Nonocclusive mesenteric ischemia

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Literature review current through: **Sep 2023.**
This topic last updated: **Dec 06, 2021.**

INTRODUCTION

Acute mesenteric ischemia refers to the sudden onset of intestinal hypoperfusion, which can be due to a nonocclusive reduction of arterial blood flow. Nonocclusive mesenteric ischemia (NOMI) is most commonly due to primary mesenteric arterial vasoconstriction. NOMI was first described in patients with heart failure [1]. The majority of cases involve spasm of branches of the superior mesenteric artery (SMA) supplying the small intestine and proximal colon. Early diagnosis is based upon a high index of clinical suspicion in patients with risk factors but often requires arteriography to firmly establish the diagnosis. NOMI is less common than in the past, and when it occurs, it is managed by reversal of inciting factors, including cessation of vasoconstrictive medicines, correction of the underlying cause of hypoperfusion (if possible), and anticoagulation to limit arterial thrombosis. Selective infusion of the SMA with papaverine or other vasodilator is an option but is uncommonly performed today.

NOMI will be reviewed here. Acute and chronic mesenteric arterial occlusion affecting the small intestine, and colonic ischemia, are discussed separately. (See "Overview of intestinal ischemia in adults" and "Mesenteric venous thrombosis in adults" and "Chronic mesenteric ischemia" and "Colonic ischemia".)

BLOOD SUPPLY TO THE SMALL INTESTINE

The circulation to the small intestines is derived primarily from 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). An extensive collateral circulation (figure 5) protects the intestines from transient periods of inadequate perfusion [2,3].

NONOCCLUSIVE ISCHEMIC INJURY

Ischemic injury to the intestine develops when delivery of oxygen and nutrients is insufficient for cellular metabolism. The likelihood of developing intestinal ischemia depends upon the adequacy of systemic perfusion and collateral circulation, the number and caliber of mesenteric vessels that are affected, and the duration of the ischemic insult. The intestine is able to compensate for approximately a 75 percent acute reduction in mesenteric blood flow for up to 12 hours without substantial injury, in part because of increased oxygen extraction [4].

The pathogenesis of NOMI is related to a homeostatic mechanism that maintains cardiac and cerebral blood flow at the expense of the mesenteric and peripheral circulation [5-8]. Vasopressin and angiotensin are likely the neurohormonal mediators of this phenomenon. Spasm may also be triggered by other vasoactive and cardiotonic drugs [6,9]. The normal physiology of the intestine and response to ischemia are discussed in more detail elsewhere. (See "Overview of intestinal ischemia in adults", section on 'Physiology and mechanisms of ischemia'.)

EPIDEMIOLOGY AND RISK FACTORS

NOMI accounts for 5 to 15 percent of patients with acute mesenteric ischemia [10-12]. In mixed patient populations with intestinal gangrene, the proportion of patients with NOMI is reported to be between 4 and 60 percent [13-15]. In a study in which 35,784 deaths occurred during the years 1970 to 1982, the estimated overall incidence of NOMI with intestinal infarction (verified at autopsy or operation) was 2.0/100,000 person-years [13]. The disease was defined as an intestinal gangrene despite open arteries and with no signs of embolism, dissection, or strangulation. Among the etiologies of acute mesenteric ischemia, NOMI is a diagnosis of exclusion. (See 'Diagnosis' below.)

The incidence of NOMI has declined approximately 50 percent since the 1970s, which has been attributed to the widespread use of invasive hemodynamic monitoring in intensive care units, coupled with prompt correction of hypotension, and the use of systemic vasodilators in cardiac

failure [10]. Despite the decline in its incidence, when it occurs, NOMI results in high mortality because of the difficulty in making the diagnosis, and reversing established NOMI [16]. (See 'Treatment' below and 'Morbidity and mortality' below.)

Risk factors — A careful review of the patient's personal medical history is important. The patient with NOMI is critically ill, typically with severe cardiovascular disease; has a life-threatening complication (sepsis, myocardial infarction, congestive heart failure, severe coronavirus disease 2019 [microvascular thrombosis may contribute]); and often is receiving multiple drugs known to reduce intestinal perfusion for inotropic support.

Risk factors for NOMI include [13,17-35]:

- Heart failure/cardiogenic shock
- Peripheral artery disease
- Aortic insufficiency
- Septic shock
- Cardiac arrhythmias
- Administration of vasoconstrictive medications (eg, digoxin, alpha-adrenergic agonists)
- Cocaine abuse/methamphetamine abuse
- Recent cardiopulmonary bypass
- Chronic renal insufficiency/dialysis
- Severe burns (associated hypovolemia/shock)
- Severe acute pancreatitis

CLINICAL FEATURES

History and physical — The severity and location of the abdominal pain that accompanies NOMI is usually more variable than the classic presentation of acute mesenteric ischemia related to mesenteric arterial obstruction (ie, rapid onset abdominal pain out of proportion to findings on physical examination).

NOMI usually starts with nonspecific symptoms, consisting of mild abdominal pain that gradually progresses and may be accompanied by a bloating sensation, nausea, and vomiting. Abdominal pain is absent in up to 25 percent of patients with NOMI. The clinical presentation of NOMI may be overshadowed by precipitating disorders, including hypotension, congestive heart failure, hypovolemia, and cardiac arrhythmias. Mental status changes are reported to occur in approximately one-third of older adult patients with acute mesenteric ischemia [36]. In addition, many of these patients are intubated and sedated, which may mask the usual clinical

symptoms. Because of these factors, in many cases, complications (necrosis, perforation) develop before a definitive diagnosis can be established. Thus, a high index of suspicion in older adult patients with risk factors for NOMI is imperative for making a prompt diagnosis.

Abdominal examination may be normal initially or reveal only mild abdominal distension or occult blood in the stool. Signs of peritoneal inflammation, such as rebound tenderness and guarding, are absent with ischemia alone. However, if ischemia progresses and transmural bowel infarction develops, peritoneal signs will develop and the abdomen will become distended as ileus develops.

Laboratory studies — Laboratory studies are nonspecific. Although abnormal laboratory values, such as an elevated white cell count, elevated serum lactate, elevated hematocrit (consistent with hemoconcentration), and metabolic acidosis, may be helpful in bolstering suspicion for acute mesenteric ischemia, these do not help determine the etiology, and normal laboratory values do not exclude the diagnosis. However, any patient with acute abdominal pain, minimal findings on abdominal examination, and metabolic acidosis should be regarded as having intestinal ischemia until proven otherwise.

The role of routine laboratory studies and experimental studies in the general diagnosis of mesenteric ischemia is reviewed in detail elsewhere. (See "Overview of intestinal ischemia in adults", section on 'Laboratory studies'.)

Plain abdominal films — Plain abdominal radiographs and ultrasound have a limited role in diagnosing mesenteric ischemia and may be completely normal in more than 25 percent of patients [2]. Plain abdominal radiographs may be helpful in excluding other pathology. Findings suggestive of NOMI include the presence of an ileus, bowel wall thickening, presence of portovenous gas, and/or pneumatosis intestinalis, which is a sign of advanced ischemia (image 1).

Although plain films do not exclude mesenteric ischemia, they may identify complications related to mesenteric ischemia (eg, necrosis, perforation) that indicate the need for immediate abdominal exploration and may identify another obvious cause of abdominal pain (eg, volvulus, small bowel obstruction).

