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Mesenteric Ischemia

Joshua Broder, MD
Assistant Professor,
Division of Emergency Medicine, Department of Surgery,
Duke University Medical Center, Durham, NC

Mesenteric ischemia is a fearful diagnosis due to its subtle and variable presentation, time-dependence, and extreme morbidity and mortality. The condition is sufficiently rare that an individual emergency physician will encounter only a few cases during an entire career. As a result, even the most skilled physician cannot expect to become an expert in the diagnosis and treatment of this condition based on experience alone. In this article, we'll consider some facts and misconceptions about mesenteric ischemia in the context of several real cases. We'll dissect pitfalls that may occur at multiple points in evaluation and treatment of the patient with mesenteric ischemia, including the initial suspicion of the diagnosis, diagnostic testing, and medical and surgical interventions. We'll closely examine the evidence from the medical literature, recognizing that in many cases the strength of evidence is scant.

Let's begin with a very brief review of abdominal vascular anatomy – which is critical to understanding why occlusion of a single vessel can lead to catastrophic loss of bowel or even death. The major mesenteric branches of the abdominal aorta are the celiac artery, superior mesenteric artery, and inferior mesenteric artery (table 1). The major vessel returning blood from the bowel to the liver is the portal vein. When blood flow through these vessels is compromised, significant ischemic complications can arise. The superior mesenteric artery is of particular concern because it perfuses nearly the entire small bowel and two-thirds of the large bowel. Inferior mesenteric artery occlusion can infarct the entire distal colon, leading to perforation and sepsis. Portal vein thrombosis restricts venous drainage of the bowel, in extreme cases leading to ischemia by preventing arterial inflow.

Poor perfusion of vessels can occur from external compression (for example, from an abdominal or retroperitoneal mass lesion), from internal obstruction (by embolus, thrombus, or arterial dissection), from volvulus of the mesentery and blood supply (eg midgut volvulus with occlusion of the superior mesenteric artery, and from cecal or sigmoid volvulus), from compression of obstructed bowel segments by adhesions, and by global hypoperfusion states.

The significance of a vascular occlusion may depend on the rate at which it occurs, and how proximal the point of occlusion is. Acute occlusions are more likely to result in end-organ ischemia, due to a paucity of collateral vessels. In contrast, patients with chronically progressive vascular disease may develop collateral blood supplies which may limit end-organ ischemia. Distal occlusions of vessels compromise shorter segments of bowel compared with proximal occlusions, and collateral perfusion may be sufficient to prevent frank infarction. Thus vascular occlusion and mesenteric ischemia are not synonymous; ischemia is a potential consequence of occlusion.

Table 1. Important abdominal blood vessels and their target organs		
Artery	Supplies	Occlusion may lead to
Celiac artery	esophagus, stomach, pancreas, liver, proximal (first part of) duodenum	ischemia/infarction of target organs
Superior mesenteric artery	pancreas, duodenum distal to first part, entire remaining small bowel (jejunum and ileum), cecum, appendix, ascending colon, transverse colon	ischemia/infarction of target organs – including nearly the entire small bowel and proximal two-thirds of colon
Inferior mesenteric artery	left (descending) colon, sigmoid colon, and rectum	ischemia/infarction of target organs, including descending and sigmoid colon
Vein	Returns	Occlusion may lead to
Portal vein	blood from the spleen and intestine to the liver – supplying approximately 75% of hepatic blood flow	ischemia/infarction of bowel drained by this vessel. Hepatic necrosis is prevented by the redundant blood supply of the liver. In addition, portal hypertension results, with potential complications including ascites and esophageal varices

What conditions are encompassed by the term "mesenteric ischemia"? Mesenteric ischemia is really a family of related disorders, including acute occlusions of mesenteric arteries from embolus, dissection, *in situ* thrombosis, and volvulus; chronic mesenteric ischemia from mesenteric atherosclerosis; ischemic colitis from low-flow hemodynamic states; and portal vein thrombosis, often complicating other abdominal and systemic conditions. Some definitions:

- Acute mesenteric ischemia implies a state of immediate and significant vascular occlusion with accompanying end-organ (bowel) ischemia or infarction. Acute mesenteric ischemia is similar to unstable cardiac angina or infarction in its acuity of

onset – and is classically described as presenting with "sudden pain out of proportion to exam." Causes include embolic occlusion from atrial fibrillation or *in situ* thrombosis of a vessel. Aortic dissections can also occlude branch vessels including mesenteric arteries, leading to bowel ischemia in a minority of cases. Although often considered a distinct pathological process, midgut volvulus in the setting of malrotation is another cause of acute mesenteric ischemia in both infants and adults. Cecal and sigmoid volvulus can similarly result in acute vascular occlusion. Closed-loop small or large bowel obstructions from hernias or adhesions can also lead to acute bowel ischemia, either by compressing the vascular supply directly or by compressing the bowel wall, limiting perfusion of the obstructed bowel segment. In patients with low cardiac output or systemic hypotension from any cause, bowel can become acutely ischemic.

- Chronic mesenteric ischemia is a state of subacute bowel ischemia, due to stenosis or compression of mesenteric vessels. Akin to stable cardiac angina, chronic mesenteric ischemia usually waxes in the face of increased demand for perfusion following a meal and wanes with bowel rest – giving rise to the term "intestinal angina." Chronic ischemia can progress to acute ischemia.
- Ischemic colitis is often differentiated from acute small bowel ischemia in the vascular territory of the superior mesenteric artery (SMA). This disease process is somewhat different in its prognosis and treatment from ischemia due to proximal SMA occlusion, as discussed in a later case. As shown in table 1, poor perfusion in the territory of the inferior mesenteric artery leads to ischemia of the descending and sigmoid colon. Hypoperfusion may occur from acute arterial occlusion, but this vascular territory is also particularly susceptible to ischemia during low-flow states, induced by hypotension from any cause including sepsis, volume depletion, anemia or hemorrhage, or cardiogenic shock. Poor perfusion of the ileocolic, right colic, and middle colic branches of the superior mesenteric artery can also induce ischemia of the ascending and transverse colon.
- Portal vein thrombosis can prevent outflow of blood from mesenteric vessels. Secondly, the engorged vascular beds cannot accommodate arterial inflow, resulting in ischemia or infarction of the involved bowel. In some cases, gas-forming organisms in infarcted bowel can release gas within the portal vein, seen on imaging studies such as CT. In addition, portal vein thrombosis raises portal venous pressures, leading chronically to ascites and esophageal varices.

In the cases that follow, we will highlight some of these differences, including differences in clinical presentation, patient risk factors, diagnosis, and treatment. We'll use the term "mesenteric ischemia" for discussions that are not specific to a particular vessel or presentation. For more specific disease processes, we'll use the relevant terminology.

How common is mesenteric ischemia?

Studies of the incidence of mesenteric ischemia are limited by several factors. As discussed above, the term encompasses a range of pathology, so the incidence depends on the definition used in each study. The diagnosis is difficult, so many cases may go unrecognized. The process is likely most common in elderly patients, who may not routinely undergo autopsy and whose cause of death may be attributed to other conditions such as sepsis or myocardial infarction. Acosta *et al* (2003) found that acute superior mesenteric artery (SMA) thrombosis occurs with an incidence of 5.3 cases per 100,000 population per year; in a second population-based study using autopsy data (2004), the same authors found an incidence of 8.6 per 100,000 person-years.^{1, 2} Interestingly, intestinal ischemia was suspected pre-mortem in only 33% of cases, emphasizing the point that much of what we "know" about mesenteric ischemia is biased by the presentations of cases recognized and diagnosed by physicians, which may represent the tip of the iceberg.² To put these numbers in perspective, according to the CDC, from 2003-2005, the age-adjusted rate of hospitalization for acute myocardial infarction (a surrogate for the actual incidence) was 242 per 100,000 per year.³ The rarity of acute SMA thrombosis manifests itself in the medical literature as small retrospective case series, often collected over a decade or more. Our discussions will necessarily be limited to studies such as this, as large, prospective, methodologically strong studies are very rare with mesenteric ischemia (see end-note on "Weakness of Medical Literature on Mesenteric Ischemia").

If acute mesenteric ischemia is truly rare, why turn our attention to this condition? The answer is that like other true emergencies such as acute myocardial infarction and abdominal aortic aneurysm rupture, the disease is extremely time-dependent and highly mortal. The time-dependence means that the emergency physician may be the only provider seeing the patient within a time-frame adequate to change the outcome. Inpatient providers may discover the diagnosis ultimately, but in these cases fatal outcomes may be inevitable due to progression of bowel infarction. Consider some sobering figures, comparing myocardial infarction to mesenteric ischemia:

- acute myocardial infarction mortality without thrombolysis or aspirin: 12% (ISIS II, placebo arm)⁴
- acute myocardial infarction mortality with modern percutaneous angioplasty and stenting: 3-6%⁵
- acute superior mesenteric artery occlusion mortality, with treatment: 50-75%.^{1, 6-10}

In multiple studies that included patients from 1980 to 2003, the mortality from acute mesenteric ischemia from SMA thrombosis ranged between 55 and 75%, with high mortality even in more recent cohorts.^{1, 6-10} These studies likely underestimate the actual mortality, as they exclude patients who died from unsuspected mesenteric ischemia which was not diagnosed prior to death or by autopsy. A large population-based study using autopsy data demonstrated an astounding 93% mortality rate in acute mesenteric ischemia from SMA thrombosis, with two-thirds of these patients having no apparent suspicion of ischemia before death.²

[Weakness of the medical literature for mesenteric ischemia - see Appendix for a detailed discussion.](#)

Case 1

A 77 year-old male presents with acute onset of abdominal pain one hour prior to emergency department arrival. The patient also notes bright blood from his colostomy. He has a history of atrial fibrillation and is in that rhythm on emergency department arrival. Due to recent falls, the patient has discontinued his use of coumadin. His vital signs show a blood pressure of 213/115, heart rate 76, respiratory rate 20, temperature 35.8C, and oxygen saturation 91% on room air. His abdominal exam shows no apparent tenderness despite his complaint of intense pain. Initial labs are notable for an elevated lactate (4.1mmol/L) and leukocytosis (12.6k). An abdominal x-ray series one hour after ED arrival shows a paucity of bowel gas but no free air, pneumatosis, or portal venous gas (**Figure 1.1**).

