis causing episodic intestinal hypoperfusion related to eating.

INTESTINAL VASCULAR ANATOMY

The arterial supply to the intestines consists primarily of the superior mesenteric artery (SMA) and inferior mesenteric artery (IMA) (figure 1 and figure 2). The venous drainage parallels the arterial circulation and drains into the portal venous system (figure 3 and figure 4).

The SMA supplies the entire small intestine except for the proximal duodenum (figure 1 and figure 3).

The SMA and IMA both supply the colon (figure 1 and figure 4).

The celiac artery, which provides blood flow to the liver and spleen, also provides collateral blood flow to the intestines.

Collateral circulation — An extensive collateral circulation protects the intestines from transient periods of inadequate perfusion. However, prolonged reduction in splanchnic blood flow leads to vasoconstriction in the affected vascular bed and eventually reduces collateral blood flow [1,2].

The major collateral pathways include (figure 5):

- The celiac axis and the SMA communicate principally through the junction of the superior and inferior pancreaticoduodenal arteries (figure 6). Several smaller conduits have also been described. Because of the rich collaterals around the stomach, gastric ischemia is rare.

- The SMA and IMA communicate via the marginal artery of Drummond and the meandering mesenteric artery. The marginal artery of Drummond represents the major collateral arcade and is composed of branches from the right, middle, and left colic arteries [1,2]. It is located within the mesentery of the colon lying adjacent to the colonic wall and runs the entire length of the colon. The meandering mesenteric artery is an inconstant communication between the SMA and IMA that is variably described and is also referred to as the central communicating artery, and historically referred to as the arc of Riolan [3,4].

- Collateralization between the IMA and systemic circulation occurs in the rectum as the superior rectal (hemorrhoidal) vessels merge with the middle rectal vessels from the internal iliac arteries.

Areas prone to ischemia — The watershed areas between the major vessels that supply the colon are at risk for ischemia. Narrow terminal branches of the SMA supply the splenic flexure, and narrow terminal branches of the IMA supply the rectosigmoid junction.

- Splenic flexure – The marginal artery of Drummond is often very small, and in 11 percent of patients, it is devoid of vasa recta for a length of approximately 1 to 3 cm [5]. Griffiths' point is defined as the site of communication of the ascending left colic artery with the marginal artery of Drummond, and anastomotic bridging between the right and left terminal branches of the ascending left colic artery at the splenic flexure of the colon [5]. It is a critical area of weakness of the blood supply of the splenic flexure that is prone to ischemia.

- Rectosigmoid junction – Another area of critical weakness in the blood supply occurs at Sudeck's point, where the descending branch of the left colic artery forms an anastomosis with the superior rectal artery [6,7].

PHYSIOLOGY AND MECHANISMS OF ISCHEMIA

Normal physiology — Changes in the resistance of mesenteric arterioles account for wide fluctuations in splanchnic blood flow. The splanchnic circulation receives between 10 to 35 percent of cardiac output, depending upon whether it is in the fed or fasted state. Although the

capillary density within the intestinal vasculature is high compared with other vascular beds, intestinal oxygen extraction is relatively low, thereby permitting sufficient oxygen to be delivered to the liver via the portal vein. As a result, intestinal blood flow must be reduced by at least 50 percent from the normal fasting level before oxygen delivery to the intestine becomes compromised [8].

Numerous control mechanisms contribute to the regulation of mesenteric vascular tone and are responsive to varying conditions such as the postprandial state or systemic hypotension [9]. Intrinsic autoregulation of blood flow is an adaptation that helps redirect blood from the gut to the brain during periods of systemic hypotension [10]. Proposed mechanisms that preserve tissue perfusion include direct arteriolar smooth muscle relaxation and a metabolic response to adenosine and other metabolites of mucosal ischemia [10].

Neural and hormonal mechanisms also contribute to the extrinsic control of intestinal blood flow. These include the sympathetic nervous system, the renin-angiotensin axis, and release of vasopressin from the pituitary gland.

Response to ischemia — The likelihood of developing intestinal ischemia depends upon the adequacy of systemic perfusion and collateral circulation, the number and caliber of the vessels that are affected, and the duration of the ischemic insult. Ischemic injury to the intestine develops when there is insufficient delivery of oxygen and nutrients required for cellular metabolism. However, intestinal injury is caused both by tissue hypoxia and reperfusion. Reperfusion injury occurs following restoration of blood flow after a period of ischemia [11]. It is a complex response characterized by release of free oxygen radicals, toxic byproducts of ischemic injury, and neutrophil activation, which can lead to multisystem organ failure [12].

Under experimental conditions, ischemic injury of the mesenteric circulation does not occur until perfusion pressure is reduced to approximately 30 mmHg or the mean mesenteric arterial pressure is reduced to 45 mmHg [13]. The intestine is able to compensate for an approximately 75 percent reduction in mesenteric blood flow for up to 12 hours without substantial injury, in part because of increased oxygen extraction and vasodilation of collateral circulation [14]. However, after a prolonged period of ischemia, progressive vasoconstriction develops in the obstructed vascular bed, increasing its pressure and reducing collateral flow [15,16]. Vasoconstriction may persist even after blood flow has been restored. Persistent ischemia can lead to full-thickness necrosis of the bowel wall and subsequent perforation.

The colonic circulation is vulnerable to hypoperfusion since it receives relatively less blood flow compared with the rest of the gastrointestinal tract. However, an observational human study performed during intestinal surgery suggested that the colonic epithelium may be more

resistant to ischemia compared with the jejunum [17]. In addition, the microvasculature plexus of the colon is less developed and is embedded in a relatively thick wall as compared with the small bowel. In the majority of patients with colonic ischemia, a specific occlusive vascular lesion cannot be identified on angiography. Approximately 85 percent of these patients develop nongangrenous ischemia, which is usually transient and resolves without surgery or further complications [18].

Etiologies of ischemia — The major etiologies of mesenteric ischemia are mesenteric arterial embolism (50 percent), mesenteric arterial thrombosis (15 to 25 percent), mesenteric venous thrombosis (5 percent), and nonocclusive mesenteric ischemia due to intestinal hypoperfusion (20 to 30 percent) [16,19].

Both arterial and venous occlusion can lead to intestinal ischemia from twisting of the bowel (ie, volvulus) around a fixed attachment (ie, adhesion, mesenteric defect) or incarceration and strangulation of intestinal contents within a hernia. Patients with excessive bowel distention from bowel obstruction can get hypoperfusion from increased venous pressure and/or venous thrombosis of the involved segment of intestine. (See "Management of small bowel obstruction in adults" and "Large bowel obstruction".)

Less frequently, acute mesenteric ischemia may also be observed in the setting of an underlying vasculitis (eg, polyarteritis nodosa) most commonly affecting the small- and medium-diameter arteries; however, it may be difficult to determine whether arterial occlusion or spasm (ie, nonocclusive ischemia) is the cause of segmental intestinal infarction from vasculitis.

Mesenteric arterial occlusion

Arterial embolism — Embolism to the mesenteric arteries is most frequently due to a dislodged thrombus from the left atrium, left ventricle, cardiac valves, or proximal aorta. (See "Acute mesenteric arterial occlusion".)

Arterial thrombosis — Acute thrombosis of the mesenteric circulation usually occurs as a superimposed phenomenon in patients with a history of chronic intestinal ischemia from atherosclerotic disease. It can also occur in the setting of abdominal trauma, infection, thrombosed mesenteric aneurysm, and aortic or mesenteric artery dissection. (See "Acute mesenteric arterial occlusion" and "Chronic mesenteric ischemia".)

Venous thrombosis — Mesenteric venous thrombosis can be either idiopathic (eg, hypercoagulable states) or from secondary causes (eg, malignancy or prior abdominal surgery). Increases in the resistance of mesenteric venous blood flow lead to bowel wall edema, and

extent of ischemia is related to the extent of venous involvement. Mesenteric venous thrombosis rarely involves the colon. (See "Mesenteric venous thrombosis in adults".)

Nonocclusive mesenteric ischemia — Nonocclusive mesenteric ischemia (NOMI) is thought to occur as a result of splanchnic hypoperfusion and vasoconstriction [20]. Nonocclusive colonic ischemia or ischemic colitis most commonly affects the "watershed" areas of the colon that have limited collateralization, such as the splenic flexure and rectosigmoid junction. (See "Nonocclusive mesenteric ischemia".)

EPIDEMIOLOGY AND RISK FACTORS

Acute insufficiency of mesenteric arterial blood flow accounts for 60 to 70 percent of cases of mesenteric ischemia [15]. The remainder is related to chronic mesenteric and colonic ischemia.

The incidence of acute mesenteric ischemia appears to be rising, which may be due, in part, to an increased awareness among clinicians and an aging population with severe cardiovascular and/or systemic disease. Another contributing factor may be due to the prolonged survival of critically ill patients.

In younger patients without cardiovascular disease, mesenteric venous thrombosis is the major cause of acute ischemia of the small bowel.

Risk factors — Risk factors for intestinal ischemia include any condition that reduces perfusion to the intestine, or that predisposes to mesenteric arterial embolism, arterial thrombosis, venous thrombosis, or vasoconstriction.

Risk factors for intestinal ischemia are listed below but vary according to the specific etiology.

- (See "Acute mesenteric arterial occlusion", section on 'Etiologies'.)
- (See "Chronic mesenteric ischemia", section on 'Etiology and associations'.)
- (See "Nonocclusive mesenteric ischemia", section on 'Risk factors'.)
- (See "Mesenteric venous thrombosis in adults", section on 'Risk factors'.)
- (See "Colonic ischemia", section on 'Risk factors'.)

The following conditions put the patient at risk for intestinal ischemia:

- **Cardiac disease** – The majority of arterial emboli originate from the heart. Cardiac embolism can be related to arrhythmia, valvular disease, ventricular aneurysm, or poor

cardiac function. Cardiac dysfunction can lead to peripheral hypoperfusion, and the treatment of certain conditions may involve the use of medications that cause vasoconstriction, leading to nonocclusive ischemia. Cardiopulmonary bypass during cardiac surgery can lead to underperfusion of the intestines, showering of microemboli, release of vasoactive substances, and alterations in coagulation [21-23]. (See "Thromboembolism from aortic plaque" and "Atrial fibrillation in adults: Selection of candidates for anticoagulation".)