Cross-sectional abdominal imaging — Cross-sectional abdominal imaging studies (eg, computed tomography [CT] of the abdomen, magnetic resonance [MR] imaging) are typically performed first in patients with abdominal pain, but the findings are generally nonspecific. Abdominal CT is generally preferred over MR in the setting of acute abdominal pain because of its lower costs and wide availability [37,38]. Abdominal CT 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. As with plain films, abdominal CT can rule out other causes of acute abdominal pain and may demonstrate gastrointestinal signs consistent with acute mesenteric ischemia such as focal or segmental bowel wall thickening, bowel dilation, mesenteric stranding, or intestinal pneumatosis with portal vein gas (image 2) [39,40].

DIAGNOSIS

The diagnosis of NOMI depends upon a high degree of clinical suspicion (algorithm 1), especially in patients with known risk factors, since clinical signs and symptoms are nonspecific and can be obscured in these typically critically ill patients. Rapid diagnosis is essential to minimize complications (eg, bowel necrosis, perforation) associated with intestinal ischemia [10,41-44].

A definitive imaging diagnosis of NOMI relies upon the demonstration of narrowing or spasm of mesenteric arcades, which can be seen as reduced intramural vessel filling (image 3 and image 4), a reduced number of mesenteric vessels (arteries and veins) (image 5), and irregularity of the arterial branches of the mesenteric vasculature on vascular imaging (alternating dilation and narrowing "chain of lakes" or "string of sausages" sign). These changes are most reliably demonstrated on selective mesenteric arteriography [45-48]. However, when arteriography is performed, spasm may be relieved, and the diagnosis may be missed [16,49]. The greatest advantage of selective mesenteric arteriography, in addition to identifying the specific site of vascular compromise, is the ability to treat mesenteric vasoconstriction with infusion of papaverine. (See 'Vasodilator infusion' below.)

Whether to proceed directly to digital subtraction arteriography, which is invasive, or to first obtain computed tomography (CT) angiography (or magnetic resonance [MR] angiography) requires clinical judgement [50]. CT or MR angiography is most useful for screening patients and identifying features consistent with an alternative vascular etiology for mesenteric ischemia (eg, arterial occlusion, venous thrombosis) [30,37,38,50-56]. In most emergency settings, CT angiography is recommended as soon as possible in patients suspected of having acute mesenteric insufficiency [57]. Like digital subtraction arteriography, a diagnosis of NOMI can still be missed on CT or MR angiography because of the dynamic and spastic pattern and functional nature of the disease [6,9]. More data comparing these modalities to digital subtraction arteriography are needed, particularly to understand whether they can accurately detect the vascular changes of NOMI prior to the onset of irreversible gastrointestinal ischemic changes [56,58].

DIFFERENTIAL DIAGNOSIS

NOMI needs to be differentiated from other causes of mesenteric ischemia due to acute embolic or thrombotic arterial occlusion, or mesenteric venous thrombosis. (See "Overview of intestinal ischemia in adults".)

Confusion may also arise in acute abdominal pain resulting from volvulus, intussusception, and acute small bowel obstruction from adhesions where the mechanical obstruction may also compress mesenteric vessels and lead to mesenteric ischemia and necrosis.

TREATMENT

The goal of treatment of patients with NOMI is to restore intestinal blood flow as rapidly as possible (algorithm 2), which is accomplished by removing inciting factors (vasoconstrictive medications), treating underlying causes (heart failure, sepsis), hemodynamic support and monitoring, and, less commonly, intra-arterial infusion of vasodilators.

Patients with acute peritoneal signs will require abdominal exploration and bowel resection.

Hemodynamic support and monitoring — Patients suspected of having NOMI should be resuscitated, including measures aimed at improving cardiac function, correction of hypovolemia and treatment of cardiac arrhythmias, correction of metabolic acidosis, initiation of broad-spectrum antibiotics, and placement of a nasogastric tube for gastric decompression. Although not subjected to rigorous study, antibiotics are generally recommended due to the high risk for bacterial translocation and sepsis as ischemia and/or infarction progresses [59].

Vasoconstricting agents and digitalis should be avoided if possible since they can exacerbate mesenteric ischemia. If an inotropic agent is required, either dobutamine, low-dose dopamine, or milrinone is preferred since the effect on mesenteric perfusion is less compared with other vasopressor agents. Determination of cardiac output, systemic vascular resistance, and mixed venous oxygen saturation is important to selecting appropriate treatment. (See "Use of vasopressors and inotropes".)

Anticoagulation — The efficacy of systemic anticoagulation as a means for improving mucosal perfusion in the face of arterial spasm has not been studied. Some clinicians anticoagulate these patients until vasoconstriction resolves and the perfusion deficit can be corrected as a means to limit the development of thrombus. However, this practice is not supported by evidence from any clinical trials.

Vasodilator infusion — Other than supportive care, the only intervention available for patients with NOMI involves infusion of vasodilators (eg, papaverine, prostaglandins, nitroglycerin) to reverse mesenteric vasoconstriction. However, there are no robust data that prove the benefit of vasodilator therapy in these patients [60]. Transcatheter infusion of vasodilators may be most useful in the setting of illicit drug overdose or accidental therapeutic drug overdoses [30,61]. The available literature consists of case reports and small series, with heterogeneous data that make interpreting outcomes difficult. If the underlying condition associated with NOMI cannot be reversed, it is not clear that vasodilator therapy in and of itself is sufficient to prevent physiologic vasoconstriction. A small retrospective series of 21 consecutive patients with NOMI suggested that earlier initiation of vasodilator therapy was associated with a higher likelihood of survival [62].

Vasodilators are administered through an arteriographic catheter, typically placed into the superior mesenteric artery. Papaverine is the predominantly used agent, but its availability is limited [63]. Papaverine should not be mixed with heparin-containing fluids due to risk of precipitation.

Heparin is, however, generally administered through the arterial sheath to prevent thrombosis of the cannulated vessel (eg, femoral artery, brachial artery) [2,10,16,64].

In patients without peritoneal signs, repeat arteriography can be performed in 24 hours to verify resolution of vasoconstriction.

Abdominal exploration — Surgery should not be delayed in patients suspected of having intestinal infarction or perforation based upon clinical or radiographic features. In patients who require segmental bowel resection, a delay in completing the intestinal anastomosis and aggressive re-exploration may improve survival (mortality of 22 versus 42 percent in one series) [65,66].

Following exploration, the abdomen can be closed provided there is no undue tension on the abdominal wall. Alternatively, the abdomen can be left open for interval closure. When a second-look operation is anticipated, leaving the abdomen open facilitates re-exploration and prevents complications that can develop related to elevated intra-abdominal pressure. Assessment of adequate intestinal perfusion and viable bowel may be aided by intraoperative use of indocyanine green (ICG) fluorescence [67].

Methods for dressing the open abdomen and timing of closure are discussed in detail elsewhere. (See "Management of the open abdomen in adults".)

MORBIDITY AND MORTALITY

Nonocclusive intestinal ischemia has the poorest survival rate among the various etiologies of mesenteric ischemia, primarily because of the severity of comorbid conditions that precipitate reduced mesenteric perfusion, and delays in diagnosis. NOMI has a mortality rate of 70 to 90 percent that has changed little over time [30].

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".)