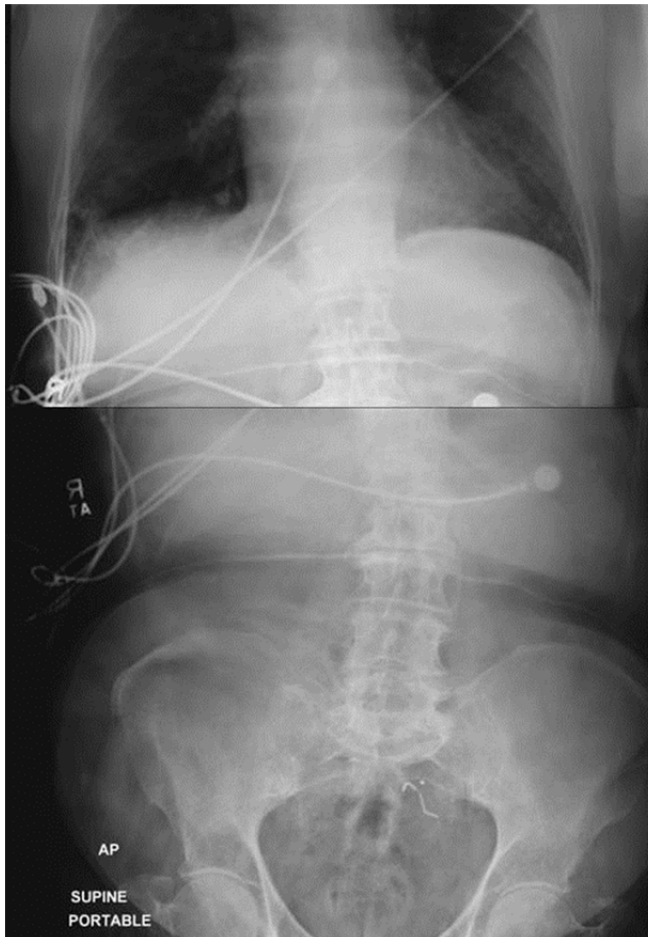


Figure 1.1 Initial x-rays in patient with superior mesenteric artery thrombosis.

No free air, pneumatosis, or air-fluid levels are seen, although this supine x-ray is limited for detection of free air or bowel obstruction. The x-rays show a paucity of bowel gas, a nonspecific finding. Abdominal x-rays are insensitive and nonspecific for most abdominal conditions and may cause diagnostic delay without providing useful information.

A CT scan with IV and oral contrast is ordered. Three hours later, a CT scan is completed and interpreted as demonstrating complete obstruction of the superior mesenteric artery near its origin (Figure 1.2 and 1.3).

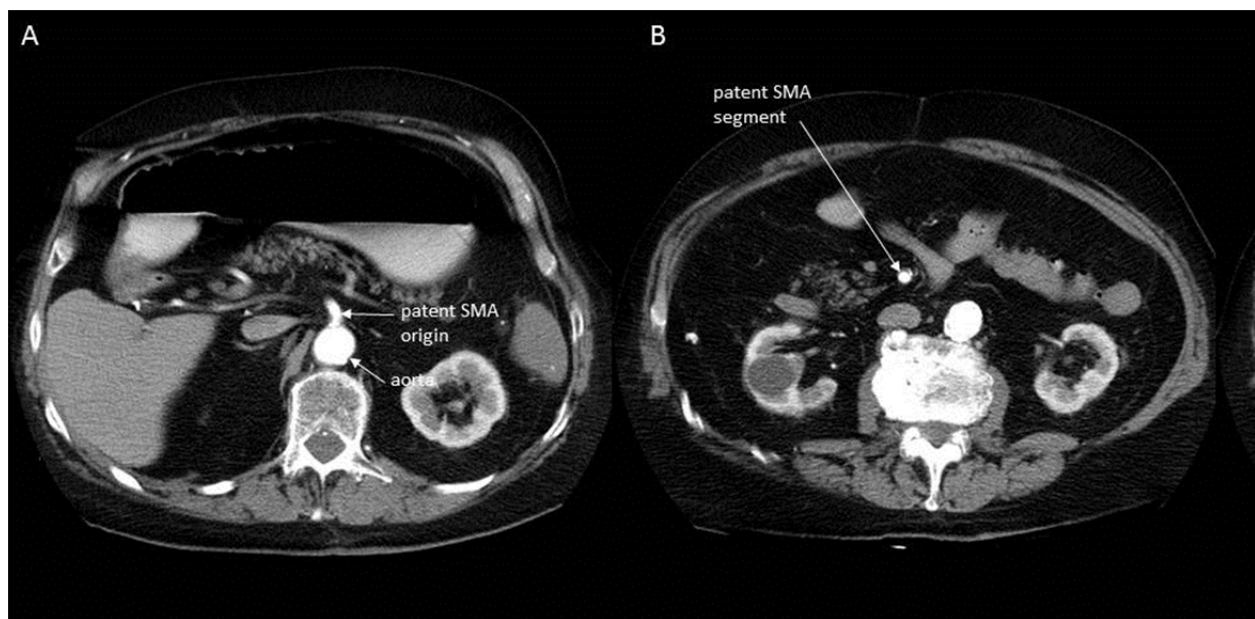


Figure 1.2 CT with IV contrast in patient with superior mesenteric artery thrombosis. Oral contrast was administered but was not necessary. IV contrast was essential to the diagnosis, revealing a filling defect in the SMA.

A, the proximal SMA is visible and is filled with contrast.

B, the SMA is seen in cross-section as a circle, still patent and filled with contrast.

C, 3 slices caudad, the SMA is thrombosed (dark) and is no longer filled with contrast.

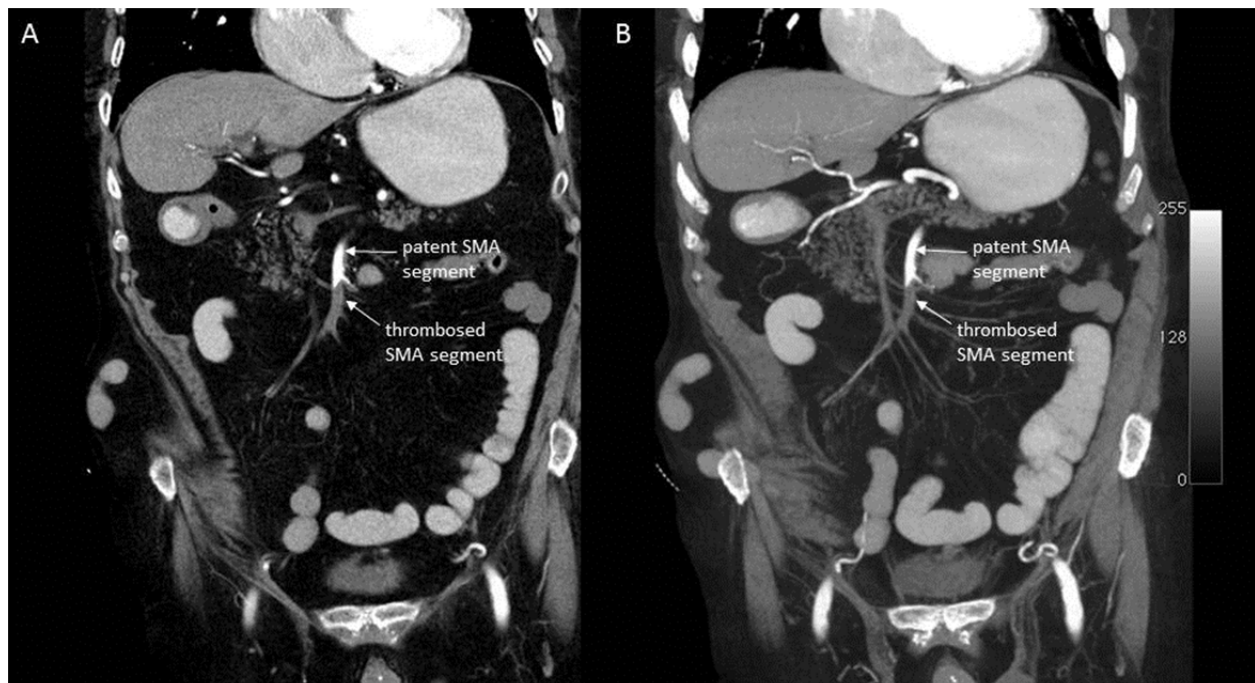


Figure 1.3 CT with IV contrast in patient with superior mesenteric artery thrombosis. Oral contrast was administered but was not required. IV contrast was essential to the diagnosis by revealing a filling defect in the SMA.

A, standard coronal reconstruction.

B, a maximum intensity projection (MIP) image, which makes more of the SMA visible in a single image.

Bowel appears normal on this initial CT scan. Meanwhile, the patient is noted to have an elevated troponin T. He is admitted and treated with angiographic placement of a catheter into the origin of the superior mesenteric artery, through which tissue plasminogen activator is infused as a bolus and drip – 11 hours after ED arrival (Figure 1.4).