- **Aortic surgery or instrumentation** – Atheroembolism can complicate cardiac catheterization, aortography, or endovascular aortic intervention. Similarly, aortic manipulation during aortic surgery can dislodge intraluminal thrombus or atherosclerotic debris, which can embolize distally into the intestinal circulation. (See "Postoperative complications among patients undergoing cardiac surgery", section on 'Physiologic complications' and "Complications of endovascular abdominal aortic repair", section on 'Ischemic complications'.)

- **Peripheral artery disease** – Patients with atherosclerotic occlusive disease of the celiac artery, superior mesenteric artery, or inferior mesenteric artery are at risk for intestinal ischemia. (See "Chronic mesenteric ischemia", section on 'Clinical presentations'.)

- **Hemodialysis** – Low flow to the intestinal circulation can lead to nonocclusive intestinal ischemia [24] or intestinal infarction [25-29]. (See "Unique aspects of gastrointestinal disease in patients on dialysis".)

- **Vasoconstrictive medications** – Many medications, as well as illicit drugs, have been implicated in the development of nonocclusive intestinal ischemia [30].

- **Acquired and hereditary thrombotic conditions** – To what extent acquired or hereditary hypercoagulable states contribute to the pathogenesis of intestinal ischemia is not well known. Up to 75 percent of patients with mesenteric venous thrombosis have an inherited thrombotic disorder [31-33]. Individuals with COVID-19 may have several complex and varied coagulation abnormalities that create a hypercoagulable state, and intestinal ischemia has been reported. (See "Overview of the causes of venous thrombosis", section on 'Inherited thrombophilia' and "Overview of the causes of venous thrombosis", section on 'Acquired risk factors' and "COVID-19: Hypercoagulability" and "Acute mesenteric arterial occlusion", section on 'Etiologies' and "Mesenteric venous thrombosis in adults", section on 'Risk factors'.)

- **Inflammation/infection** – Inflammation affecting the small or large intestines can lead to mesenteric venous thrombosis. Arterial infection can lead to the formation of aneurysms,

which can lead to thrombosis. Underlying vascular disorders, such as vasculitis, may also predispose the patient to intestinal ischemia. (See "Overview of gastrointestinal manifestations of vasculitis".)

- **Hypovolemia** – Hypovolemia reduces the circulating blood volume, leading to vasoconstriction and shunting of blood flow away from the intestines. Extreme exercise (as occurs in marathon running or triathlon competition) accompanied by dehydration can lead to intestinal ischemia.

- **Segmental ischemia** – Segmental ischemia from bowel strangulation can be due to external or internal hernias, bowel volvulus, or overdistention of the bowel (eg, bowel obstruction, superior mesenteric artery syndrome). Ischemia may also be the etiology of pain in patients with median arcuate ligament syndrome. (See "Etiologies, clinical manifestations, and diagnosis of mechanical small bowel obstruction in adults", section on 'Etiologies' and "Superior mesenteric artery syndrome" and "Celiac artery compression syndrome".)

- **Vascular compression** – Patients with retroperitoneal fibrosis or other tumors can present with abdominal symptoms due to mesenteric artery compression. (See "Clinical manifestations and diagnosis of retroperitoneal fibrosis".)

CLINICAL FEATURES

History — A careful review of the patient's personal and family history is important. A history of a prior embolic event is present in approximately one-third of patients with acute embolic mesenteric ischemia. A personal or familial history of a deep vein thrombosis or pulmonary embolism is present in approximately one-half of patients with acute mesenteric venous thrombosis [34]. Patients with acute mesenteric arterial thrombosis frequently have antecedent symptoms of chronic mesenteric ischemia, including postprandial abdominal pain, an aversion to eating, and unintentional weight loss.

Abdominal pain — Abdominal pain is the most common presenting symptom in patients with intestinal ischemia. The general evaluation of the patient with abdominal pain is discussed in detail elsewhere. (See "Evaluation of the adult with nontraumatic abdominal or flank pain in the emergency department".)

The classic clinical description for acute intestinal ischemia is "abdominal pain out of proportion to the physical examination." The onset and severity of pain depends upon the duration of occlusion and the effectiveness of the collateral circulation. Several features of the pain and its

presentation may provide clues to the etiology of the ischemia and help distinguish small intestinal from colonic ischemia (table 1):

- Pain associated with arterial embolism to the proximal superior mesenteric artery is typically sudden, severe, periumbilical, and often accompanied by nausea and vomiting. (See "Acute mesenteric arterial occlusion".)

- Patients with underlying peripheral artery disease who have a thrombotic mesenteric arterial occlusion may report worsened postprandial pain or have symptoms indistinguishable from mesenteric arterial embolism. (See "Chronic mesenteric ischemia", section on 'Clinical presentations'.)

- Patients with mesenteric venous thrombosis are more likely to present with a more insidious onset of abdominal pain that can wax and wane for a period of time before a diagnosis is established [15,35]. (See "Mesenteric venous thrombosis in adults", section on 'Anticoagulation'.)

- The severity and location of the abdominal pain that accompanies nonocclusive mesenteric ischemia (NOMI) is usually more variable than the classic severe pain of acute occlusive mesenteric ischemia. Symptoms may be overshadowed by precipitating disorders including hypotension, heart failure, hypovolemia, and cardiac arrhythmias. Thus, a high index of suspicion in older patients with risk factors for NOMI is imperative for making a prompt diagnosis. (See "Nonocclusive mesenteric ischemia", section on 'Clinical features'.)

- Patients with acute colonic ischemia usually present with rapid onset of mild abdominal pain and tenderness over the affected bowel, commonly on the left side of the abdomen. Mild to moderate amounts of rectal bleeding or bloody diarrhea typically develop within 24 hours of the onset of abdominal pain. (See "Colonic ischemia", section on 'Clinical features'.)

Patients with chronic mesenteric ischemia complain of recurrent abdominal pain after eating, which is due to an inability to increase blood flow to meet the demand of the intestine postprandially. Consequently, these patients develop food fear and can lose a considerable amount of weight. (See "Chronic mesenteric ischemia", section on 'Clinical presentations'.)

Physical examination — The abdominal examination may be normal initially or show only mild abdominal distension with no signs of peritoneal inflammation, such as rebound tenderness and guarding. Occult blood may be present in the stool. However, as bowel ischemia progresses and transmural bowel infarction develops, the abdomen becomes grossly distended, bowel

sounds become absent, and peritoneal signs develop. A feculent odor to the breath may also be appreciated. Signs consistent with dehydration and shock indicate a deteriorating clinical course.

Laboratory studies — Laboratory studies are nonspecific; while abnormal laboratory values may be helpful in bolstering suspicion for acute mesenteric ischemia, normal laboratory values do not exclude acute mesenteric ischemia and do not justify delaying urgent radiologic evaluation when clinical suspicion for acute mesenteric ischemia exists.

Findings may include a marked leukocytosis with a predominance of immature white blood cells, an elevated hematocrit consistent with hemoconcentration, and a metabolic acidosis. A useful clinical guideline is that any patient with acute abdominal pain and metabolic acidosis has intestinal ischemia until proven otherwise.

Many individual laboratory values have been examined to ascertain their utility in diagnosing mesenteric ischemia or infarction [36]. Unfortunately, most abnormalities arise only after the ischemic insult has progressed to bowel necrosis.

- A systematic review reported a pooled sensitivity for l-lactate for acute mesenteric ischemia of 86 percent (95% CI 73 to 94 percent) and a pooled specificity of 44 percent (95% CI 32 to 55 percent) [37]. The specificity of an elevated serum lactate level does improve significantly when conditions such as shock, diabetic ketoacidosis, and renal and hepatic failure can be excluded [38].

- Elevated serum amylase levels have been observed in approximately one-half of patients with intestinal ischemia [39,40], while phosphate elevations have been found in 80 percent [41].

- Normal D-dimer levels may help to exclude acute intestinal ischemia, but elevated levels are less useful for making a diagnosis [42]. Animal models of acute intestinal ischemia have demonstrated an increase in D-dimer levels beginning 30 minutes after the ischemic event [43]. However, elevated levels can also be seen in a variety of conditions, such as in patients with acute pancreatitis and those with an abdominal aortic aneurysm [44,45]. In a systematic review, the pooled sensitivity for D-dimer for acute mesenteric ischemia was 96 percent (95% CI 89 to 99 percent) with a pooled specificity of 40 percent (95% CI 33 to 47 percent) [37].

Experimental tests — Measurement of serum alpha-glutathione S-transferase (alpha-GST) intestinal fatty acid-binding protein (I-FABP) and others have been evaluated as markers of

intestinal ischemia [40,46-53]. None are widely available, and they are rarely used in clinical practice.

Plain radiographs — Plain abdominal radiography is relatively nonspecific and may be completely normal in more than 25 percent of patients [15]. Findings suggestive of mesenteric ischemia include the presence of an ileus with distended loops of bowel, bowel wall thickening (particularly prominent in acute mesenteric venous thrombosis) (image 1), and/or pneumatosis intestinalis (image 2 and image 3). The latter may be observed in patients with advanced ischemia. Obvious findings, such as free intraperitoneal air, indicate the need for immediate abdominal exploration.

DIAGNOSIS

Our diagnostic approach is given in the algorithm (algorithm 1).

Rapid diagnosis is essential among patients with clinical features and risk factors suggestive of intestinal ischemia to reduce the potential for intestinal infarction, although these are generally nonspecific [54]. Intestinal ischemia should be considered a potential diagnosis in any critically ill patient who is deteriorating without adequate explanation.

- For patients who present with peritonitis or obvious bowel perforation, the diagnosis will necessarily be made in the operating room.
- For those without indications for immediate abdominal exploration, a definitive diagnosis requires advanced abdominal imaging. We recommend computed tomographic (CT) angiography as the initial test for most patients with clinical features consistent with intestinal ischemia.
- Although duplex ultrasound can identify arterial stenosis or occlusion of the celiac or superior mesenteric arteries, the test is often technically limited by the presence of air-filled loops of distended bowel. In addition, the sensitivity of duplex is limited for detecting more distal emboli or in the assessment of nonocclusive mesenteric ischemia.
- Colonoscopy or sigmoidoscopy is often required to establish or confirm the diagnosis of ischemic colitis.