SUMMARY AND RECOMMENDATIONS

- Nonocclusive mesenteric ischemia (NOMI) accounts for up to 20 percent of cases of acute mesenteric ischemia. (See 'Introduction' above and 'Morbidity and mortality' above.)
- Risk factors for NOMI include known cardiovascular disease, myocardial infarction, aortic insufficiency, sepsis, cardiac arrhythmias, administration of digoxin or alpha-adrenergic agonists, cocaine, cardiopulmonary bypass, and dialysis. (See 'Risk factors' above.)
- The typical patient with NOMI is an older adult man or woman with cardiovascular disease who has a life-threatening complication (such as a myocardial infarction or congestive heart failure) and is being treated with drugs known to reduce intestinal perfusion (such as diuretics). Abdominal pain that accompanies NOMI is variable compared with the classic severe pain of acute mesenteric ischemia due to arterial embolism or thrombosis. Abdominal pain is absent in up to 25 percent of patients, and the clinical picture may be overshadowed by precipitating disorders. (See 'Clinical features' above.)
- Rapid diagnosis is essential to prevent complications (eg, bowel necrosis, perforation) associated with acute mesenteric ischemia. The diagnosis of acute mesenteric ischemia depends upon a high degree of clinical suspicion in the patient with risk factors. However, early signs and symptoms of mesenteric ischemia are nonspecific. Abdominal computed tomographic (CT) angiography, without oral contrast, is useful as a screening examination for patients with acute abdominal pain and a suspicion for acute mesenteric ischemia. CT angiography has high degree of accuracy for diagnosing acute mesenteric ischemia and is

useful in excluding other causes of acute abdominal pain, and other etiologies of acute mesenteric ischemia. (See 'Diagnosis' above.)

- The goal of treatment of patients with NOMI is to restore intestinal blood flow as rapidly as possible. Initial management of NOMI includes aggressive hemodynamic monitoring and support, correction of metabolic acidosis, initiation of broad-spectrum antibiotics, and placement of a nasogastric tube for gastric decompression. (See "Overview of intestinal ischemia in adults", section on 'Initial management' and 'Hemodynamic support and monitoring' above.)

- For patients with risk factors and clinical features that suggest NOMI rather than another etiology for acute mesenteric ischemia, we suggest selective arteriography of the mesenteric circulation over other vascular imaging modalities. Digital subtraction arteriography offers the option of vasodilator infusion directly into the vasoconstricted mesenteric circulation. (See 'Vasodilator infusion' above.)

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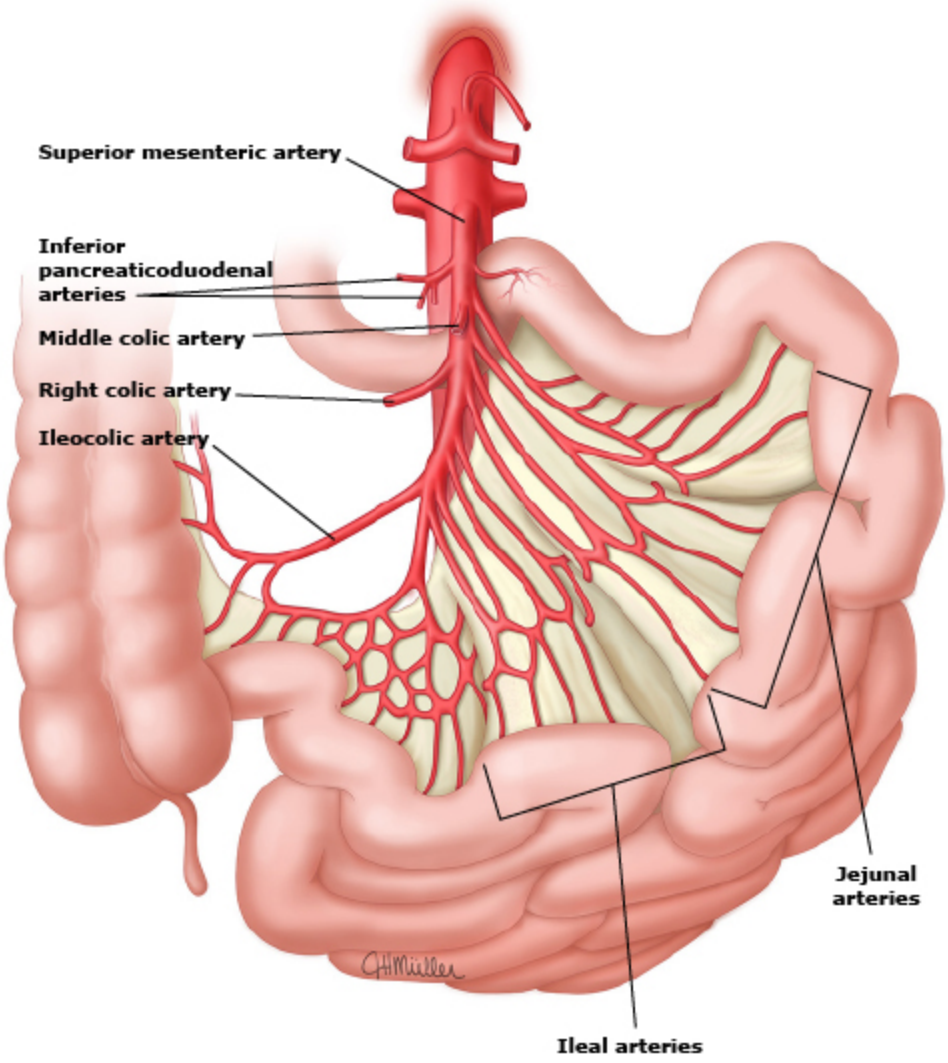
GRAPHICS