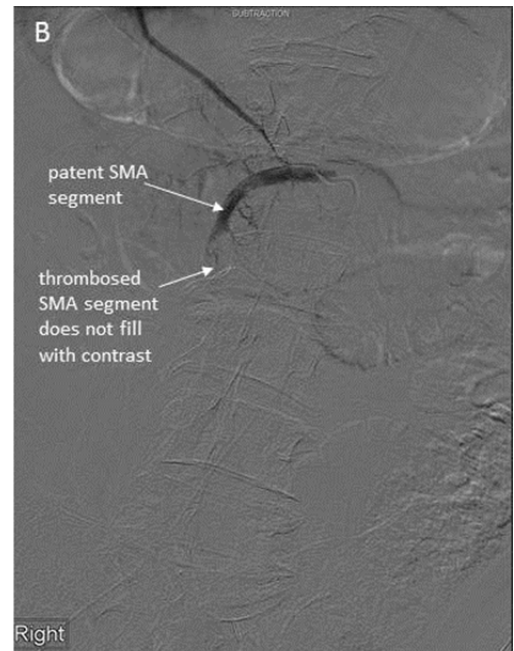
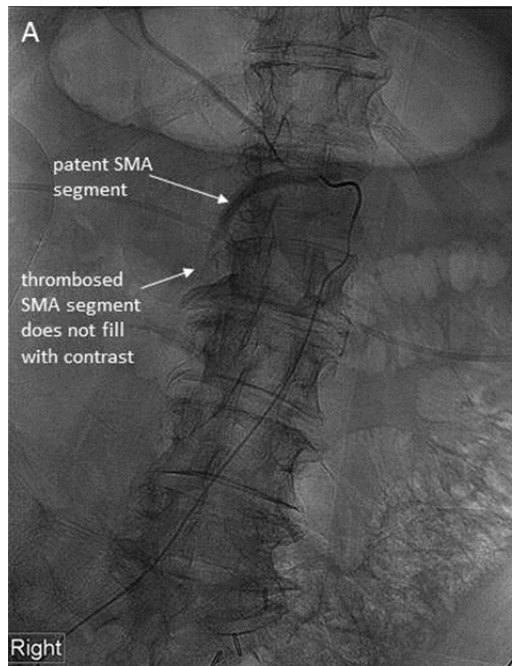


Figure 1.3. Angiogram in same patient.

A, an angiogram showing a catheter infusing contrast into the proximal SMA. This fills but then abruptly ends at the point of thrombosis. B, the same image, with static structures digitally subtracted. Compare with the prior figure showing the coronal CT images.

24 hours later a repeat mesenteric angiogram demonstrates no improvement in the occlusion of the superior mesenteric artery. An emergency laparotomy is performed and shows extensive small bowel infarction. Bowel resection is performed with no attempt at embolectomy. Despite ICU care, the patient develops multiorgan failure and pneumonia, and the family decides to withdraw care. The patient expires 14 days after ED presentation.

Discussion

This case demonstrates a classic presentation of mesenteric ischemia with acute embolic occlusion of the superior mesenteric artery (SMA) resulting from atrial fibrillation. The patient's age, past medical history, and clinical presentation are typical. The laboratory abnormalities are also well-described, with both an elevated lactate and leukocytosis. Some important learning points from this case emerge.

Atrial fibrillation and mesenteric ischemia

Atrial fibrillation has been implicated as a major risk factor for mesenteric ischemia, particularly embolic occlusion of the superior mesenteric artery. Acosta *et al* (2003) found atrial fibrillation to be present in 95% of patients with acute SMA occlusion – though this study included only 20 such patients, due to the rarity of the condition.¹ In this study, only 10% of such patients were being treated with Coumadin at the time of their embolic event. Gorey (1988) found atrial fibrillation in 43% of 65 cases of extensive mesenteric infarction.⁹

Lactate measurement and mesenteric ischemia

An elevated lactate level is commonly found in cases of mesenteric ischemia, although the degree of elevation or the presence of any elevation is likely dependent on many factors, including the duration of ischemia before ED presentation and the presence of collateral blood flow. In one study of 85 patients, including 20 ultimately diagnosed with intestinal ischemia, lactate elevation was 100% sensitive although only 42% specific.¹² Studies such as this fall short of proving the sensitivity of lactate testing, for several reasons. Small numbers necessarily result in wide confidence intervals; in this case, the lower 95% confidence interval shows a sensitivity of only about 80%, meaning that as many as 1 in 5 cases of ischemia might have normal lactate values. Larger studies are needed to surmount this limitation. More fundamentally, studies such as this may suffer from biases related to our preconceived notions of intestinal ischemia. It may be that patients with non-classical presentations do not undergo lactate testing, or that patients with normal lactate values do not undergo definitive testing for intestinal ischemia. Unless cases such as these are included in the study population, and unless the outcomes for all cases are rigorously confirmed, spectrum,¹³ selection, and verification (also called confirmation) biases undermine the validity of a study's results ([see table in appendix](#)). Verification bias has been shown to substantially alter the results of calculations of sensitivity for diagnostic tests.¹⁴

X-rays and mesenteric ischemia

X-rays should play little role in the diagnosis of acute abdominal pain in general, and of mesenteric ischemia in particular. Numerous studies have documented the lack of utility of abdominal x-rays for the vast majority of patients with abdominal pain. Eisenberg *et al* (1982, 1983) found that only 10% of abdominal x-rays showed abnormalities.^{15, 16} McCook *et al* (1982) prospectively analyzed x-rays in 96 consecutive patients with abdominal complaints and found that 98% were either negative or had positive findings unrelated to the presenting clinical problem.¹⁷ Campbell *et al* (1988) found that the rates of false positive findings on abdominal x-ray were so high that the information gained was more likely to be misleading than helpful.¹⁸ MacKersie *et al* (2005) compared unenhanced CT and 3-view acute abdominal x-ray series in 91 emergency department patients with non-traumatic acute abdominal pain, using a diagnostic criterion standard of surgical or pathological findings or clinical follow-up. Abdominal x-rays had a sensitivity of only 30% and a specificity of 88%, while unenhanced CT with 5mm slice thickness was 96% sensitive and 95% specific.¹⁹ Kellow *et al* (2008) found plain x-rays to assist in confirming the suspected diagnosis in only 2 to 8% of emergency department cases, with follow-up imaging performed in 50% of cases regardless of the x-ray findings.²⁰

In the setting of mesenteric ischemia, key x-ray findings such as intramural pneumatosis intestinalis or portal venous gas are rare, making x-rays relatively unhelpful. Smerud *et al* (1990) retrospectively reviewed 23 cases of proven mesenteric infarction, comparing x-ray and CT findings. Pneumatosis intestinalis was observed in only 1 case (4%). In an additional 6 patients (26%), focally edematous bowel was suggested by x-ray. 70% of patients with proven infarction had no x-ray findings specifically suggesting the diagnosis.²¹ Chien-Hua *et al* (2007) reviewed 12 cases of intestinal ischemia

and found 3 cases (25%) with abnormal right upper quadrant gas or pneumatosis. 9 patients (75%) had only dilated loops of bowel.²² Gorey *et al* (1988) found that 9 of 42 (21%) patients with proven mesenteric ischemia had normal pre-operative x-rays, while 33 (79%) showed non-specific fluid levels. None had findings specific for intestinal ischemia.⁹

CT scan in mesenteric ischemia

Modern CT protocols for the detection of mesenteric ischemia do not call for the use of oral contrast.^{23, 24} The use of oral contrast is associated with an interval of approximately two to three hours before contrast is ingested and CT scan is performed,^{25, 26} a potentially critical delay if bowel infarction is progressing. In this patient's case, the sole abnormality noted was an occlusive thrombus in the proximal SMA. The use of oral contrast was neither necessary nor beneficial to the diagnosis. We'll discuss CT of mesenteric ischemia in more detail later in this article.

Treatment of mesenteric ischemia

Another critical decision in this case was the choice of treatment: surgical embolectomy versus intra-arterial administration of thrombolytic therapy. Due to the presence of non-ST segment elevation myocardial infarction suggested by the patient's elevated troponin, angiographically directed thrombolytic therapy was selected. However, failure of this therapy allowed progression of bowel infarction, likely contributing to the patient's death. While many factors must be weighed in the decision to operate, in this case the presence of a positive troponin motivated a non-operative intervention which failed to achieve its goal. Of course we can never know from any individual case whether the outcome would have been different if the patient had undergone surgical embolectomy. However, because nearly the entire small bowel is perfused via the SMA, the stakes are particularly high in this case. The evidence for nonsurgical therapy of acute SMA thrombosis is limited to case series. Schoenbaum *et al* (1992) describe 4 patients with SMA thrombosis successfully treated with intra-arterial urokinase during angiography – but 2 patients required laparotomy for resection of infarcted bowel. Angiography does not allow visual inspection of bowel for signs of ischemia, an advantage of laparotomy or laparoscopy. Even if recanalization of the affected artery is achieved with thrombolytic therapy, a patient may develop SIRS or sepsis if infarcted bowel is not recognized early – possibly leading to death. Schoots *et al* (2005) performed a systematic review of the literature from 1966 to 2003 and found only 20 case reports and 7 case series totaling 48 patients treated angiographically for acute SMA occlusion. The authors noted angiographic resolution of thrombus in 43 of 48 patients (90%). However, because angiographic resolution of thrombus does not rule out bowel necrosis, one third of patients with angiographic "success" in this series underwent laparotomy to inspect for ischemic bowel, and 8 of 43 (19%) required resection of necrotic bowel. Moreover, 5 patients with angiographic resolution died between 3 days and 2 months after therapy – but the authors attributed the deaths to causes other than bowel ischemia, such as sepsis or myocardial ischemia. These results almost certainly reflect biases including selection bias and publication bias, with poor outcomes not being reported in the medical literature, so the true success rate with angiographic reperfusion therapies may be even lower.²⁷ The ACC/AHA 2005 Practice Guidelines for the Management of Patients with Peripheral Arterial Disease (Lower Extremity, Renal, Mesenteric, and Abdominal Aortic) recommend surgical treatment of

acute obstructive intestinal ischemia with revascularization, resection of necrotic bowel, and a "second look" operation in 24 to 48 hours in some cases. This recommendation is based on level "B" evidence ([see table in appendix](#)), indicating the lack of methodologically strong studies comparing treatment approaches. Endovascular approaches including transcatheter lytic therapy, balloon angioplasty, and stenting are given a level "C" evidence rating, with a specific caveat that laparotomy may still be required.²⁸⁻³⁰