In cases of vasculitis, which typically affects the small- and medium-diameter arteries, stenoses and/or microaneurysms may be detected on arteriography typically without evidence of obstruction of the main mesenteric arteries. However, the bowel wall changes associated with vasculitis are nonspecific and simply reflect the changes one sees with varying degrees of

ischemia, including mural edema and/or hemorrhage. If vasculitis-associated aneurysms are large enough, they may be detectable on ultrasound. (See "Overview of visceral artery aneurysm and pseudoaneurysm".)

Advanced abdominal imaging — Abdominal CT is commonly used to screen hemodynamically stable patients with acute abdominal pain [55-63].

For patients in whom the index of suspicion for intestinal ischemia is high, multidetector CT angiography and magnetic resonance (MR) angiography have improved the ability to diagnose acute mesenteric ischemia [55-63]. The CT scan should be performed **without** oral contrast, which can **obscure** the mesenteric vessels, obscure bowel wall enhancement, and can lead to a **delay** of the diagnosis. The origins of the celiac axis and superior mesenteric artery should also be evaluated for the presence of calcification that indicates an underlying atherosclerotic process as a possible etiology for mesenteric ischemia.

CT is preferred over MR because of its lower costs, speed, and wide availability [57,59]. However, MR angiography may be more sensitive for the diagnosis of mesenteric venous thrombosis and may be necessary for those with an allergy to iodinated contrast. CT can demonstrate findings consistent with acute ischemia, such as focal or segmental bowel wall thickening, intestinal pneumatosis with portal vein gas (image 4), bowel dilation, mesenteric stranding, portomesenteric thrombosis, or solid organ infarction, in addition to ruling out other causes of acute abdominal pain [64,65]. It is important to note that the bowel wall changes of acute mesenteric ischemia, while sensitive, are not specific [64,65]. The presence of pneumatosis intestinalis on CT does not necessarily indicate that transmural infarction has occurred, but transmural infarction is more likely in patients with pneumatosis and portomesenteric venous gas [66]. Mesenteric arterial occlusions can be identified as a lack of enhancement of the arterial vasculature with timed intravenous contrast injections. When present, thromboembolic occlusion is specific for the diagnosis; however, the absence of a filling defect is not sufficiently sensitive to rule out acute mesenteric ischemia [64,65,67].

The accuracy of CT was evaluated in a review that identified six studies involving 619 patients, 142 of whom had acute mesenteric ischemia based upon surgical findings or clinical outcome [68]; the pooled sensitivity for CT was 93.3 percent (95% CI 83 to 98 percent), and pooled specificity was 96 percent (95% CI 91 to 98 percent). Another study compared CT findings in 39 patients who had surgically proven acute mesenteric ischemia with 24 controls in whom suspected acute mesenteric ischemia was disproved at surgery [67]. A single finding of either arterial or venous thrombosis, intramural gas, portal venous gas, focal lack of bowel-wall enhancement, or liver or splenic infarcts had a sensitivity and specificity of 64 and 92 percent, respectively. In another review, the sensitivity of CT for the diagnosis of mesenteric venous

thrombosis was approximately 90 percent [32]. In a later prospective study of 79 patients with suspected acute mesenteric ischemia evaluated using multidetector CT angiography, a positive predictive value of 100 percent and a negative predictive value of 94 percent were found using criteria that required the presence of visualized arterial occlusion, intestinal pneumatosis, portomesenteric venous gas, or bowel wall thickening, in combination with portomesenteric thrombosis or solid organ infarction [65]. The diagnosis was made by the failure to opacify the mesenteric veins with intravenous contrast. Similarly, multidetector CT angiography was used to evaluate 91 patients with suspected acute mesenteric ischemia in another retrospective review [64]. It correctly diagnosed acute mesenteric ischemia in 16 of 18 patients with confirmed acute mesenteric ischemia, while supporting another diagnosis in an additional 38 patients. There were two false negative and two false positive examinations.

More data comparing these modalities to conventional arteriography are needed, particularly to understand whether CT and MR can accurately detect the presence of small thromboemboli; early, reversible ischemia; or nonocclusive ischemia [62]. Anteroposterior and lateral views are needed to adequately assess the mesenteric vasculature. The origins of the celiac axis and SMA are visualized only with the lateral view, while the distal celiac axis and remainder of the SMA are assessed best with anteroposterior projections. One systematic review that evaluated CT angiography for acute mesenteric ischemia reported a pooled sensitivity of 94 percent (95% CI 90 to 97 percent) with a specificity of 95 percent (95% CI 93 to 97 percent) [37]. Conventional catheter-based arteriography is still recommended if the diagnosis of mesenteric ischemia remains in question (image 5) [69].

Mesenteric venous thrombosis may be diagnosed with CT angiography or conventional arteriography by performing delayed imaging to allow for contrasted filling of the mesenteric venous system. The diagnosis is made by the presence of venous filling defects or the absence of flow. Reflux of contrast into the aorta on selective arteriography may indicate a highly resistant venous system with resultant low arterial flow. Contrast extravasation into the bowel lumen indicates active bleeding. In patients with nonocclusive mesenteric ischemia (NOMI), angiography can demonstrate areas of segmental narrowing in major branches with a string-of-beads appearance, decreased or absent flow in the smaller vessels, and an absent submucosal "blush."

Differential diagnosis — Intestinal ischemia needs to be differentiated from other causes of abdominal pain. (See "Evaluation of the adult with abdominal pain" and "Causes of abdominal pain in adults".)

INITIAL MANAGEMENT

Initial management includes gastrointestinal decompression, fluid resuscitation, hemodynamic monitoring and support, correction of electrolyte abnormalities, pain control, anticoagulation under most circumstances, and initiation of broad-spectrum antibiotics [70].

Vasoconstricting agents and digitalis should be avoided since they can exacerbate mesenteric ischemia. If vasopressors are needed, dobutamine, low-dose dopamine, or milrinone are preferred since they have less of an effect on mesenteric perfusion as compared with other vasopressors.

Pain control — The patient's pain should be judiciously controlled, typically using parenteral opioids. (See "Use of opioids for postoperative pain control".)

Anticoagulation — For patients with acute intestinal ischemia due to mesenteric arterial or venous occlusion, or nonocclusive mesenteric ischemia, we recommend systemic anticoagulation to prevent thrombus formation and propagation, unless patients are actively bleeding, as in ischemic colitis related to nonocclusive ischemia. For those who require abdominal exploration, anticoagulation is typically continued after surgery to prevent new thrombus formation [16].

Antibiotics — Broad-spectrum antibiotic therapy is recommended for patients with acute mesenteric and colonic ischemia.

ABDOMINAL EXPLORATION

Intestinal ischemia develops as a consequence of severe hypoperfusion leading to transmural necrosis of the bowel wall, which can progress to sepsis, peritonitis, free intra-abdominal air, or extensive gangrene. Surgery should not be delayed in patients suspected of having intestinal infarction or perforation based upon clinical, radiographic, or laboratory parameters, regardless of etiology. For patients with nonocclusive mesenteric ischemia, surgical exploration should be limited to patients with peritoneal signs.

TREATMENT OF SPECIFIC ETIOLOGIES

The management of intestinal ischemia may require any number of treatment options. The management of specific etiologies of intestinal ischemia due to arterial occlusion or thrombosis, mesenteric venous thrombosis, and nonocclusive mesenteric ischemia are summarized briefly below and reviewed in detail elsewhere.

- Mesenteric arterial occlusion – The traditional treatment of acute mesenteric arterial embolism is early surgical laparotomy with embolectomy, which may be the preferred treatment for patients with a solitary, proximal superior mesenteric embolus, since it provides rapid treatment and allows direct inspection of the bowel. An alternative but less established approach for acute embolus, particularly in those with severe comorbidities, is local infusion of a thrombolytic agent; however, this option is reserved for patients with a shorter duration of symptoms and without signs of peritonitis. Thrombolysis risks possible catheter-related embolism to the more distal arterial mesenteric branches. For mesenteric arterial thrombosis, choices include surgical revascularization, or thrombolysis with endovascular angioplasty and stenting [71-73]. The choice depends on the time course, severity of ischemia, and medical comorbidities of the patient. (See "Acute mesenteric arterial occlusion" and "Chronic mesenteric ischemia".)

- Mesenteric venous thrombosis – Anticoagulation may be all that is needed in the treatment of patients with mesenteric venous thrombosis (algorithm 2). However, for those with persistent symptoms, venous thrombolysis has been reported in small case series. If symptoms progress, abdominal exploration may be needed to evaluate for nonviable bowel. (See "Mesenteric venous thrombosis in adults".)

- Nonocclusive mesenteric ischemia – The treatment of nonocclusive mesenteric ischemia focuses on removing inciting factors (vasoconstrictive medications), treating underlying causes (heart failure, sepsis), hemodynamic support and monitoring, and intra-arterial infusion of vasodilators, if necessary (algorithm 3). (See "Nonocclusive mesenteric ischemia" and "Colonic ischemia".)

OUTCOMES

The outcomes related to the treatment of intestinal ischemia depend upon the mechanism. Survival of an acute ischemic event is worse for patients with an arterial etiology compared with a venous etiology. For acute mesenteric ischemia, mortality rates exceed 60 percent [15]. In one systematic review, the pooled operative mortality rate for acute mesenteric ischemia was 47 percent [37]. Patients who survive an acute event are likely to die of complications related to the underlying condition that predisposed them to intestinal ischemia.

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: Intestinal ischemia".)

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 education: Ischemic bowel disease (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Intestinal ischemia** – Intestinal ischemia, which can affect the small or large intestine, can be caused by any process that reduces intestinal blood flow, such as arterial occlusion, venous occlusion, or vasoconstriction. Intestinal ischemia can be classified according to the time course of onset and quality of symptoms (acute or chronic), the degree to which blood flow is compromised, and the segment of bowel that is affected. Ischemia affecting the small intestine is generally referred to as mesenteric ischemia, while ischemia affecting the large intestine is referred to as colonic ischemia. Acute intestinal ischemia accounts for 60 to 70 percent of cases of mesenteric ischemia and results in high mortality rates that can exceed 60 percent. The remainder is due to chronic intestinal ischemia. (See 'Physiology and mechanisms of ischemia' above.)

- **Risk factors** – Risk factors for intestinal ischemia include any condition that reduces perfusion of the intestines or predisposes to mesenteric arterial embolism, arterial thrombosis, venous thrombosis, or vasoconstriction. The likelihood of developing intestinal ischemia depends upon the adequacy of systemic perfusion and collateral

circulation, the number and caliber of the vessels that are affected, and the duration of the ischemic insult. (See 'Risk factors' above.)