Blood supply to the small intestine

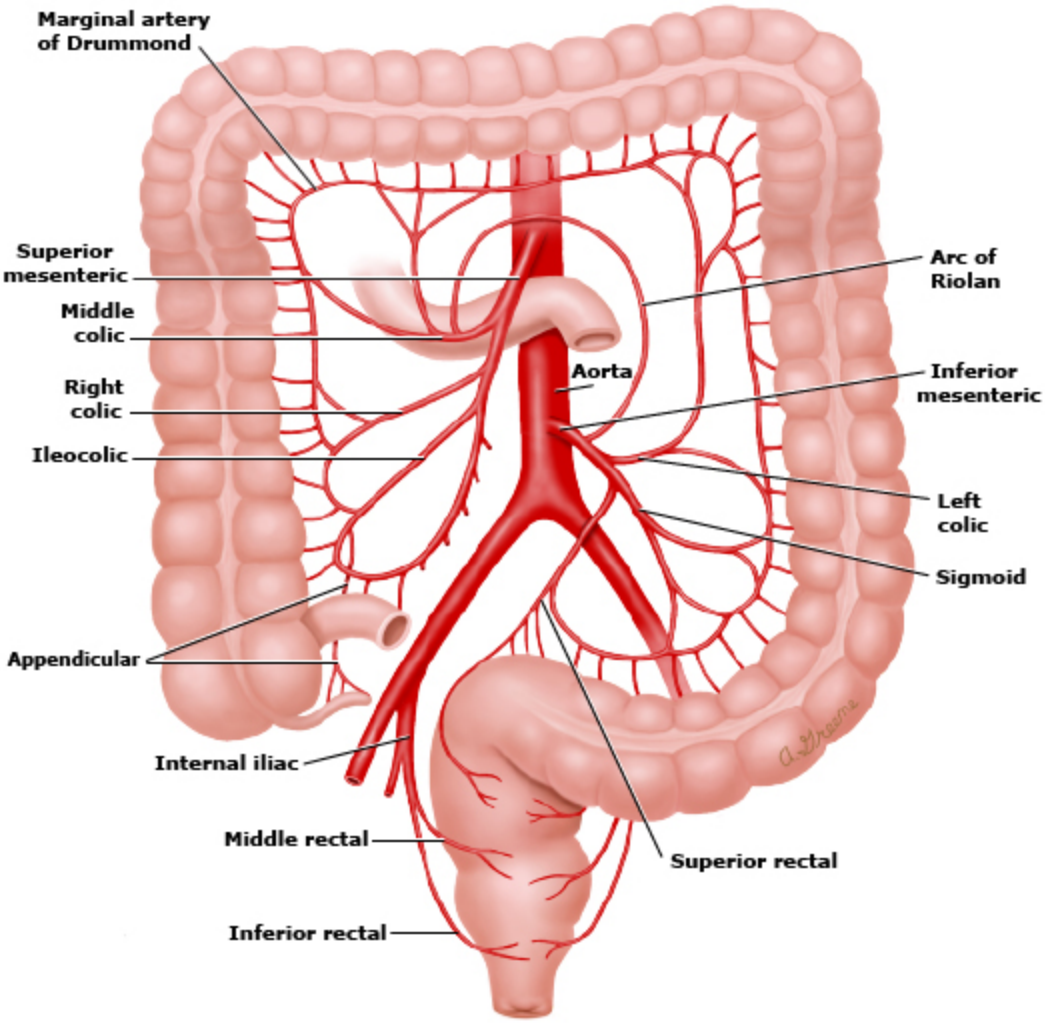


The blood supply to the small and large bowel is derived from the celiac artery and SMA. The celiac axis primarily provides blood flow to the stomach, liver, spleen, and pancreas but is also a source of collateral flow when blood flow in the SMA is reduced. The SMA gives rise to the inferior pancreaticoduodenal 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:

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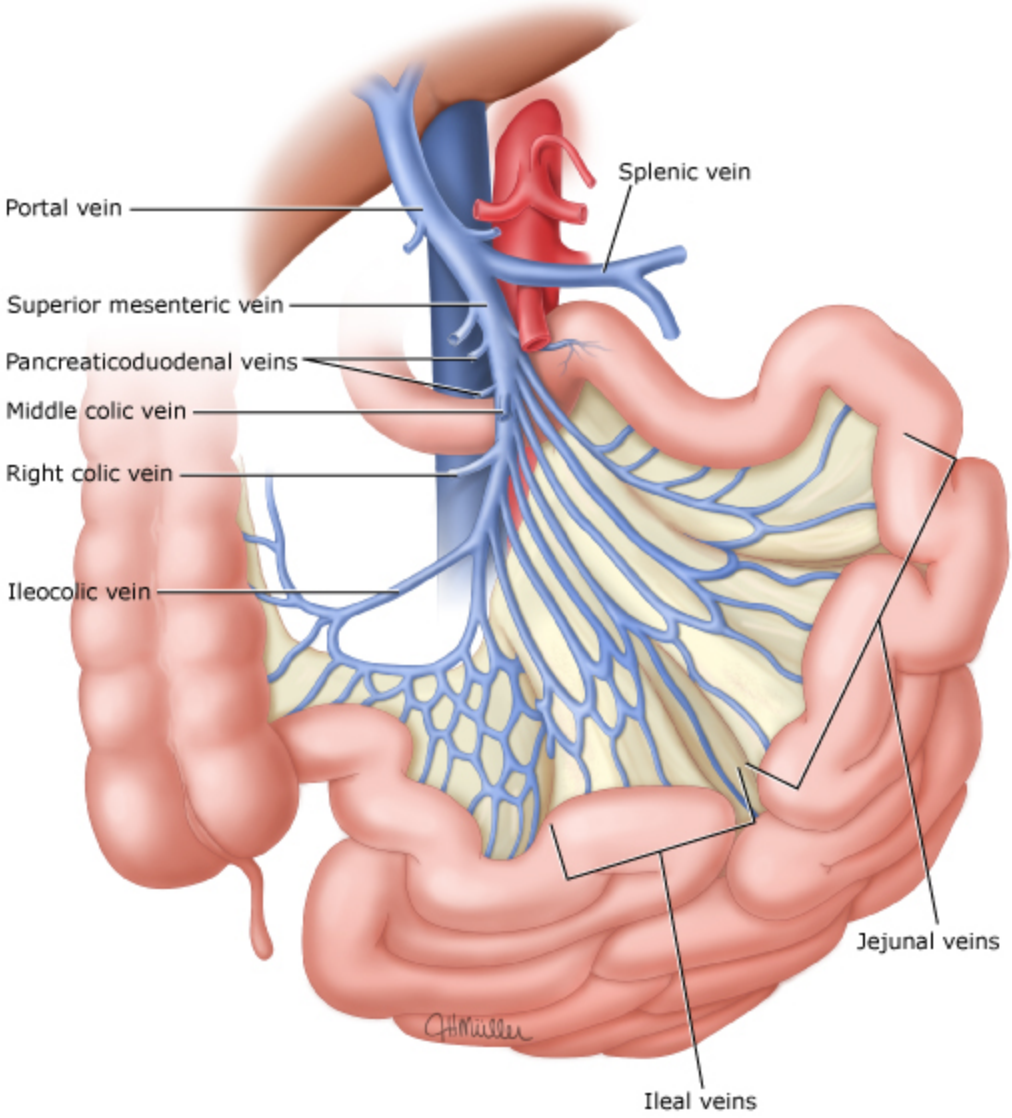
Graphic 73756 Version 12.0