Treatment delay in mesenteric ischemia

This patient experienced a delay of 11 hours from ED arrival until angiography was performed. Surgery was delayed in this patient by another 24 hours due to the decision to perform transcatheter thrombolysis initially. Delay in treatment and progression of infarction is associated with worsened outcomes. Ritz *et al* (2005) found that the time until surgery was a strong prognostic factor.⁷ Acosta *et al* found that the length of ischemic bowel predicted acute mortality (2003).¹ Gorey *et al* (1988) found an association between shorter interval to laparotomy and less bowel resected with survival.⁹ Deehan *et al* (1995) found that a diagnostic delay less than 12 hours was associated with improved survival.¹⁰

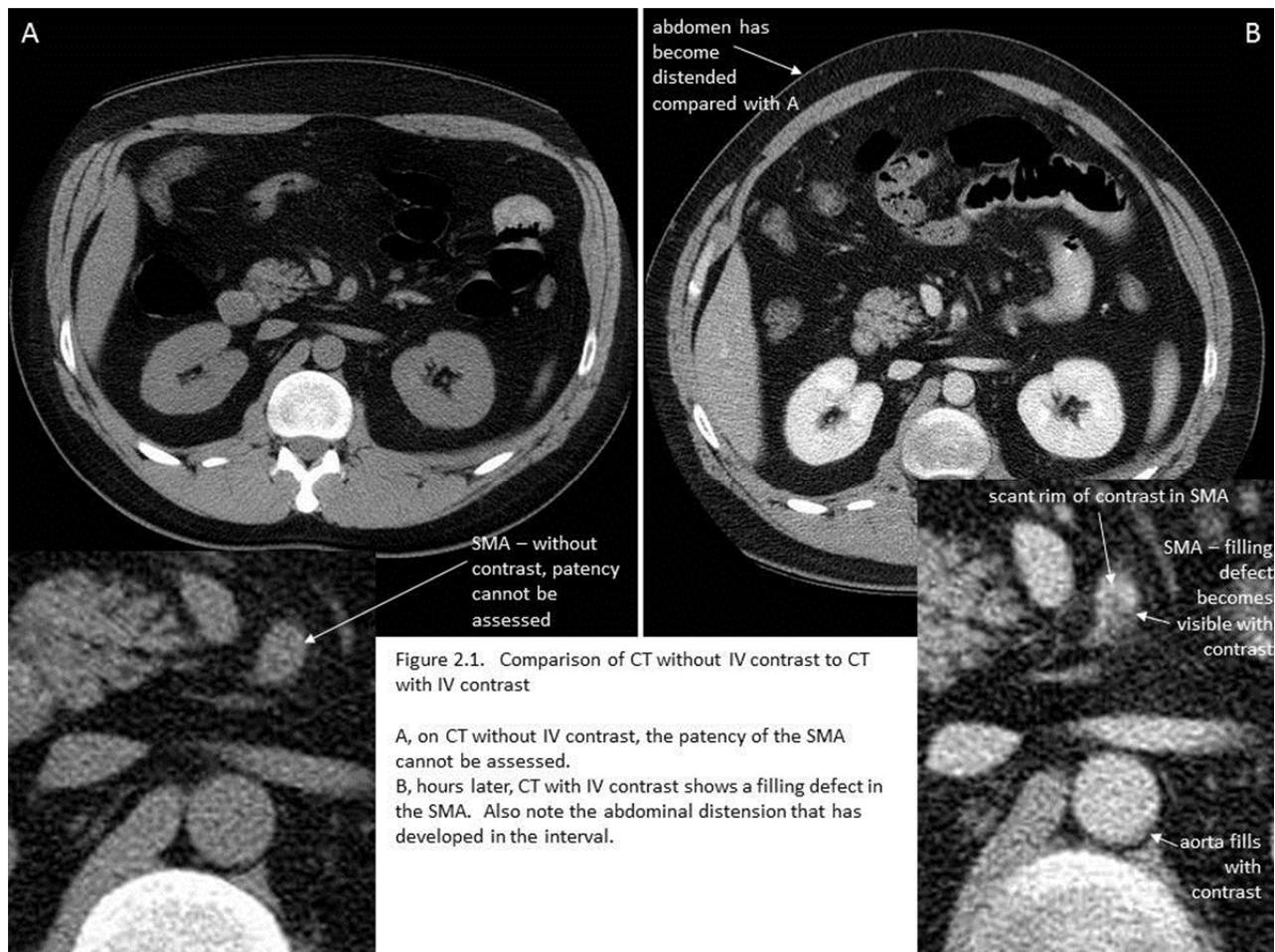
Pearls and Pitfalls

- No oral contrast is needed for CT mesenteric angiography. Oral contrast introduces an unnecessary time delay.
- Abdominal plain films rarely provide useful information in the setting of mesenteric ischemia. They introduce unnecessary delay and should generally be avoided. Often, the plain film findings may be falsely reassuring by providing an alternative diagnosis of ileus or bowel obstruction.
- Elevation of lactate is common in mesenteric ischemia, although nonspecific. A normal lactate may be encountered early in the course of ischemia, prior to the onset of true infarction. Lactate elevations may reflect sepsis or other states of systemic hypoperfusion, rather than bowel infarction.
- No large randomized controlled trials exist comparing surgical embolectomy to intra-arterial thrombolysis and other endovascular therapies. Surgery has the advantage of allowing visual inspection of bowel for evidence of ischemia or infarction, findings which may be missed by CT or fluoroscopic angiography. If the patient can tolerate surgery, this may be the best option, as progression of bowel infarction may offer no second chance if initial thrombolytic therapy fails.

Case 2

A 37-year-old male with no past medical or surgical history presents with abdominal pain of 24 hours duration, worsening 6-8 hours before emergency department arrival. His vital signs are normal (blood

pressure 119/77, heart rate 93, respiratory rate 20, temperature 35.6 C, oxygen saturation 99% RA), and he receives a triage level 3. A resident examines him in a triage room and orders a non-contrast abdominal-pelvic CT following a renal colic protocol, with the stated indication being left flank pain. CT is performed and demonstrates normal findings, including normal liver, gallbladder, spleen, pancreas, and bowel, with no renal or ureteral calculi (**figure 2.1a**). The patient is ultimately placed in a room, and both the resident and attending physician note right upper quadrant tenderness. The patient is noted to have leukocytosis (25k), new hyperglycemia (glucose 239mg/dL), and an anion gap of 27. Given the patient's right upper quadrant pain and an AST of 47U/L (upper limit of normal 41), a right upper quadrant ultrasound is obtained but is normal. EKG shows normal sinus rhythm. A venous blood gas is obtained due to the patient's hyperglycemia and demonstrates a lactate of 3.6mmol/L and a pH of 7.32. Mesenteric ischemia is suspected at this point and CT scan with IV and oral contrast is ordered. This CT is performed 12 hours after ED arrival and is interpreted by the overnight radiology resident as consistent with inflammatory bowel disease, based on findings including mesenteric fat stranding and mild thickening of the distal small bowel. Four hours later, an attending radiologist reviews the CT and concludes that the superior mesenteric artery is occluded near its origin (**figure 2.1b**).



Surgery is consulted and the patient undergoes emergency laparotomy nearly 19 hours after emergency department arrival. Operative findings demonstrate near complete infarction of the small bowel

(240cm) and right and transverse colon, which are resected. An SMA embolectomy is performed. The patient survives but is left dependent on total parenteral nutrition.

Discussion

This difficult case illustrates several important points.

Atypical patient profile: the young patient with ischemic bowel

The patient's young age and lack of any known risk factors contribute to an apparently low pretest probability of mesenteric ischemia. Numerous studies show advancing age to be a significant risk factor for mesenteric ischemia, but these same studies demonstrate a wide age range. Gorey *et al* (1988) studied 65 patients over a 10 year period and found an age range of 20 to 96 years, with a mean age of 69 years.⁹ Bjorck *et al* (2002) studied 60 patients undergoing revascularization procedures and found a median age of 76 years – but with a range of 35 to 90 years.³¹ Acosta *et al* (2003) found a median age of 83.5 (range 63-96 years). Acosta *et al* (2004) conducted a large population-based study of the incidence of acute SMA thrombosis and found 2 cases in patients between the ages of 35 and 39 years and 2 cases in patients of ages 45 to 49 years over a twelve year period – all resulting in death. In this study, the median age was 81 years, with a range from 39 to 103 years.² Mesenteric ischemia is a disease heavily skewed toward an older population, but young patients will rarely develop the condition. When dismissing the possibility of ischemia, add 30 years to the patient's age and reconsider the possibility.

Pain out of proportion to exam: a typical presentation of mesenteric ischemia?

The patient had localized pain initially, variously noted by physicians to be in the right flank or right upper quadrant. None of the physician notes documented an appearance of "pain out of proportion to exam." Does "pain out of proportion to exam" accurately reflect the presentation of patients with acute mesenteric ischemia? Two studies suggest that it may not in many patients. Edwards *et al* (2003) retrospectively reviewed the charts of 76 patients ultimately diagnosed with acute mesenteric ischemia over a 10 year period at Wake Forest University. According to the medical records, 64% demonstrated peritonitis at ED presentation.³² Deehan *et al* (1995) reviewed 43 cases of acute ischemia over a 10 year period and found that 48% had diffuse non-specific tenderness at presentation, while 52% had signs of peritonitis.¹⁰ How can we explain this aberration from our paradigm for mesenteric ischemia? Of course, it is possible that these two small studies include patient populations that are not representative of the vast majority of mesenteric ischemia cases. Nonetheless, they suggest that an impressively abnormal abdominal exam may be present when ischemia is present. One explanation is that mesenteric ischemia has a range of presentations, depending on the degree of ischemia or infarction and the elapsed time from symptom onset to ED presentation. Patients who

present in a delayed fashion may have progressed to frank bowel infarction or perforation, in which case the abdominal exam may also progress to nonspecific findings of peritonitis. Perhaps early-presenters would be characterized by pain disproportionate to examination.

It may appear counterintuitive that a concerning physical exam with findings of peritonitis might lead to diagnostic delay, but a simple explanation may be at play. Significant tenderness or peritoneal signs may alert the emergency physician to the presence of a serious intra-abdominal condition, but they may at the same time cause the physician to categorize the patient as having an infectious or inflammatory condition, requiring urgent care but not immediate operative therapy. The ED course may then consist of appropriate interventions such as antibiotic and fluid administration and surgical consultation, but without the extreme urgency warranted for an ischemic insult. Leukocytosis may have a similar effect on the physician, raising concerns about infections such as appendicitis, cholecystitis, or diverticulitis rather than bowel ischemia.