- **Clinical features** – Abdominal pain is the most common presenting symptom in patients with intestinal ischemia. The classic clinical description for acute intestinal ischemia is "abdominal pain out of proportion to the physical examination." The onset of pain depends upon the time course of occlusion and the effectiveness of the collateral circulation. Patients with chronic mesenteric ischemia complain of recurrent abdominal pain after eating. Specific features of the pain and its presentation may provide clues to the etiology of the ischemia and for distinguishing small intestinal from colonic ischemia (table 1). (See 'Clinical features' above.)

- **Diagnosis** – The diagnosis of mesenteric ischemia depends upon a high level of clinical suspicion, especially in patients with known risk factors. Rapid diagnosis is essential among patients with clinical features and risk factors suggestive of acute intestinal ischemia to reduce the potential for intestinal infarction. However, early signs and symptoms of acute mesenteric ischemia are nonspecific, and definitive diagnosis often requires invasive testing. We obtain computed tomographic (CT) angiography without oral contrast as an initial test for patients suspected of having mesenteric ischemia. CT angiography has a high degree of accuracy for diagnosing mesenteric ischemia and is useful in excluding other causes of acute abdominal pain. Arteriography may still be necessary if the diagnosis of mesenteric ischemia remains in question. Colonoscopy or sigmoidoscopy is used to establish the diagnosis of colonic ischemia. (See 'Diagnosis' above.)

- **Treatment** – The goal of treatment for patients with acute intestinal ischemia is to restore intestinal blood flow as rapidly as possible after initial supportive management. Patients with acute intestinal ischemia should be anticoagulated to prevent thrombus formation or propagation, provided there are no contraindications. The management of specific etiologies of acute or chronic intestinal ischemia depends on the specific etiology (ie, arterial occlusion or thrombosis, mesenteric venous thrombosis, and nonocclusive mesenteric ischemia). Treatment options include arterial embolectomy, arterial bypass, arterial stenting, arterial or venous thrombolysis, and intra-arterial vasodilator infusion. (See 'Initial management' above and 'Treatment of specific etiologies' above.)

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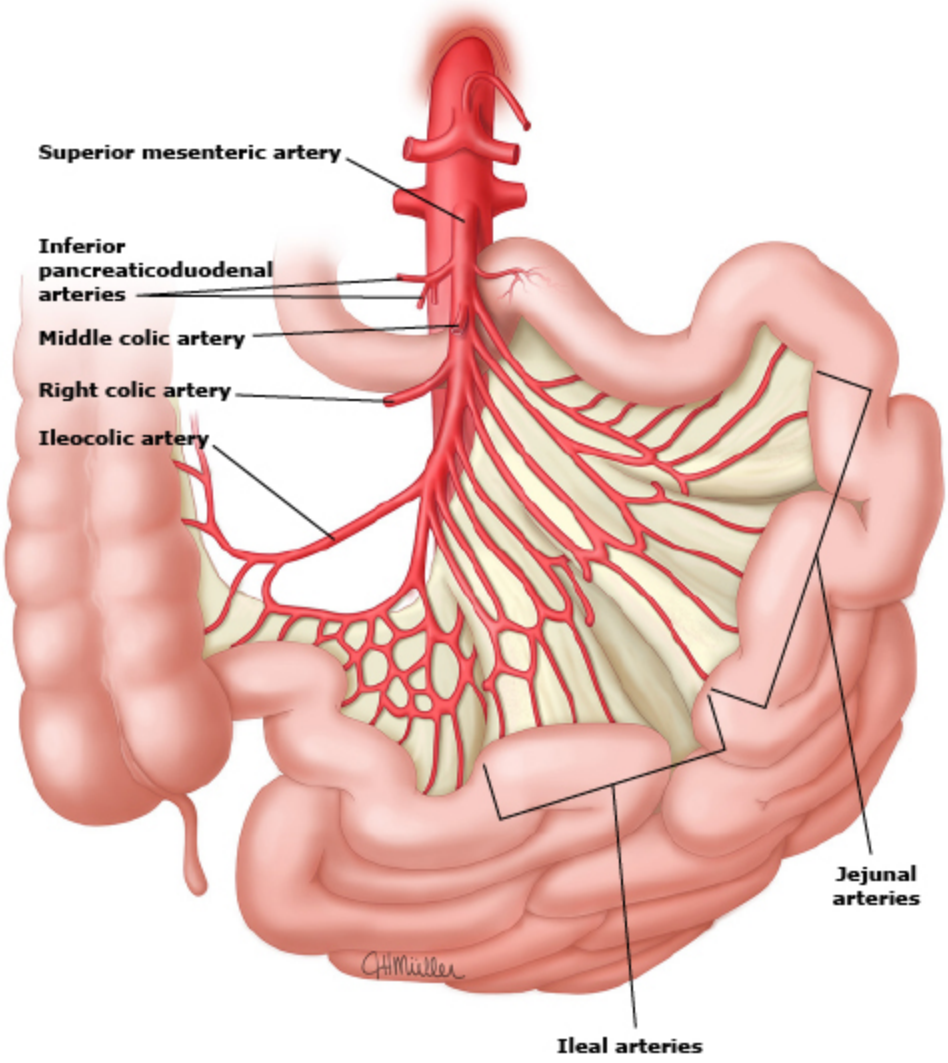
Topic 2559 Version 33.0

GRAPHICS

Blood supply to the small intestine

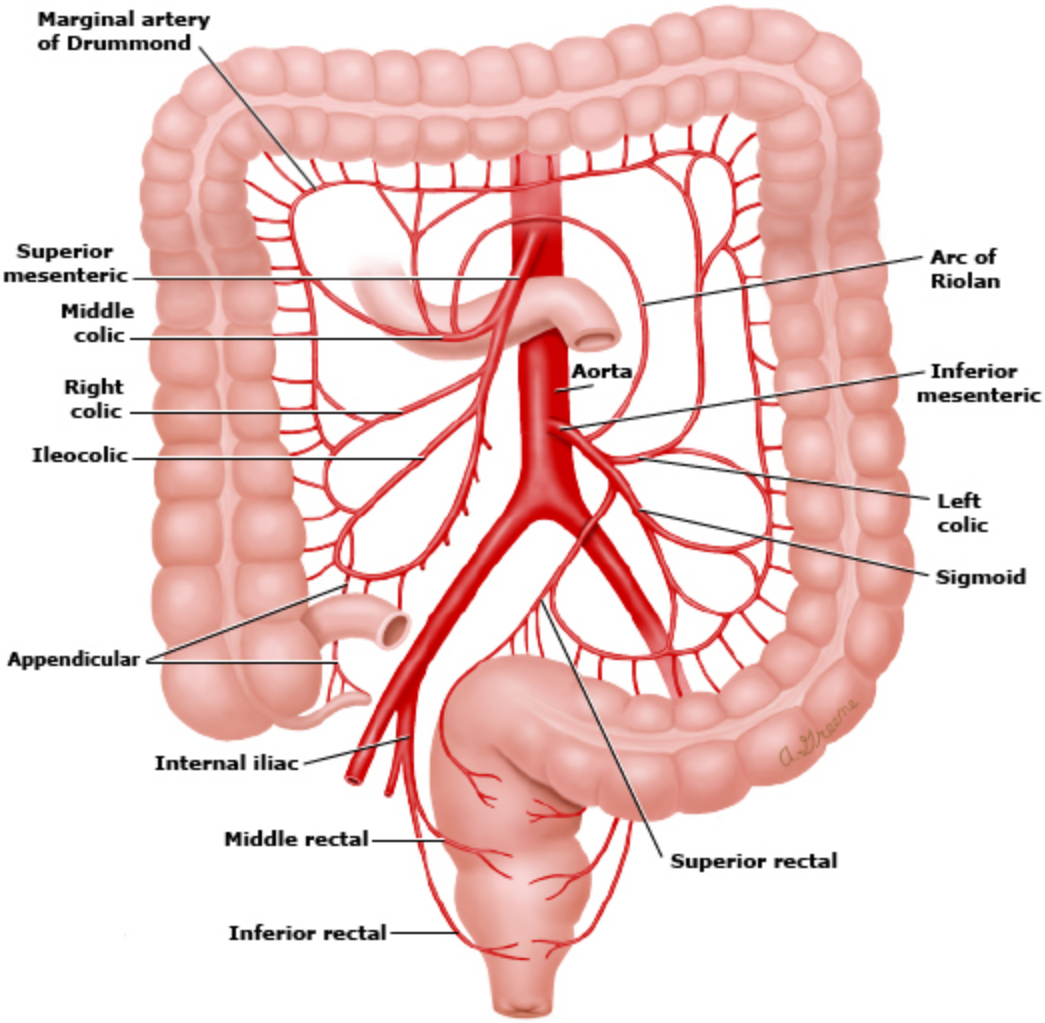


The blood supply to the small and large bowel is derived from the celiac artery and SMA. The celiac axis primarily provides blood flow to the stomach, liver, spleen, and pancreas but is also a source of collateral flow when blood flow in the SMA is reduced. The SMA gives rise to the inferior pancreaticoduodenal artery, the middle colic artery, right colic artery, and many jejunal and ileal branches. The jejunal and ileal branches supply the jejunum and ileum, respectively. The ileocolic artery supplies the distal ileum, cecum, and proximal ascending colon.

SMA: superior mesenteric artery.

Graphic 89910 Version 5.0

Blood supply to the colon and rectum



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 blood to the proximal to midtransverse colon. However, it occasionally provides the predominant blood flow to the splenic flexure.

The right colic artery supplies blood to the mid-distal ascending colon. In anatomical studies, the right colic artery arises independently from the SMA in 28% of individuals, which is depicted in this figure. More frequently, the right colic artery arises with, or as a branch of, the middle colic, ileocolic, or left colic arteries. The right colic artery is absent in 13% of individuals.[1]

The ileocolic artery supplies blood to 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 and sigmoid arteries continuing as the superior rectal (hemorrhoidal) artery. It is largely responsible for supplying blood distal to the transverse colon.