Venous drainage of the small intestine

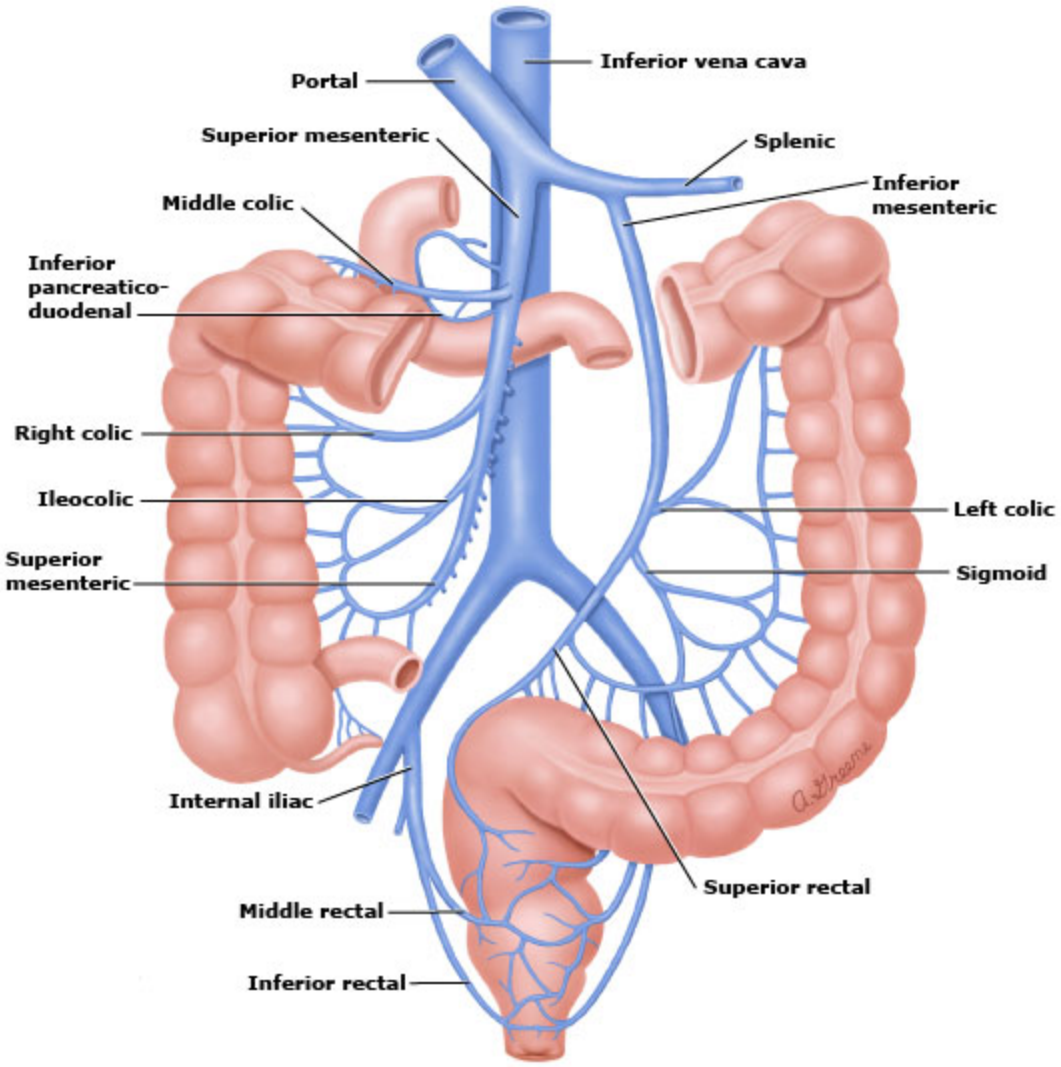


The mesenteric veins parallel their corresponding arteries. The SMV drains the small intestine, cecum, ascending, and transverse colon via the jejunal, ileal, ileocolic, right colic, and middle colic veins. The SMV joins the splenic vein to drain into the portal vein.

SMV: superior mesenteric vein.

Graphic 88949 Version 4.0

Venous drainage of the colon and rectum

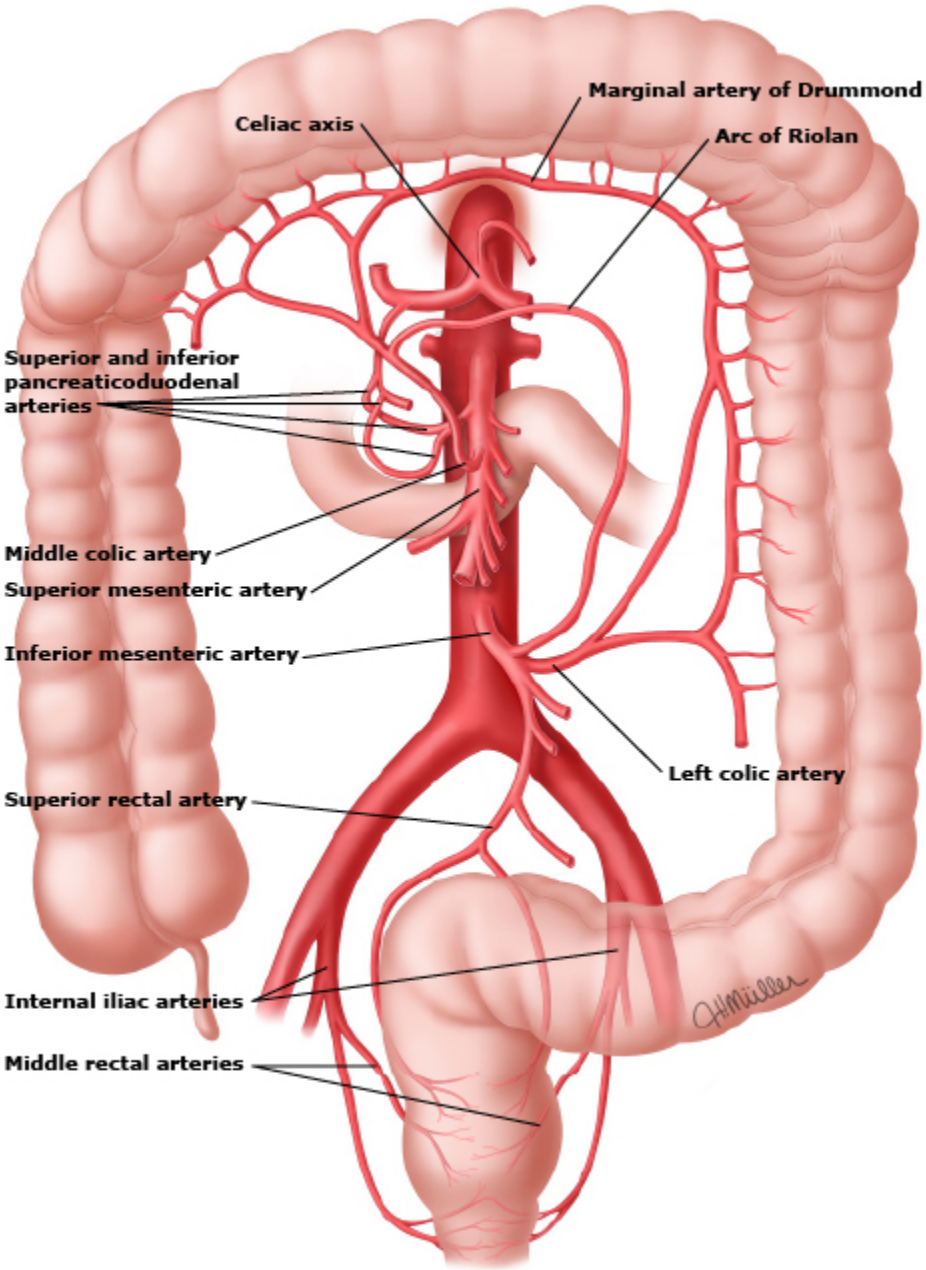


The mesenteric veins parallel their corresponding arteries. The SMV drains the small intestine, cecum, and ascending and transverse colon via the jejunal, ileal, ileocolic, right colic, and middle colic veins. The IMV drains the descending colon through the left colic, the sigmoid through the sigmoid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



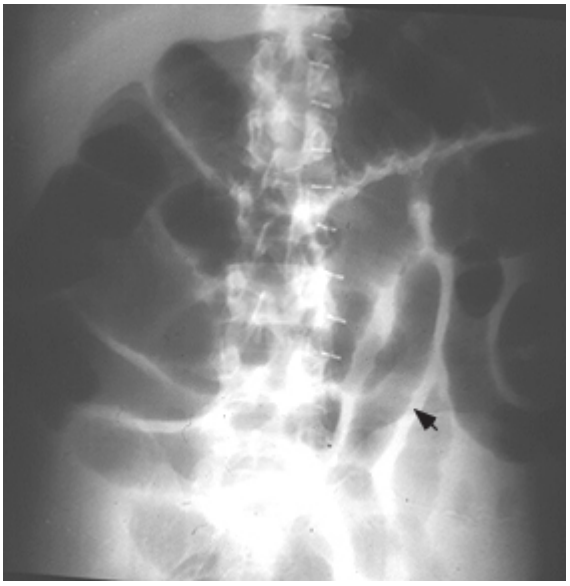
An abundant collateral blood supply exists between the SMA and IMA and the IMA and internal iliac arteries. The arcades of the SMA and IMA interconnect at the base and border of the mesentery. The connection at the base of the mesentery is called the arc of Riolan, whereas the connection along the mesenteric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