Vital signs in mesenteric ischemia

This patient had normal vital signs at triage despite experiencing a life-threatening condition. Normal vital signs contribute to a lower triage acuity score and may cause physicians to be falsely reassured about the patient's stability and the need for rapid evaluation. Unfortunately, mesenteric ischemia, like other ischemic conditions, may cause irreparable harm despite normal vital signs. Consider the case of myocardial infarction, ischemic stroke, or testicular or ovarian torsion – each of which can present with normal vital signs. Time is muscle, time is brain, time is testicle, time is ovary,... and time is bowel. Emergency physicians must be schooled to add mesenteric ischemia to the list of conditions which can kill or irreversibly injure a patient despite "clinical stability." Gorey *et al* (1988) found that in patients with extensive intestinal necrosis, only 28% presented with hypotension.⁹ Patients with mesenteric ischemia fool emergency physicians in another insidious way – they die in a delayed fashion, though 60% will die within 72 hours after admission.⁸ This life-threatening and time-dependent condition can appear clinically benign to the emergency physician who will not witness the patient's death from delayed diagnosis or treatment.

Lab profiles in mesenteric ischemia

The patient's laboratory findings are classic for mesenteric ischemia, with profound leukocytosis, an anion gap, and an elevated lactate. Despite this, other less time-critical diagnoses such as biliary disease and renal colic appeared to take precedence due to atypical features of the clinical presentation. Gorey *et al* (1988) found that in patients with extensive mesenteric infarction, 65% had leukocytosis, and 67% had metabolic acidosis. If the patient in this case had been an elderly male with known vascular disease, perhaps the diagnosis of mesenteric ischemia would have been pursued more aggressively early in the patient's course, based on these lab findings.

CT scan technique in mesenteric ischemia

The initial CT scan performed without any form of contrast was unable to diagnose a filling defect in the proximal superior mesenteric artery - calling attention to the necessity for vascular

contrast when mesenteric ischemia is suspected. Once the diagnosis of mesenteric ischemia was suspected, oral contrast was not necessary and resulted in a delay to performance of the CT scan. After the CT scan had been performed, abnormalities of the bowel wall were misattributed to inflammatory bowel disease, when these findings can be signatures of bowel ischemia. An abnormality of the origin of the superior mesenteric artery was overlooked-- reinforcing the need to communicate the suspected diagnosis clearly to the radiologist to ask specifically whether mesenteric vessels are patent. Although a surgical consult was ultimately obtained and surgery was performed, a combination of factors contributed to diagnostic and treatment delay with a catastrophic outcome.

CT technique plays an important role in the diagnosis of mesenteric ischemia, described in detail in the appendix. Modern CT scan protocols designed for evaluation of mesenteric ischemia differ from those used for "standard" or "generic" abdominal pain in four important ways: timing of image acquisition relative to IV contrast administration to include arterial and portal venous phases of enhancement, thin CT slice thickness to allow detection of small vascular abnormalities, the use of multiplanar reformations, and the omission of oral contrast. Oral contrast is not required in published CT protocols for evaluation of mesenteric ischemia ([described in detail in the appendix](#)).^{23, 24}

Because mesenteric ischemia is a time-critical diagnosis, the patient with suspected mesenteric ischemia must receive a high priority to undergo CT scan as soon as possible. In addition, the radiologist protocoling and interpreting the CT scan should evaluate the images directly for findings of occlusion of mesenteric blood vessels, as well as for secondary findings of mesenteric ischemia including bowel wall thickening, pneumatosis, abdominal free fluid, mesenteric fat stranding, and pneumoperitoneum, described below and in the accompanying figures. Discussion with the radiologist allows a CT tailored to the patient's complaint to be performed.³⁶

What are the CT findings of mesenteric ischemia?

CT findings of mesenteric ischemia include direct signs of arterial or venous occlusion, secondary signs of bowel ischemia, and findings in other organs suggesting embolic disease. Direct vascular findings include filling defects in the celiac, superior mesenteric, or inferior mesenteric arteries, or in the portal or inferior mesenteric veins. Portal venous gas may rarely be seen (5% in one study). Secondary signs of bowel ischemia include pneumatosis intestinalis (seen in 38% in one study), bowel wall thickening, lack of bowel wall enhancement with IV contrast, and mesenteric fat stranding.^{23, 24} These signs are nonspecific, but when multiple findings are identified in concert, they paint a pattern consistent with ischemia. Circumferential bowel wall thickening, a narrowed intestinal lumen, and a segmental distribution can be seen in both bowel ischemia and intramural hematoma.³⁷ Macari *et al* (2003) found an overlap in the appearance of ischemic bowel and bowel with intramural hemorrhage, though thickening less than 1cm and long segment thickening (>30cm) were more suggestive of ischemia.³⁸ Solid organs such as the liver, spleen, and kidneys may show wedge-shaped hypodensities suggesting embolic infarction of these organs, which can support a diagnosis of embolic mesenteric ischemia.

How sensitive is CT? Does a normal CT rule out bowel ischemia?

Klein *et al* (1995) compared x-ray, CT, and angiography to final clinical diagnosis in 54 cases of mesenteric infarction. X-ray diagnosed only 28% of cases, angiography diagnosed 14 of 16 cases (sensitivity 87.5%), and CT diagnosed 18 of 22 (82%). In this small study, no statistical difference between angiography and CT was observed.³⁹ More recent studies of CT using tailored mesenteric ischemia protocols (described above) show higher sensitivity. Ofer *et al* (2009) reported a sensitivity of 88.8% and specificity 97.2%, using a 16 slice CT scanner with arterial and portal venous phase imaging with IV contrast and no oral contrast.⁴⁰ Aschoff *et al* (2009) reported sensitivity 93% and specificity 100% with 16 or 40 slice CT angiography. Zandrino *et al* (2006) reported sensitivity 92% and specificity 100% using a similar protocol.²⁴ Kirkpatrick *et al* (2003) reported a sensitivity of 96% and specificity of 94%.²³ These studies are all relatively small and may reflect a selected patient population with advanced or severe disease. The sensitivity of CT might be expected to vary depending on the degree of ischemia or infarction and the time elapsed from symptom onset to performance of CT. In patients with significant concern for ischemia, a negative CT should be viewed skeptically, given the morbidity and mortality if misdiagnosis occurs. Without IV contrast, direct visualization of vascular anomalies is unlikely, and CT would likely only be positive in more advanced cases of ischemia with secondary findings of bowel injury. The prognosis in these cases would be expected to be poor.

Pearls and Pitfalls

- Rarely, young patients without known medical history can develop mesenteric ischemia. Severe and unexplained abdominal pain should prompt consideration of mesenteric ischemia.
- Substantial leukocytosis can be seen in mesenteric ischemia. The presence of leukocytosis should not deflect the emergency physician from the diagnosis of mesenteric ischemia, although infectious and inflammatory etiologies must also be considered.
- An elevated lactate level is a sensitive but nonspecific finding which should raise suspicion of mesenteric ischemia
- An anion gap metabolic acidosis (due to lactic acidosis) is often seen in mesenteric ischemia. The anion gap can be readily calculated from standard laboratory data, and an elevated anion gap should prompt consideration of the cause, including possible mesenteric ischemia.
- Pain may appear localized, and significant abdominal tenderness may occur. The patient may not have pain out of proportion to exam.
- Atrial fibrillation not always be present, as *in situ* thrombosis can occur
- Non-contrast CT cannot demonstrate vascular filling defects. Early in the course of mesenteric ischemia, when intervention to restore bowel perfusion might be most beneficial, secondary CT findings such as bowel wall thickening, free fluid, free air, and fat stranding may not yet be present. A normal non-contrast CT scan does not rule out mesenteric ischemia.
- Oral contrast is unnecessary for the diagnosis of mesenteric ischemia and introduces diagnostic delay.
- Communication with the radiologist about the suspected diagnosis is important before CT scan is performed and for optimal image interpretation. Speaking with the radiologist in advance of the CT scan allows the CT images to be acquired using a protocol optimized for evaluation of the mesenteric vessels.

- Observation, a hallmark strategy for unexplained abdominal pain in emergency medicine practice, can be devastating in the case of mesenteric ischemia. Ischemic bowel can progress to infarction within a matter of hours. By the time exam findings change or laboratory values worsen, the window for successful therapeutic intervention may have closed. Once the diagnosis is suspected, it should be aggressively pursued as quickly as possible.
- Delayed diagnosis and treatment is associated with increased mortality.