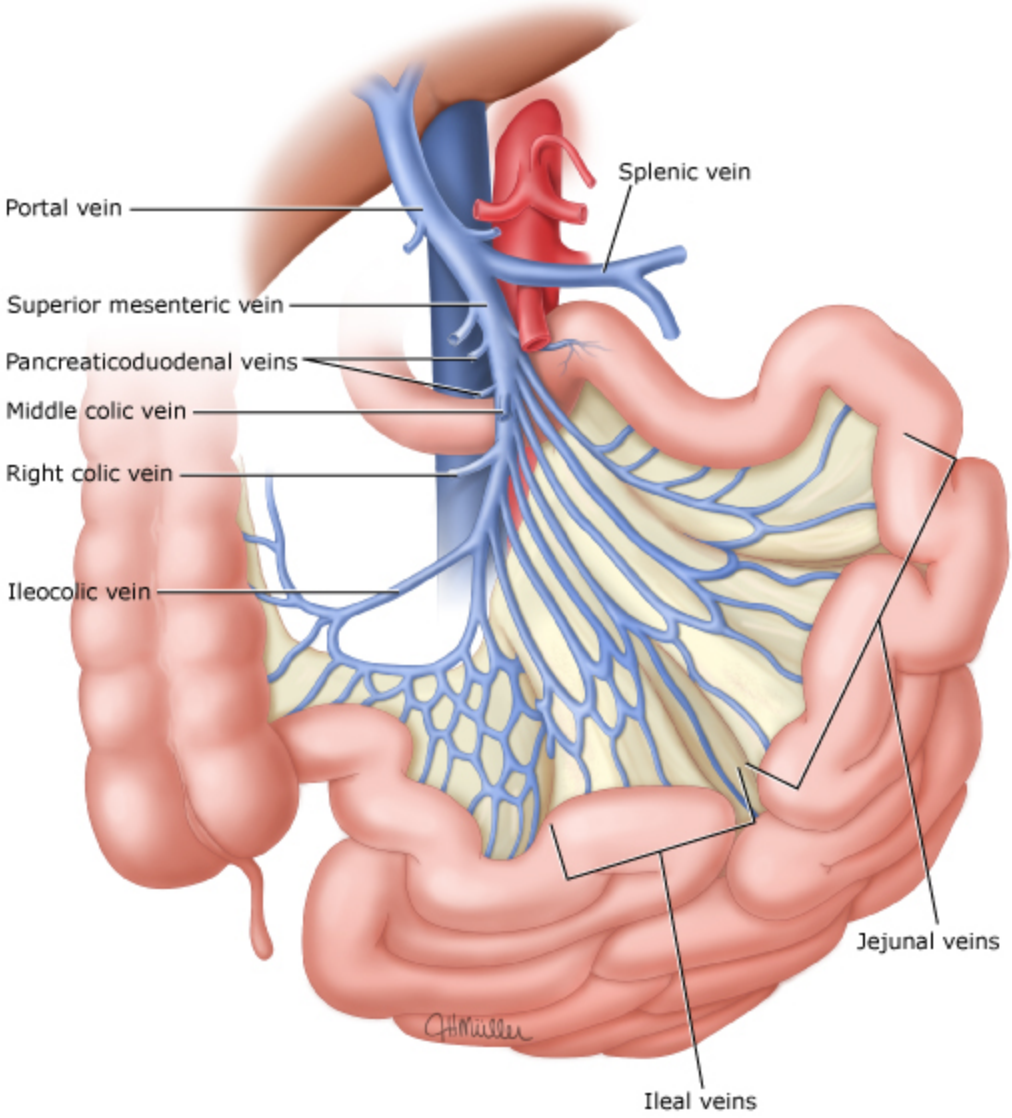
SMA: superior mesenteric artery; IMA: inferior mesenteric artery.

Reference:

1. *Bergman RA, Thompson SA, Afifi AK, Saadeh FA. Compendium of Human Anatomic Variation: Text, Atlas, and World Literature, Urban & Schwarzenberg, Baltimore, MD 1988.*

Graphic 73756 Version 12.0

Venous drainage of the small intestine

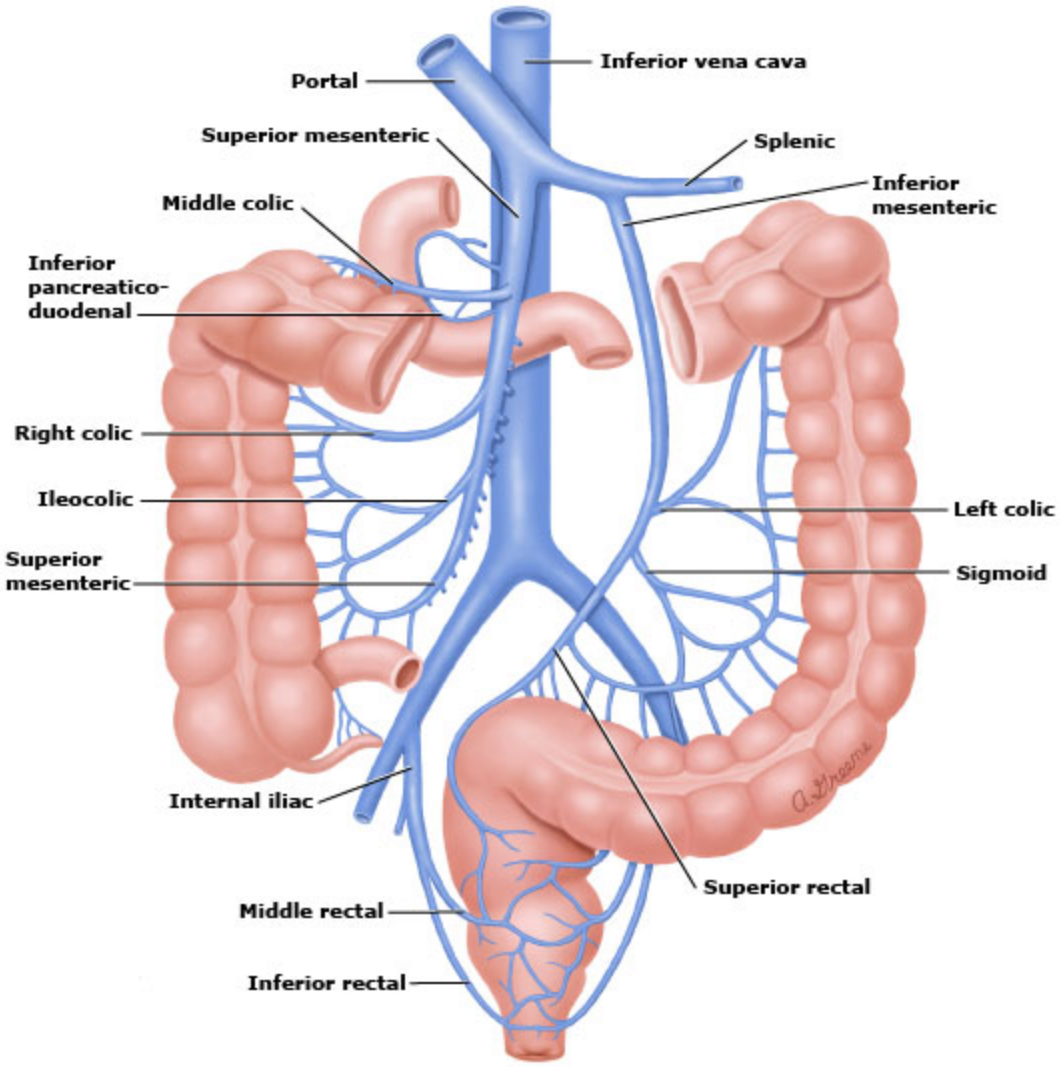


The mesenteric veins parallel their corresponding arteries. The SMV drains the small intestine, cecum, ascending, and transverse colon via the jejunal, ileal, ileocolic, right colic, and middle colic veins. The SMV joins the splenic vein to drain into the portal vein.

SMV: superior mesenteric vein.

Graphic 88949 Version 4.0

Venous drainage of the colon and rectum

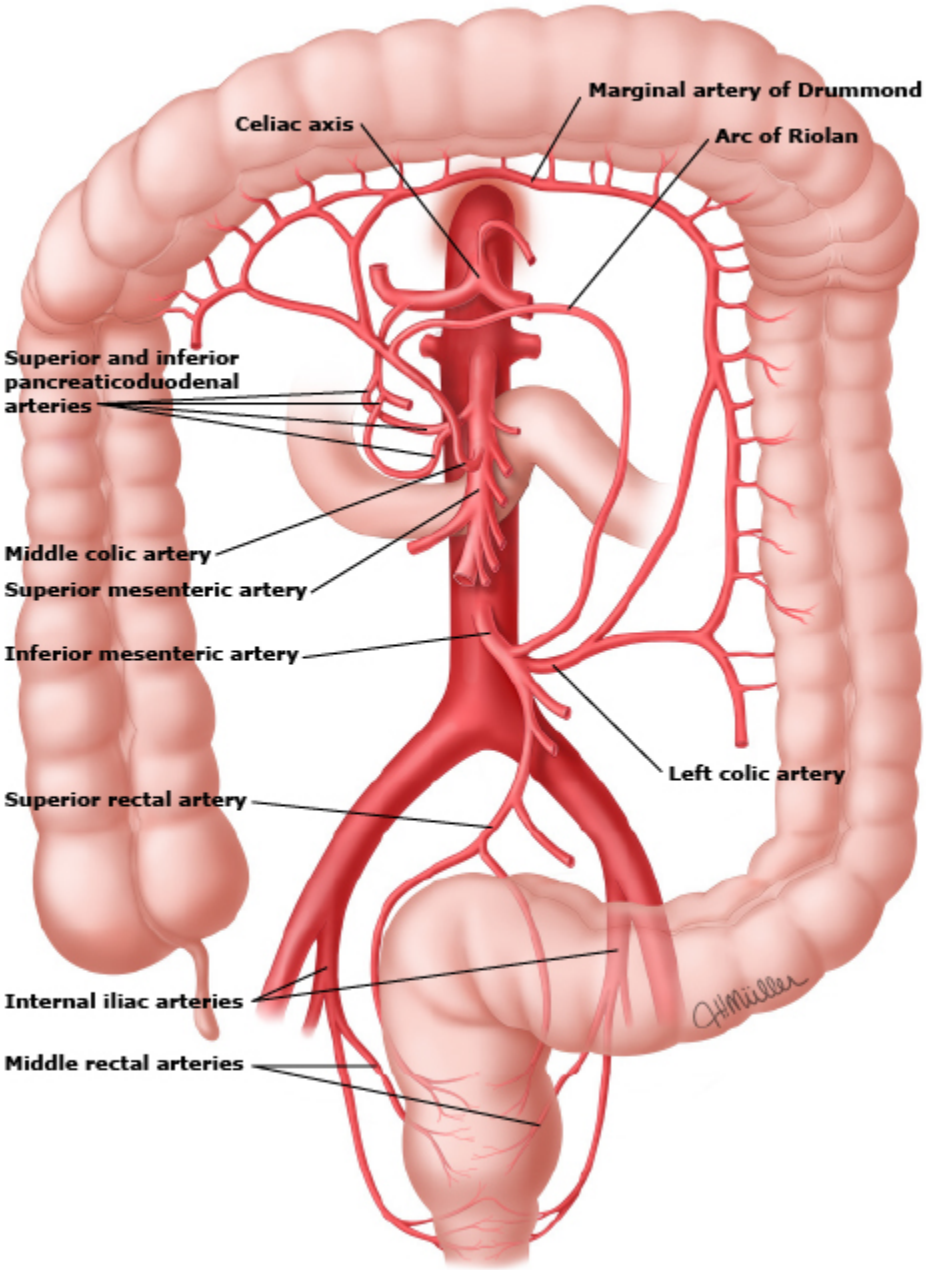


The mesenteric veins parallel their corresponding arteries. The SMV drains the small intestine, cecum, and ascending and transverse colon via the jejunal, ileal, ileocolic, right colic, and middle colic veins. The IMV drains the descending colon through the left colic, the sigmoid through the sigmoid 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 4.0