Small bowel ischemia on plain abdominal film

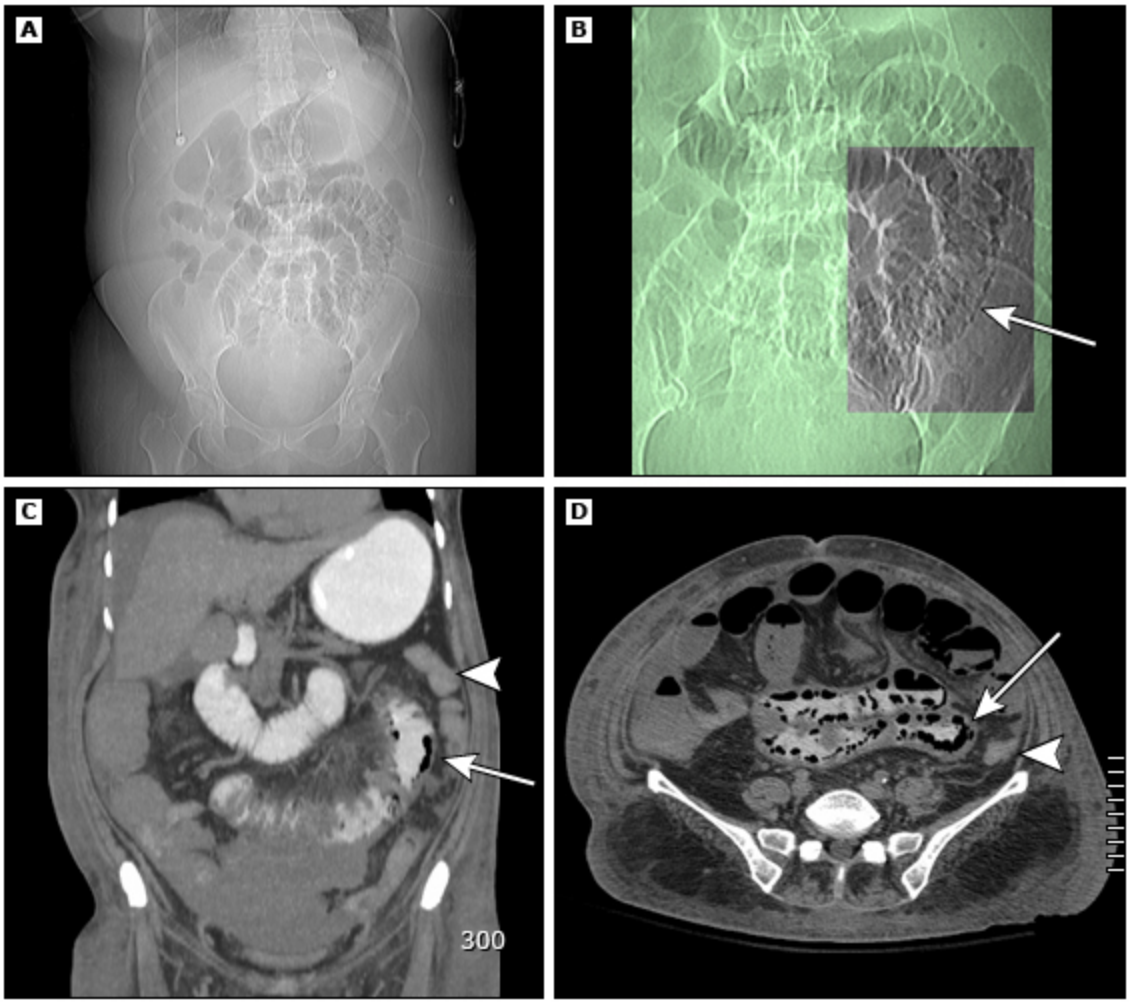


A plain radiograph of the abdomen demonstrates distended featureless loops of small bowel with wall thickening (arrow) and separation of the bowel loops. These findings are consistent with hemorrhage into the bowel wall secondary to ischemia.

Courtesy of Jonathan B Kruskal, MD, PhD.

Graphic 74055 Version 3.0

CT scan of pneumatosis coli

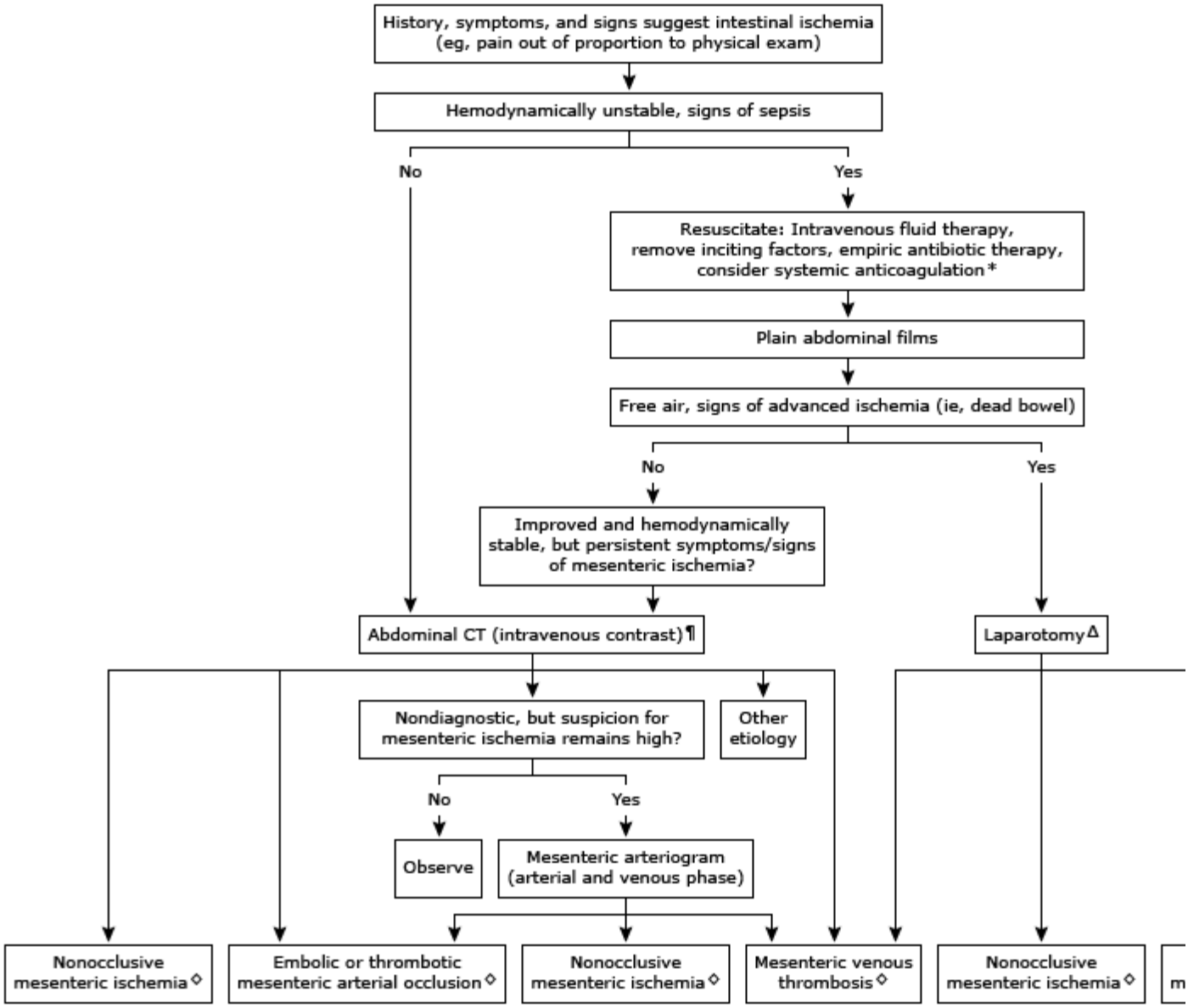


Computed tomography (CT) imaging of the abdomen is from a 55-year-old female who presented with acute abdominal pain and distension. Image A is a scout film of the abdomen and image B is a magnified view of the small bowel from the scout film. The images reveal air within the wall of the dilated loops 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

Diagnosis and initial management of intestinal ischemia



CT: computed tomography.