Case 3

*An 81 year-old female with a history of lymphoma and atrial fibrillation presents with vomiting and abdominal pain of 12 hours duration. She is in atrial fibrillation on emergency department arrival and has a subtherapeutic INR (0.9). Her vital signs are normal (blood pressure 124/71, heart rate 73, respiratory rate 18, temperature 36.5C, oxygen saturation 99% on room air). Her exam shows diffuse abdominal tenderness with guarding. A chest x-ray 90 minutes after arrival shows no free air. The patient has a white count of 19.7k on labs drawn earlier in the day in clinic, compared with her baseline value of 4.5k. Her initial ED labs show a white blood cell count of 7.2k, a bicarbonate of 26mmol/L, and an anion gap of 7. Given the patient's age and atrial fibrillation, the emergency physician considers a broad differential diagnosis including bowel obstruction, infection, and mesenteric ischemia. A venous blood gas shows a pH 7.25 and lactate 8.5mmol/L (upper limit of normal 1.6). Oral contrasted CT is ordered, resulting in a delay of over 2 hours before CT. Despite this, the patient is unable to tolerate the contrast, vomiting it repeatedly. CT is performed and shows gross free air and free fluid, as well as bowel wall thickening in the descending colon (**figure 3.1, also 3.2 for comparison with a similar case**).*

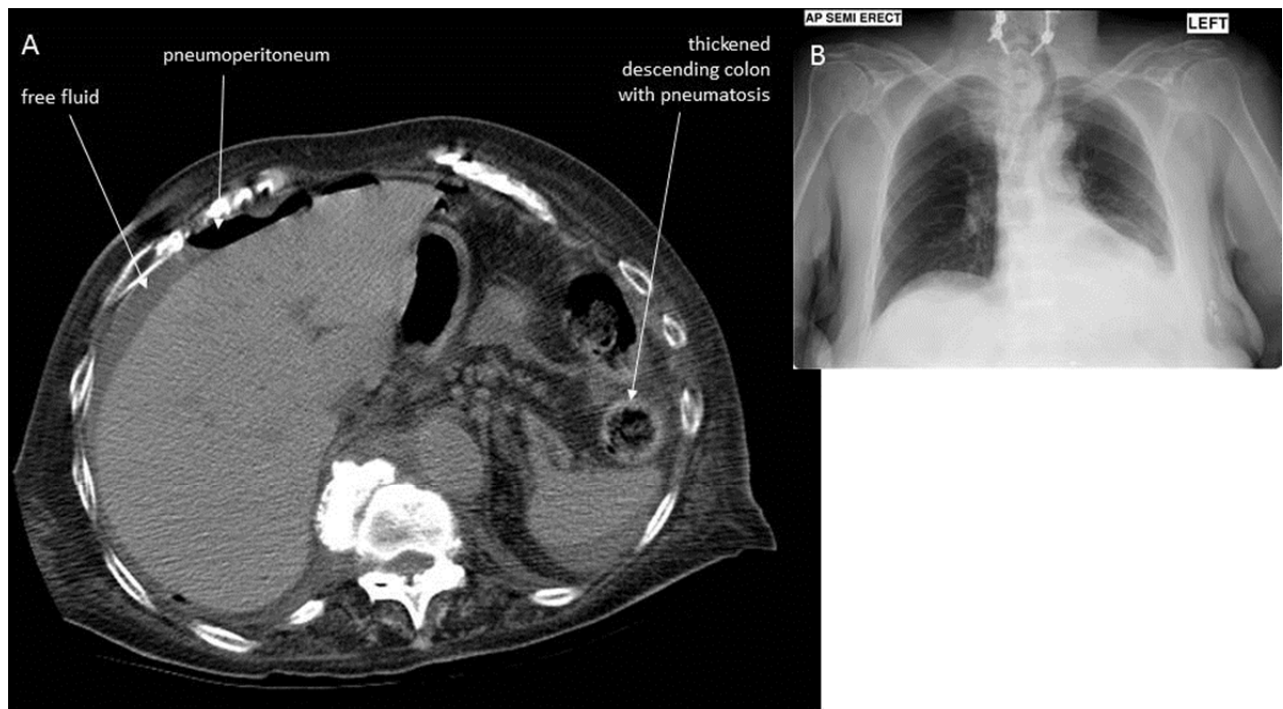


Figure 3.1 Ischemic colitis with free air

While evaluation for vascular occlusion is compromised on this CT due to lack of IV contrast, free air and fluid are readily seen (A).

Considering the large amount of free air seen on CT, the absence of visible free air on x-ray (B) is surprising – but supported by literature indicating poor sensitivity of plain film.

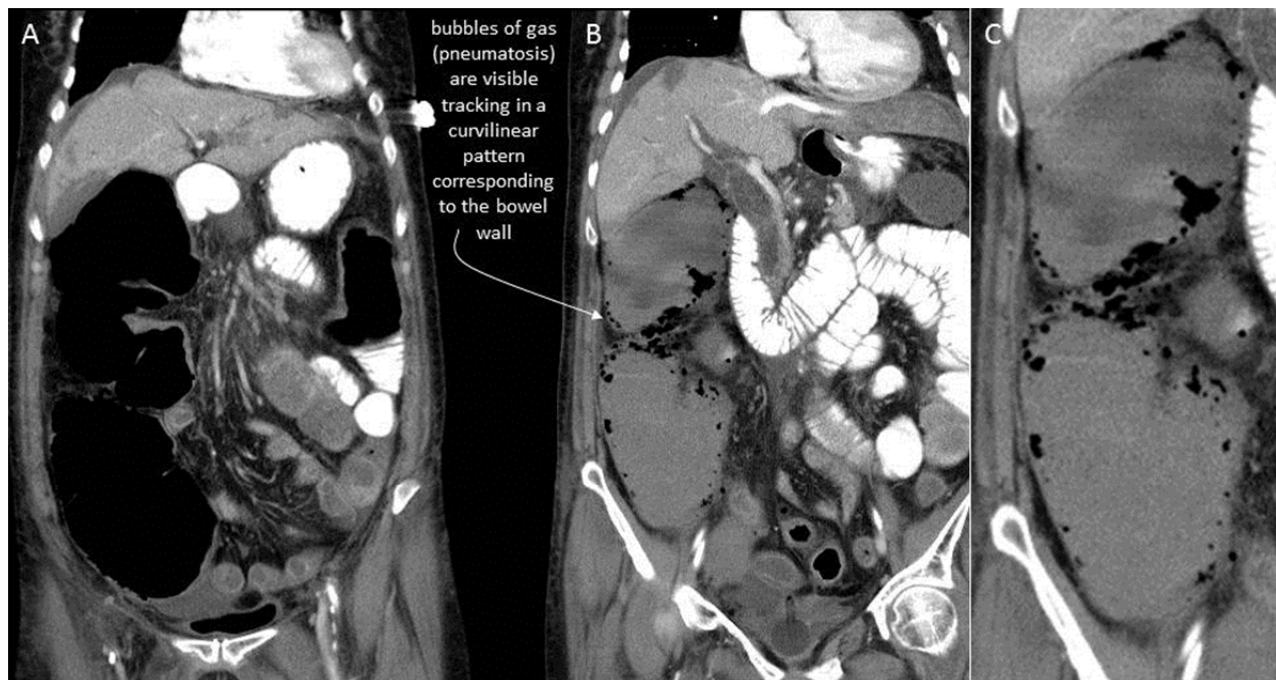


Figure 3.2 Bowel infarction (ischemic colitis with pneumatosis), CT with IV and oral contrast (abdominal windows)

Another patient with ischemic colitis. This 61 year old female presented with sepsis, lactic acidosis, and frank pneumatosis on x-ray, due to ischemic colitis. Unstable despite pressor medications, she underwent exploratory laparotomy, extensive lysis of adhesions, subtotal colectomy with end ileostomy, small-bowel resection with primary anastomosis, and splenectomy. Miraculously, she survived, given the severity of her illness on presentation. The operative findings suggested that her ischemia may have been due to extensive adhesions, rather than due to a single vascular obstruction.

Coronal reconstructions show massive dilatation of large bowel, as well as frank pneumatosis. A, an anterior slice shows the bowel to be dilated and air-filled (remember, the patient was supine during CT, so an anterior coronal plane represents the superior portion of the abdomen). B, a

Without IV contrast, no specific vascular occlusion is identified. Within hours, the patient becomes unstable in the emergency department, is intubated and treated with vasopressors and antibiotics, and then undergoes emergency laparotomy – 12 hours after ED arrival. During this interval, the patient became neutropenic with her total white blood cell count falling to 0.9k only 19 hours after her original count of 19.7k. Laparotomy reveals stool in the abdominal cavity. The sigmoid colon is found to be necrotic with multiple areas of perforation and is resected. 4 days later, the patient is in multiorgan failure. The family withdraws care and the patient expires.

Discussion

This patient illustrates some classic features of mesenteric ischemia, as well as some important clinical dilemmas. She is elderly and is in atrial fibrillation, both risk factors for mesenteric ischemia – but other aspects of her presentation are less suspicious. Her vomiting may suggest bowel obstruction as a leading diagnosis – though studies show that ileus and even obstruction are common plain x-ray findings that can mislead the physician. It should come as little surprise that malperfused bowel would exhibit an appearance of ileus, since peristalsis ceases in the face of ischemia. Moreover, bowel ischemia occurs as a secondary complication of as many as 14% of cases of primary mechanical bowel

obstruction, and necrosis occurs in 9%, so findings of obstruction should not allay fears of ischemia.⁴¹ The patient's lactate elevation and leukocytosis fit with mesenteric ischemia, but also with incipient sepsis from an intra-abdominal source of infection.

This patient has ischemic colitis, a variant of gastrointestinal ischemia estimated in some studies to account for over half of all intestinal ischemia cases. As many as 3 cases per 1000 hospital admissions are thought to occur, although the condition may be underdiagnosed.⁴² CT findings of colitis are nonspecific, and may overlap between infectious, inflammatory, and ischemic forms. Findings suggesting ischemia include bowel wall thickening in a regional distribution corresponding to a vascular territory. Often no large vessel occlusion is seen.⁴³ Ischemic colitis typically occurs in patients at advanced ages and may complicate systemic hypoperfusion and hypovolemic states. Although the disease is by definition one of vascular insufficiency, often vascular volume resuscitation and antibiotics are sufficient to reverse the clinical course. The clinical presentation can resemble infectious colitis, with bloody diarrhea or frank rectal bleeding. Laboratory testing can be nonspecific, with elevated lactate having an uncertain significance – systemic hypoperfusion versus focal bowel ischemia. Unlike acute superior mesenteric artery occlusion, ischemic colitis often does not require surgery, although colectomy can be required in severe cases.