Collateral circulation to the intestines



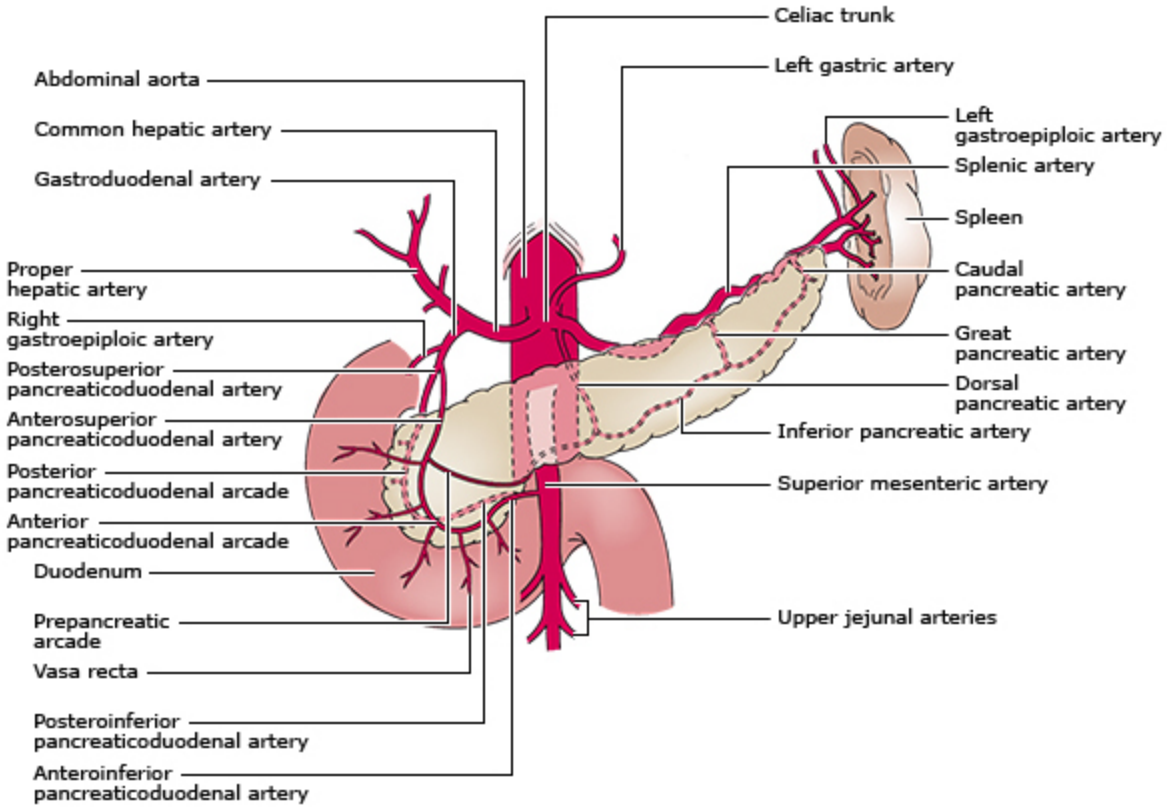
An abundant collateral blood supply exists between the SMA and IMA and the IMA and internal iliac arteries. The arcades of the SMA and IMA interconnect at the base and border of the mesentery. The connection at the base of the mesentery is called the arc of Riolan, whereas the connection along the mesenteric 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.

Graphic 89911 Version 5.0

Arterial supply to the pancreas



Reproduced from: Mulholland MW, Maier RV, et al. Greenfield's Surgery: Scientific Principles And Practice, Fourth Edition. Philadelphia: Lippincott Williams & Wilkins, 2006. Original illustration from: Woodburne RT. Essentials of Human Anatomy, New York: Oxford University Press, 1973. Copyright © 1973.

Graphic 53700 Version 1.0

Features of acute small bowel versus acute colonic ischemia

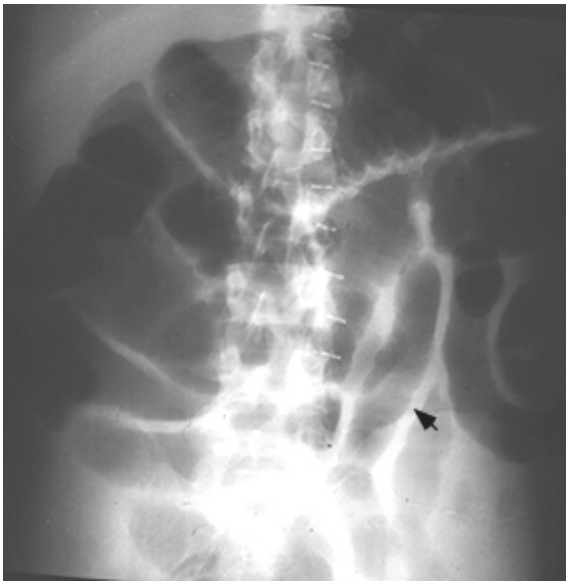
Acute small bowel ischemia	Acute colonic ischemia
Age varies with etiology of ischemia	90 percent of patients over age 60 years
Acute precipitating cause is typical	Acute precipitating cause is rare
Patients appear severely ill	Patients do not appear severely ill
Pain is usually severe, tenderness is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

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

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

Graphic 62738 Version 8.0

Small bowel ischemia on plain abdominal film

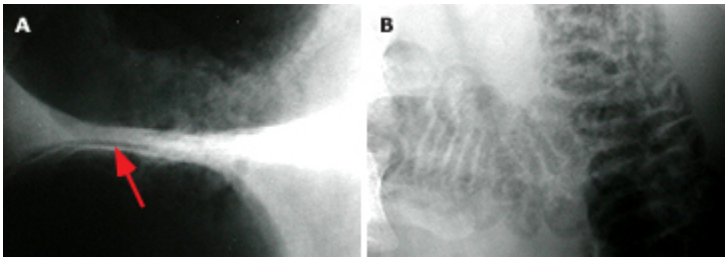


A plain radiograph of the abdomen demonstrates distended featureless loops of small bowel with wall thickening (arrow) and separation of the bowel loops. These findings are consistent with hemorrhage into the bowel wall secondary to ischemia.

Courtesy of Jonathan B Kruskal, MD, PhD.

Graphic 74055 Version 3.0

Pneumatosis intestinalis

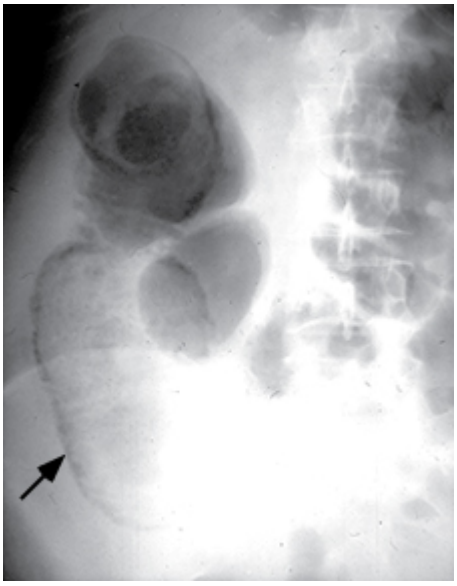


Panel A is a magnified image of a single fold in the colon demonstrating air within the wall of the colon, which appears as a thin linear lucency on this plain film (arrow). Panel B is a plain film from a 68-year-old male with ischemic bowel, demonstrating numerous linear collections of air within the bowel wall.

Courtesy of Jonathan B Kruskal, MD, PhD.