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

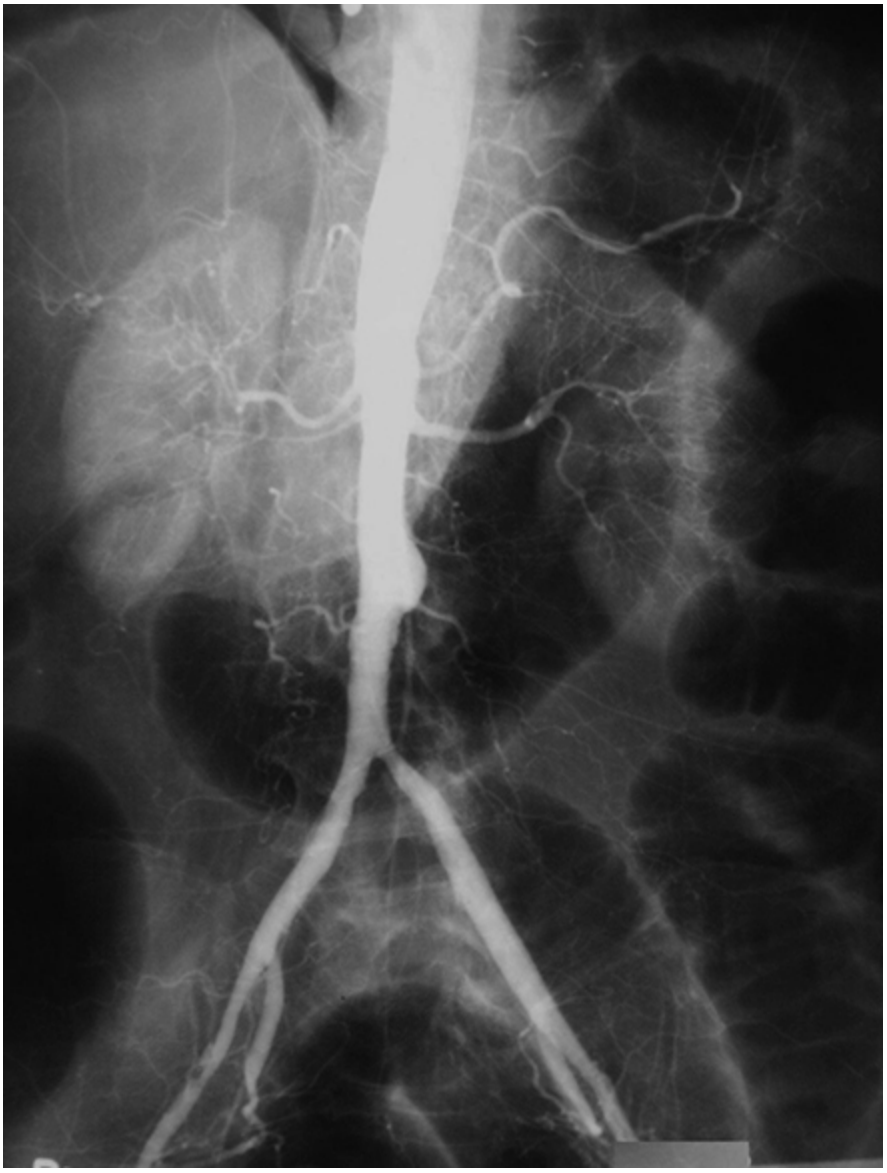
¶ Imaging signs associated with mesenteric ischemia include focal or segmental bowel wall thickening, inte portal vein gas, portomesenteric thrombosis, mesenteric arterial calcification, and mesenteric artery occlusi

Δ Medically fit patients.

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

Graphic 62760 Version 6.0

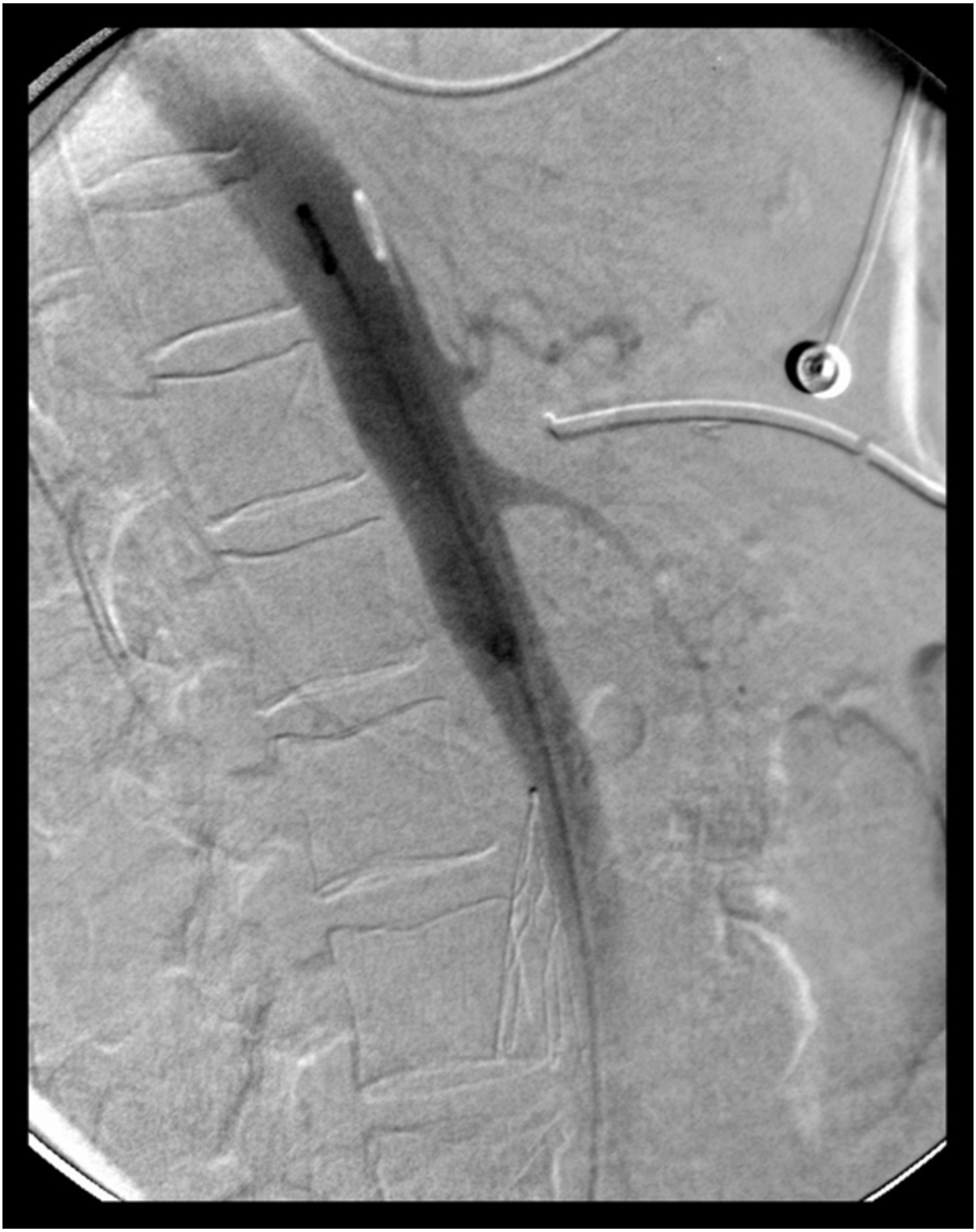
Nonocclusive ischemia on aortography



Aortogram demonstrating diffuse spasm of all the arteries in the abdomen, including the hepatic, splenic, renals, and mesenteric vessels, with reduced vessel filling consistent with nonocclusive mesenteric ischemia. Small bowel ileus is seen in the left lower quadrant.