Diagnostic delays

The patient suffers diagnostic delay from a now familiar cause: an attempt at oral contrast administration. While oral contrast may benefit diagnosis of some abdominal conditions, it is required neither for diagnosis of mesenteric ischemia, nor for leading competing diagnoses, including pneumoperitoneum and small bowel obstruction. In its published evidence-based "appropriateness guidelines," the American College of Radiology gives abdominal CT with IV but without oral contrast its highest rating, specifically commenting that positive oral contrast can obscure findings of bowel wall ischemia.⁴⁴ Other serious abdominal disorders including appendicitis and diverticulitis can be diagnosed with high sensitivity and specificity using CT without oral contrast, although controversy remains about the potential benefit of oral contrast.^{1, 45}

Role of IV contrast in CT

IV contrast is not administered in this case – for uncertain reasons, as the patient has no reported allergy and normal renal function. In earlier cases and figures in this article, we've emphasized the key importance of IV contrast to reveal arterial occlusions in the diagnosis of mesenteric ischemia. Can CT without IV contrast assist in the diagnosis? Well-designed trials have not addressed this question, but several letters to the editor of major radiology journals point out that secondary findings of ischemia, such as free fluid, free air, and fat stranding, can be recognized without IV contrast.⁴⁶⁻⁴⁸ These findings are relatively nonspecific for ischemia but may be sufficient to direct therapy, including laparotomy in the case of free air. Therefore, while a normal CT without IV contrast could fail to detect mesenteric ischemia (**see figure 2.1**), an abnormal scan might reveal sufficient clinically relevant information. Additional imaging could be performed if rapid non-contrast CT did not reveal the diagnosis. If iodinated IV contrast is contraindicated, other diagnostic options could include MR

angiography with gadolinium, laparoscopy, or laparotomy. MRI with gadolinium should be avoided when possible in patients with acute renal failure or chronically diminished creatinine clearance including patients receiving hemodialysis, due to the risk of gadolinium-associated fatal nephrogenic systemic fibrosis.⁴⁹ This condition has been predominantly associated with use of a specific gadolinium contrast agent, Omniscan (gadodiamide), and remains quite rare, with an incidence of 0.4% in patients receiving chronic hemodialysis and 8.8% in patients with GFR < 15mL/min not undergoing dialysis and exposed to high-dose gadolinium. If MRI with gadolinium is used in patients with moderate to end-stage renal disease, the American College of Radiology (ACR) recommends written informed consent and minimizing the contrast dose. For hemodialysis patients, the ACR also recommends dialysis within 2 hours of the administration of the gadolinium agent.⁵⁰

Pearls and Pitfalls

- Elderly patients with abdominal pain should be suspected of mesenteric ischemia including ischemic colitis, even when plain films suggest an alternative explanation. In most cases, plain films should be avoided as they can introduce diagnostic delay. When x-rays are obtained, ischemia must be considered regardless of x-ray findings.
- Atrial fibrillation should raise suspicion of mesenteric ischemia
- An elevated lactate, while nonspecific, should raise suspicion of mesenteric ischemia and sepsis or other shock states. Definitive diagnostic testing and initiation of treatment should occur as quickly as possible.
- An elevated white blood cell count, while nonspecific, is commonly seen with mesenteric ischemia and should not be attributed solely to "stress response" or infection. A falling white blood cell count is an ominous prognostic sign.
- Oral contrast is not required for the diagnosis of mesenteric ischemia, small bowel obstruction, or free air.
- If IV contrast cannot be safely administered due to problems such as renal insufficiency, CT without oral or IV contrast can reveal a range of important findings including pneumoperitoneum, small bowel obstruction,⁵¹ free fluid, fat stranding, appendicitis,⁵² diverticulitis,⁵³ and abdominal aortic aneurysm.
- Rectal bleeding and abdominal pain can suggest ischemic colitis, particularly in the elderly.
- The presentation of ischemic colitis may overlap with acute mesenteric ischemia from SMA occlusion, which should also be ruled out.
- Infectious or inflammatory colitis may have a similar clinical, laboratory, and CT appearance.
- Treatment of ischemic colitis is often supportive, with antibiotics and fluid resuscitation
- Colectomy may be required in advanced or severe cases.

Case 4

A 21-year-old woman presents with two weeks of severe right upper quadrant abdominal pain which she describes as "like labor contractions." She has no surgical history. She is three months postpartum from an uncomplicated vaginal delivery. Her vital signs are normal (blood pressure 134/67, heart rate 80,

respiratory rate 20, temperature 36.0C), and exam shows a tender right upper quadrant and epigastric area, without guarding or rebound. Suspecting biliary colic or acute cholecystitis, the emergency physician performs a bedside transabdominal ultrasound. This demonstrates no gallstones, pericholecystic fluid, gallbladder wall thickening, or sonographic Murphy sign. The patient has normal liver function tests and white blood cell count, but impressed with the patient's reported pain, the emergency physician orders a formal right upper quadrant ultrasound, concluding that he has perhaps missed an impacted common bile duct stone. The ultrasound reveals portal vein thrombosis (**figure 4.1**), and CT scan confirms the finding (**figure 4.2**), with no additional findings to suggest mesenteric ischemia.

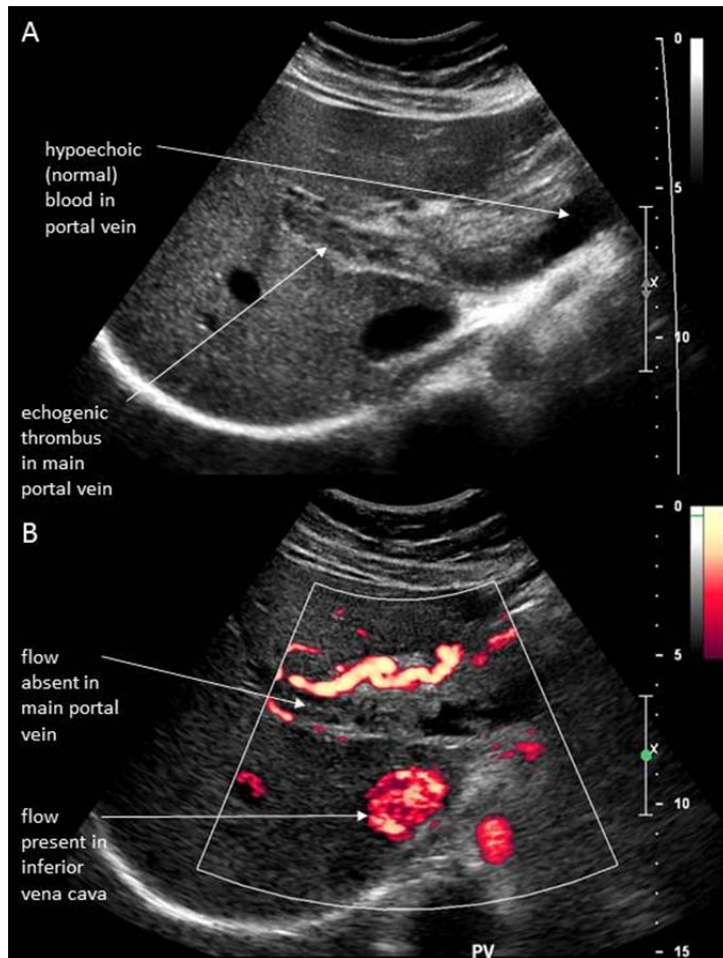


Figure 4.1. Portal vein thrombosis, ultrasound

22 year old female, 3 months post-partum, presenting with 2 weeks of right upper quadrant pain. Ultrasound shows portal vein thrombosis.

A, ultrasound gray-scale image showing thrombus in the main portal vein.

B, Doppler ultrasound shows no flow within the portal vein.

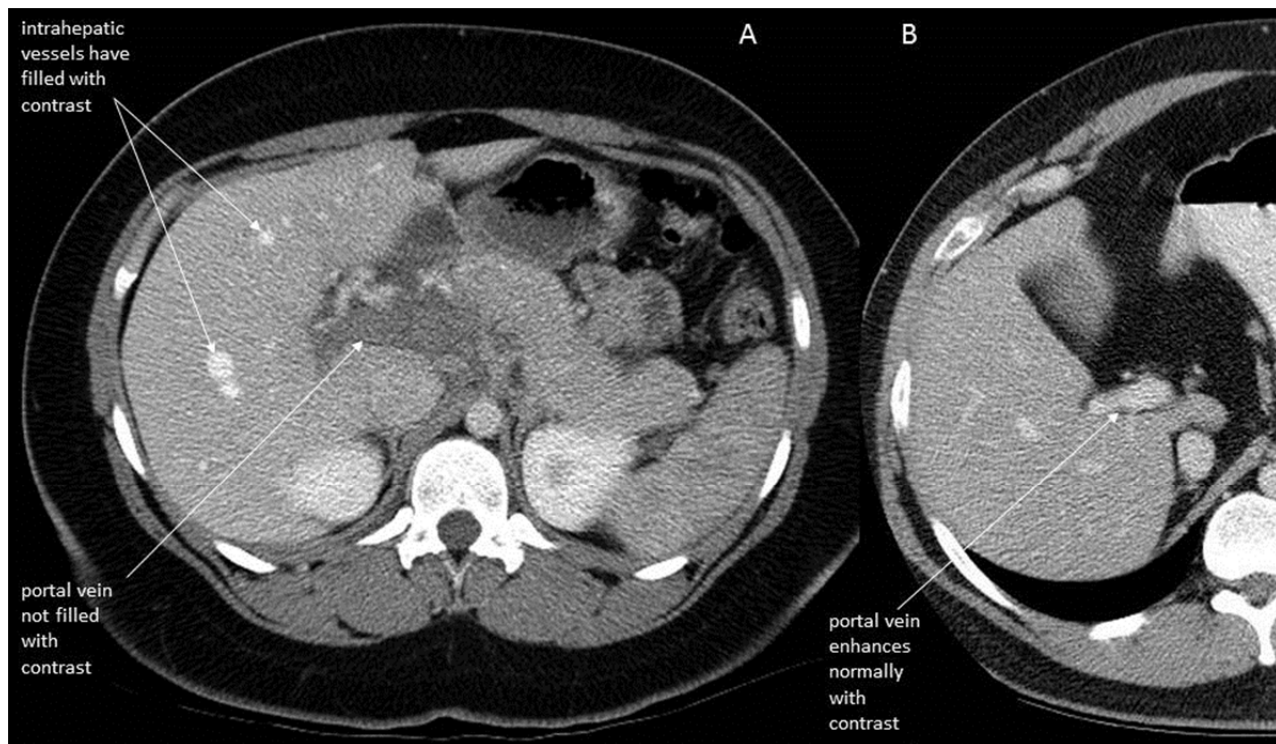


Figure 4.2. Portal vein thrombosis, CT with IV contrast

A, same patient as prior figure, with thrombosis of the portal vein. Note how other vessels including the aorta and intrahepatic vessels have enhanced with IV contrast. The portal vein remains dark in appearance as it has not filled with contrast, due to lack of blood flow. Compare with ultrasound in the prior figure. In order to confirm that portal vein thrombosis is present, IV contrast must be used, and delayed images must be obtained approximately 1 minute after contrast injection. Images acquired earlier could show lack of enhancement of the portal vein even when the vein is patent, if contrast has not yet reached the portal vein, falsely simulating thrombosis. B, CT image from another patient shows normal contrast filling of the portal vein, ruling out thrombosis.