Graphic 72081 Version 2.0

Pneumatosis intestinalis of the colon on plain film



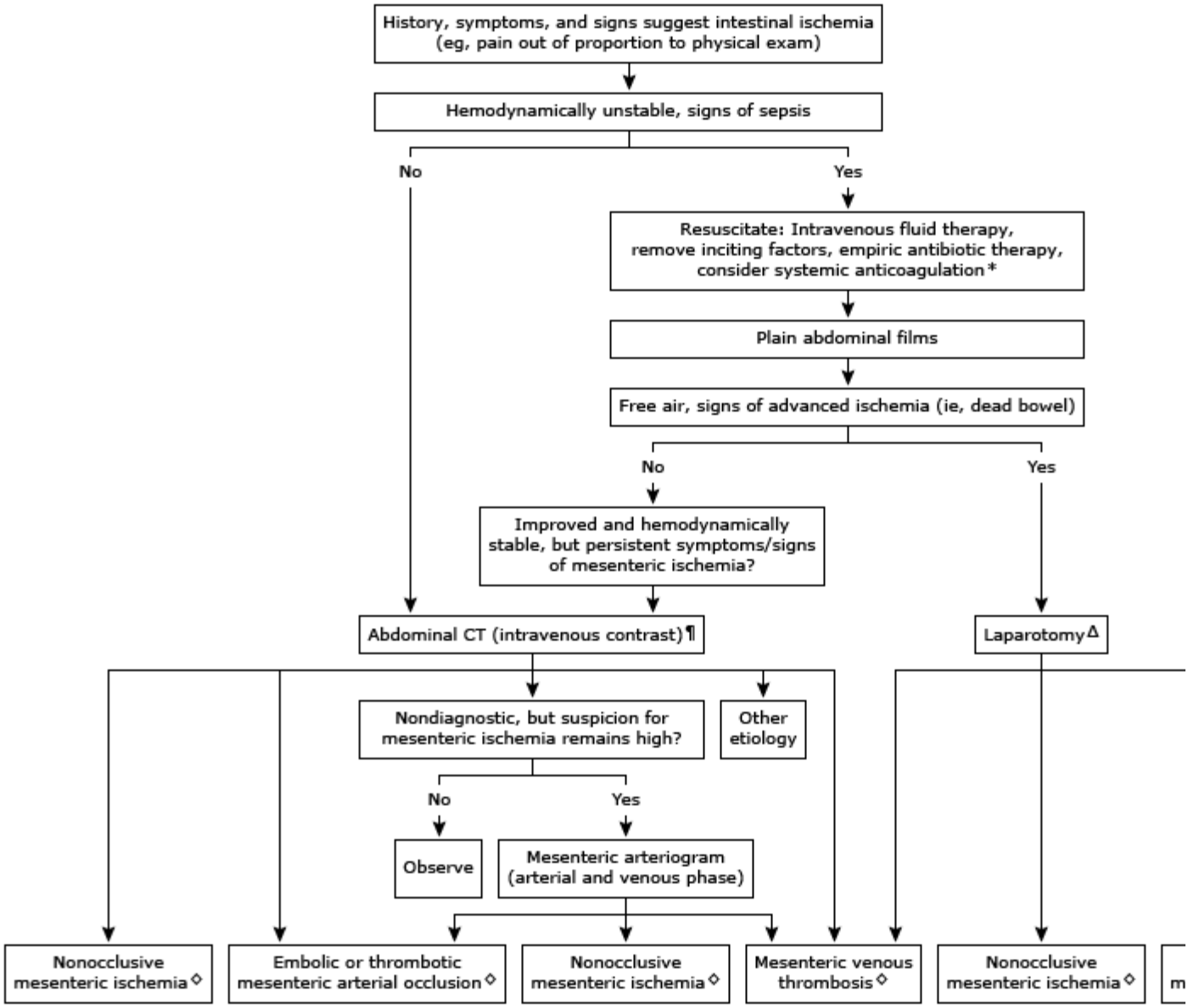
A plain film of the abdomen demonstrates air within the wall of the ascending colon (arrow). This can be seen in ischemic bowel but may also occur after colonoscopy or surgical anastomoses and in patients taking corticosteroids or with HIV infection.

HIV: human immunodeficiency virus.

Courtesy of Jonathan Kruskal, MD.

Graphic 81253 Version 5.0

Diagnosis and initial management of intestinal ischemia



CT: computed tomography.

* Patients ultimately identified with nonocclusive mesenteric ischemia will not benefit from anticoagulation, be discontinued.

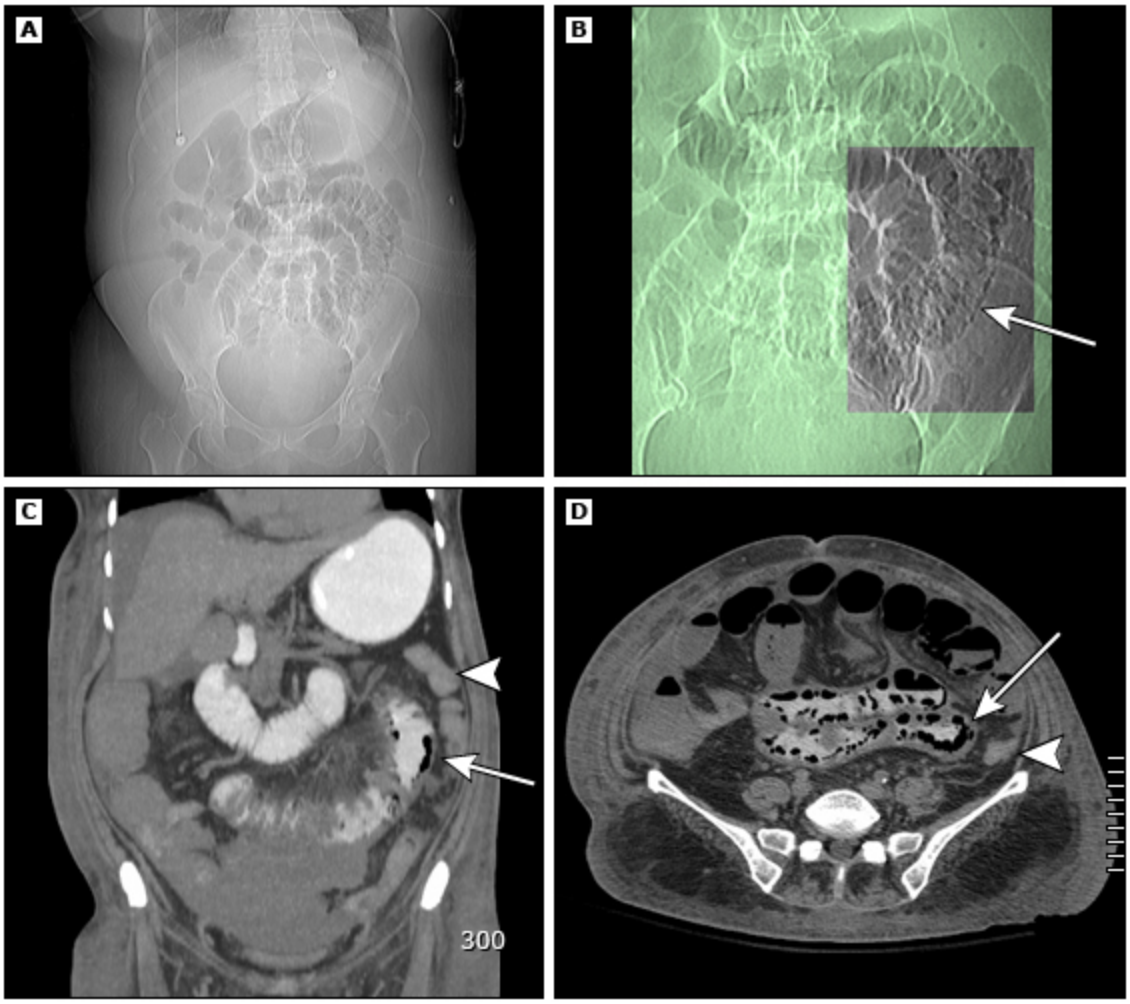
¶ Imaging signs associated with mesenteric ischemia include focal or segmental bowel wall thickening, inte portal vein gas, portomesenteric thrombosis, mesenteric arterial calcification, and mesenteric artery occlusi

Δ Medically fit patients.

◇ Refer to associated UpToDate algorithms on mesenteric ischemia (acute or chronic, occlusive or nonocclu venous).

Graphic 62760 Version 6.0

CT scan of pneumatosis coli

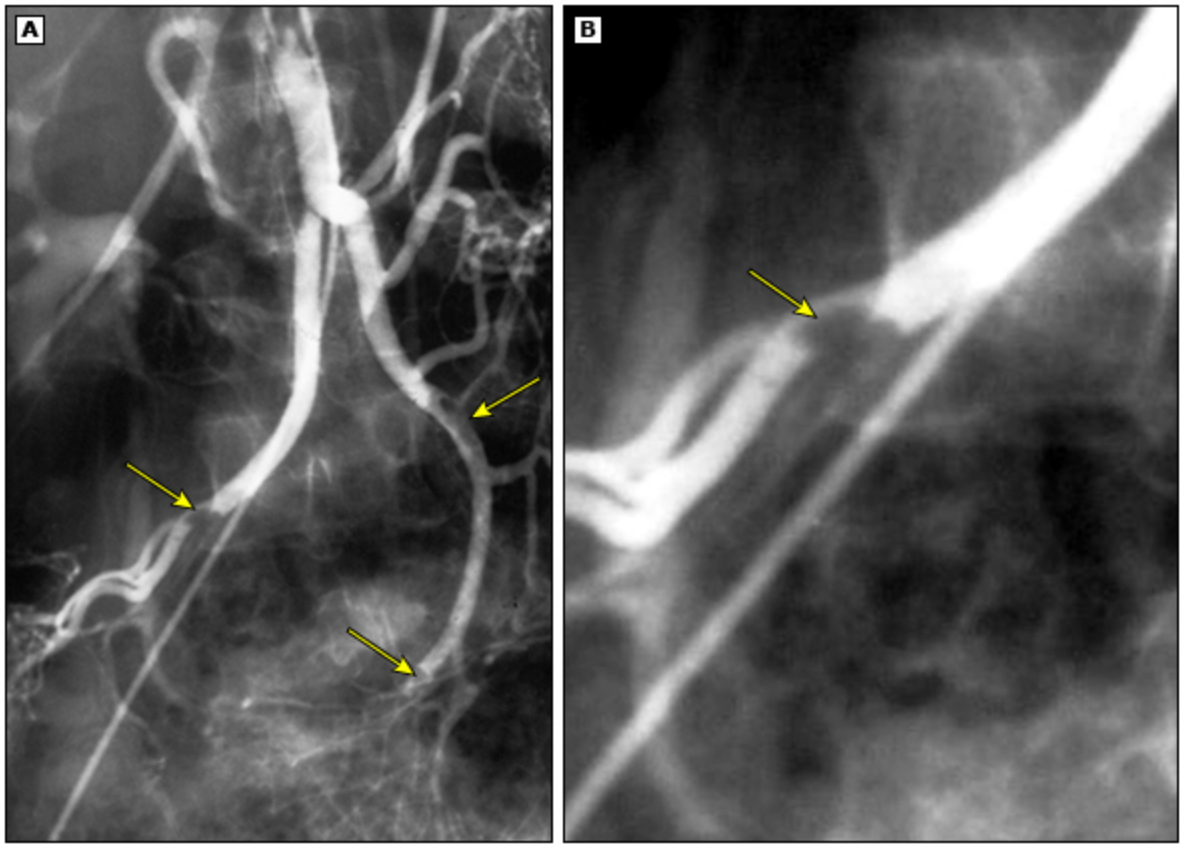


Computed tomography (CT) imaging of the abdomen is from a 55-year-old female who presented with acute abdominal pain and distension. Image A is a scout film of the abdomen and image B is a magnified view of the small bowel from the scout film. The images reveal air within the wall of the dilated loops of small-bowel, characteristic of pneumatosis intestinalis. The paucity of gas in the colon and rectum suggests SBO or severe ileus. Image C is coronal reformat through the abdomen confirming the accumulation of bubbles of air within the wall of the small bowel (white arrow). Image D is an axial image through the affected small bowel loop that shows more extensive accumulation of air within the wall of the thickened small bowel wall. Associated findings include decompressed large bowel (arrowheads), suggesting small bowel obstruction or severe ileus. In the appropriate clinical setting, the findings are highly suggestive of acute ischemia of the small bowel causing a severe ileus and functional obstruction. Less likely but also possible is small bowel obstruction with secondary ischemia.