Graphic 95988 Version 1.0

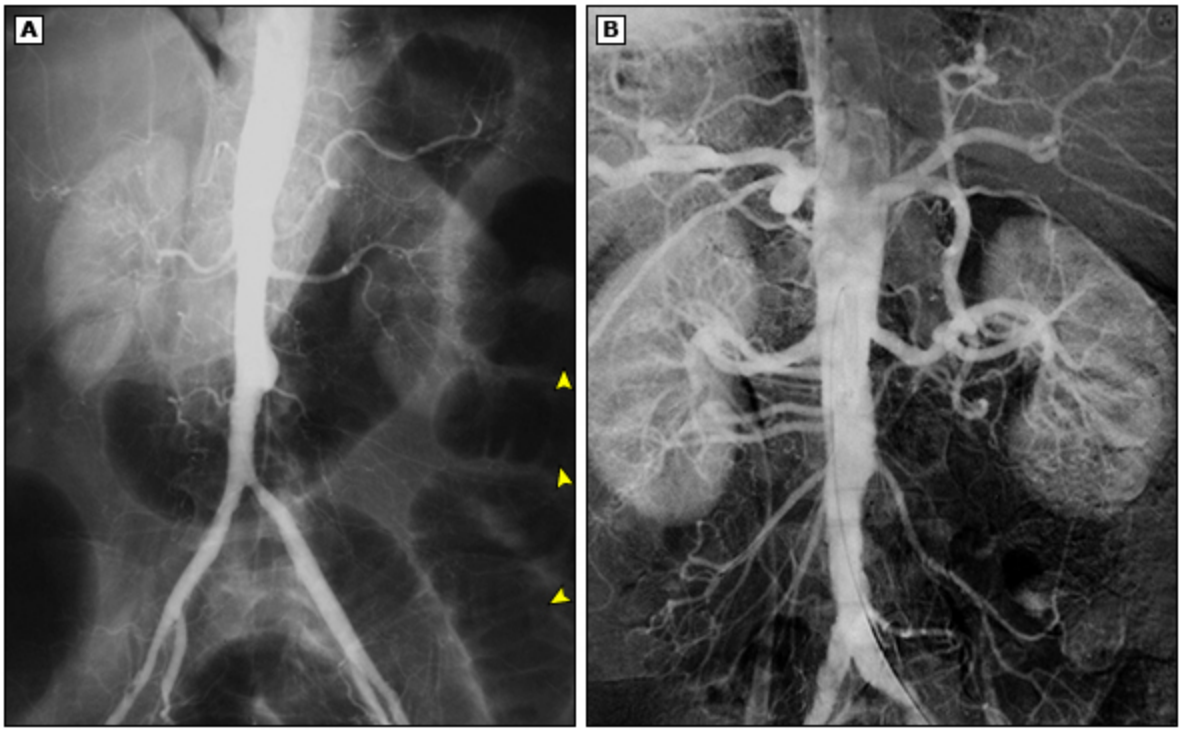
Lateral aortogram nonocclusive ischemia



Lateral aortogram demonstrating narrowing or spasm of the celiac axis and superior mesenteric artery with reduced intramural vessel filling consistent with nonocclusive mesenteric ischemia.

Graphic 95989 Version 1.0

Nonocclusive mesenteric ischemia on angiography

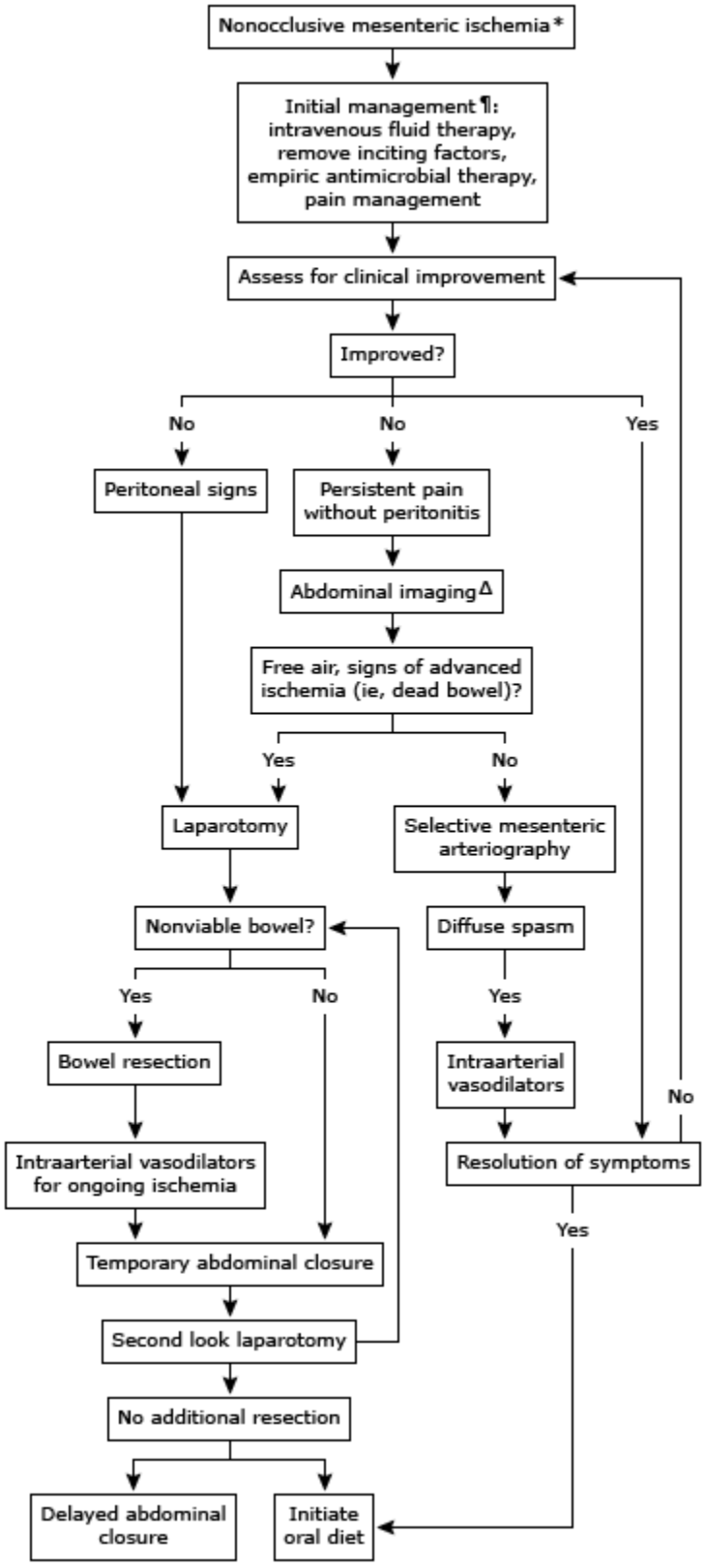


The A-P view aortogram (A) is of a patient with severe hypotension and shows diffuse spasm of the aorta and all the visceral vessels of the abdomen. Ischemic small bowel (arrowheads) was resected. Normal visceral perfusion is demonstrated in image B.

A-P: anteroposterior.

Graphic 93267 Version 2.0

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.

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

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