CT: computed tomography; SBO: small bowel obstruction.

Graphic 58309 Version 3.0

Emboli to SMA on arteriography

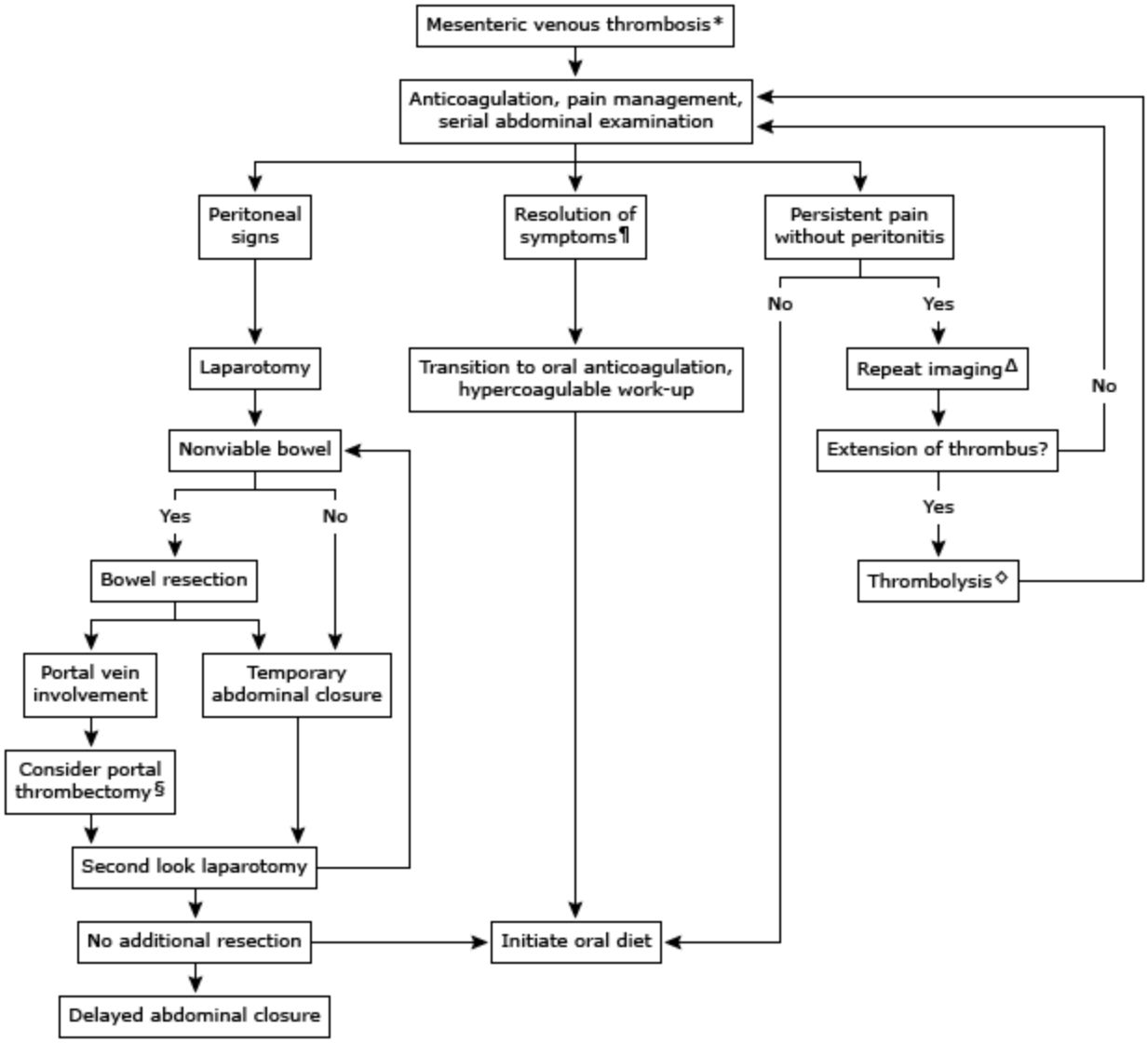


A selective arteriogram of the superior mesenteric artery (Image A) shows multiple emboli (arrows). Image B is a magnified view of the embolus of the ileocolic branch of the SMA (arrow).

SMA: superior mesenteric artery.

Graphic 93263 Version 2.0

Management of mesenteric venous thrombosis



CT: computed tomography.

* Diagnosis is typically made on CT angiography.

¶ In uncomplicated cases, symptoms typically resolve steadily over time.

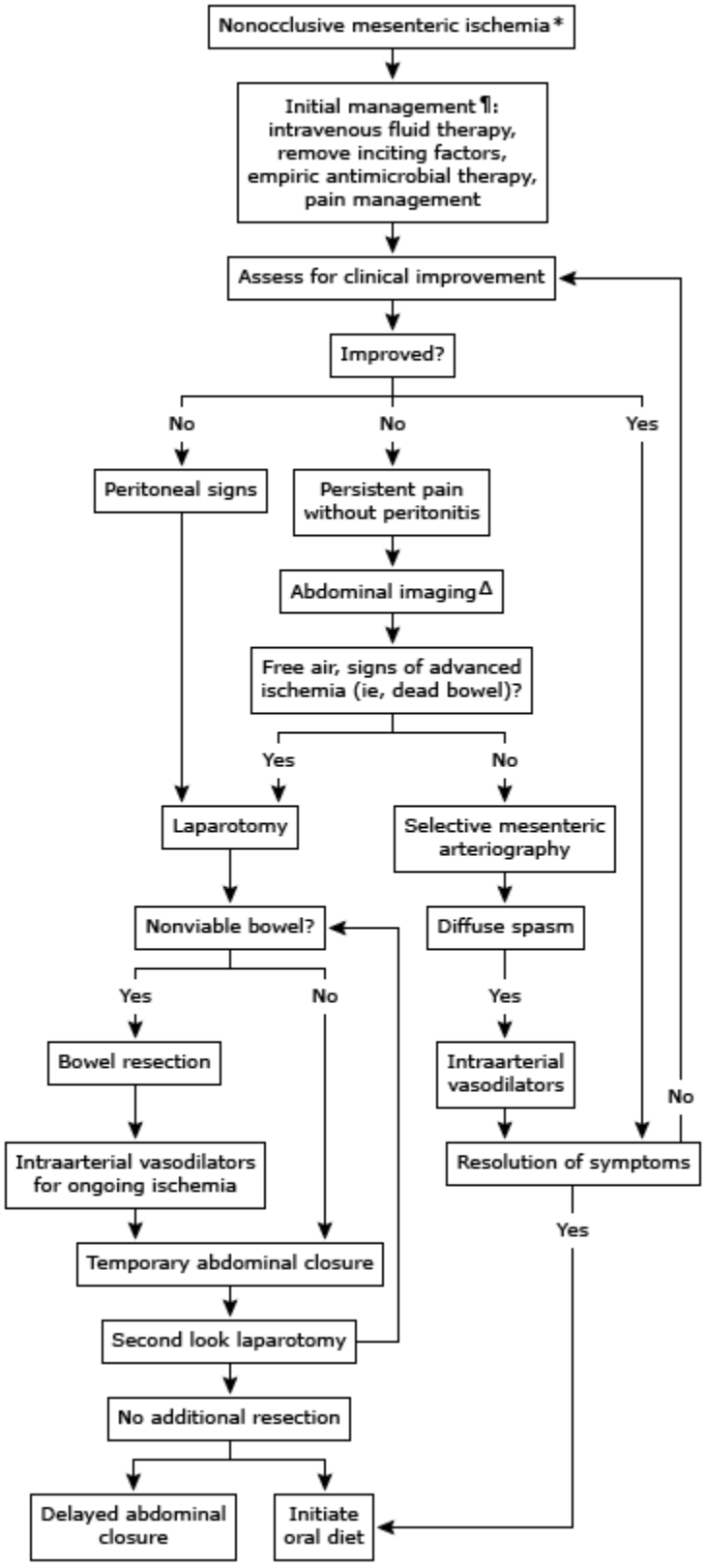
Δ Imaging may include magnetic resonance venography.

◇ If thrombolysis is contraindicated, continue anticoagulation.

§ Portal embolectomy is uncommonly performed.

Graphic 76406 Version 4.0

Management of nonocclusive mesenteric ischemia



CT: computed tomography.

* Diagnosis typically made based on history and risk factors, and exclusion of embolic or thrombotic mesenteric arterial occlusion,

and mesenteric venous thrombosis.

¶ Discontinue anticoagulation (if initiated) once other ischemic etiologies have been excluded.

Δ Depending on the clinical situation, plain abdominal films or CT of the abdomen are appropriate.

Graphic 76899 Version 4.0

Contributor Disclosures

David A Tendler, MD No relevant financial relationship(s) with ineligible companies to disclose. **J Thomas Lamont, MD** Equity Ownership/Stock Options: Allurion [Weight loss]. Consultant/Advisory Boards: Teledoc [Gastrointestinal diseases]. All of the relevant financial relationships listed have been mitigated. **John F Eidt, MD** Grant/Research/Clinical Trial Support: Syntactx [Clinical events and data/safety monitoring for medical device trials]. All of the relevant financial relationships listed have been mitigated. **Joseph L Mills, Sr, MD** No relevant financial relationship(s) with ineligible companies to disclose. **Kathryn A Collins, MD, PhD, FACS** No relevant financial relationship(s) with ineligible companies to disclose.

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