A lactate is normal. The patient is admitted for anticoagulation. On further questioning, she reveals a maternal history of deep venous thrombosis, and her own use of depo-provera for contraception. The patient is discharged on Coumadin and enoxaparin therapy and does well at 3 month follow-up. Follow-up ultrasound 6 months later continues to show portal venous thrombosis. A hypercoagulability workup by a hematologist is unrevealing.

Discussion

Portal vein thrombosis can be considered a form of "really deep" deep venous thrombosis, and risk factors for this condition are similar to those for DVT. Cancer, advanced age, and local inflammatory factors within the abdomen such as pancreatitis or mass lesions can contribute to portal vein thrombosis. Hypercoagulable states, including as long as 2 months post-partum state, have been described as risk factors. Portal vein thrombosis can lead to high portal venous system pressures and the development of ascites and esophageal varices, familiar to emergency physicians in the setting of cirrhosis with portal hypertension.⁵⁴ In fact, portal vein thrombosis is the second-leading cause of portal hypertension following cirrhosis in western countries.⁵⁵ Portal vein thrombosis can lead to mesenteric ischemia and infarction when extensive.⁵⁶ Treatment of portal vein thrombosis consists of systemic anticoagulation, but the optimal duration of therapy is unknown. Anticoagulation for 3-6 months is

recommended by some authors, while life-long anticoagulation may be required in patients with persistent prothrombotic conditions.⁵⁷ Thrombolytic therapy or TIPS can be performed in patients who fail to recanalize despite anticoagulation.⁵⁶

The mortality of acute portal vein thrombosis has decreased from 30% historically to around 10% with anticoagulation therapy. At 5 years, survival is around 85%, though it appears that the underlying disease state that led to portal venous thrombosis is more often the cause of mortality.⁵⁶ The outcomes for patients with isolated portal venous thrombosis are less well-studied. Plessier *et al* (2009) prospectively followed 102 patients with acute portal vein thrombosis unrelated to cirrhosis or malignancy. 21% had a local (intra-abdominal) risk factor, and 52% had a prothrombotic condition. Recanalization of the portal vein occurred in 39% of anticoagulated patients compared with only 13% of controls – after a median of 234 days (almost 8 months) of therapy. Patients with ascites or splenic vein thrombosis were less likely to achieve portal vein patency. On a positive note, the feared complications of portal vein thrombosis, gastrointestinal bleeding and intestinal infarction, occurred in only 9 and 2%, respectively. In this study, no deaths were attributed to portal vein thrombosis.⁵⁸

Diagnosis of portal vein thrombosis

Ultrasound can directly visualize echogenic thrombus in the main portal vein, which can be seen on routine transabdominal ultrasound without Doppler flow assessment.⁵⁹ Emergency physicians may miss this diagnosis due to their focus on findings of gallbladder disease or abdominal free fluid. An absence of Doppler flow confirms the finding. Contrast-enhanced ultrasound may have higher sensitivity than Doppler ultrasound, though not routinely used in the United States.⁵⁹ Although some studies suggest that contrast enhanced ultrasound is superior to CT scan for detection of portal vein thrombosis,⁶⁰ CT scan with IV contrast (as described earlier in this article) can demonstrate the extent of thrombosis and important additional findings such as bowel wall thickening, pneumatosis, and adjacent abdominal processes (eg pancreatitis or pancreatic mass). CT confirms portal vein thrombosis by demonstrating a filling defect in the portal vein lumen, as well as rim-enhancement of the vessel wall with contrast.⁶¹

Pearls and Pitfalls

- Portal venous thrombosis (PVT) can present with localized pain in the right upper quadrant or generalized abdominal pain
- PVT may be seen as an incidental or secondary finding in patients with other advanced abdominal disease such as pancreatitis or malignancy
- Hypercoagulable states including the post-partum state are risk factors
- Though elderly patients are at higher risk, young patients should be assessed for risk

Case 5

A 77 year-old male presents with 4 days of vomiting, now bilious, and abdominal pain and distension. He reports no bowel movements in 6 days. He has a history of prior abdominal surgeries including an episode of bowel obstruction. He is well-appearing with blood pressure 126/67, heart rate 84,

respiratory rate 25, oxygen saturation 95% on 2 liters O₂ per nasal cannula. His abdominal exam is firm and distended with mild diffuse tenderness. Neither rebound nor guarding is noted by the emergency physician. The patient's white blood cell count is normal (8.6k) and his anion gap is 16. His creatinine is acutely elevated to 2.0mg/dL from his baseline of 1.0. A portable abdominal x-ray is obtained but the patient suddenly vomits while in the radiology department and suffers PEA arrest, with unsuccessful attempts at resuscitation. The x-ray obtained moments before his death shows multiple air-fluid levels consistent with bowel obstruction – and mural pneumatosis consistent with bowel infarction (**figure 5.1**).

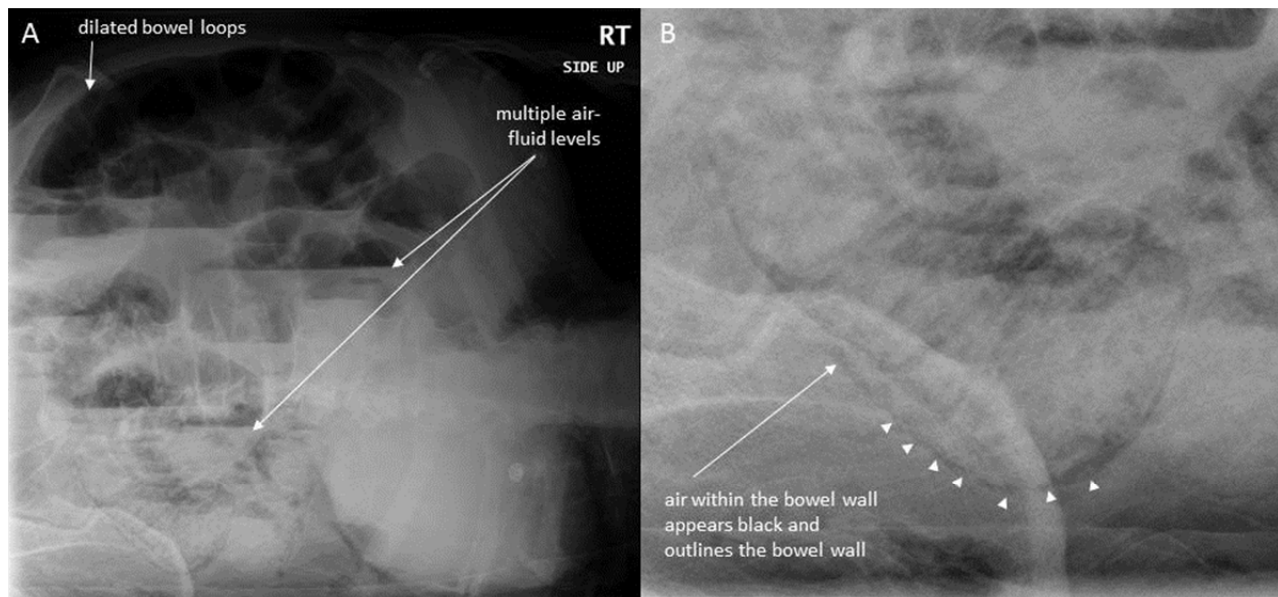


Figure 5.1 Bowel obstruction with pneumatosis

This 77 year old male presented with abdominal distension, pain, and vomiting. Bowel obstruction was suspected but the patient vomited and arrested in the radiology department. A, x-rays obtained minutes before his death show multiple air-fluid levels and dilated bowel loops consistent with small bowel obstruction. B, an enlarged view pneumatosis intestinalis. Two possible scenarios may account for this appearance: generalized bowel ischemia, leading to ileus, or mechanical bowel obstruction with a strangulated loop. Ischemia may complicate 15-20% of bowel obstructions. X-rays are rarely this diagnostic.

Discussion

Mesenteric ischemia or infarction can complicate small bowel obstruction, reportedly in as many as 14-20% of cases. One explanation is a so-called "closed loop obstruction," in which a loop of bowel torse or becomes incarcerated at its base, restricting blood flow.^{62, 63} Findings on CT include fluid-filled distended small bowel loops, an abrupt transition to collapsed small bowel distal to the point of obstruction, and a "U"-shaped distended bowel loop.⁶²

X-ray findings

Pneumatosis is likely a late finding in mesenteric ischemia, due to bowel infarction and the presence of gas-forming anaerobic organisms within the bowel wall. Although it may rarely occur in less

ominous conditions, the presence of pneumatosis on x-ray should prompt immediate surgical consultation. Pneumatosis is rarely seen on x-ray, and arguably x-rays should rarely be obtained due to poor sensitivity and specificity, as discussed earlier in this article. However, if x-rays are obtained and show pneumatosis, additional imaging such as CT may not be necessary and may delay operative therapy. If pneumatosis is a late finding, why did this patient have a normal or only slightly elevated anion gap and white blood cell count? Did the patient really die from ischemic bowel, or was aspiration the cause of death? No autopsy was performed, and we can never know based on this case. Nonetheless, it reiterates the variable presentation of bowel ischemia and the need for a high degree of suspicion, even in the face of normal laboratory testing.

CT for bowel ischemia complicating small bowel obstruction

CT with IV contrast but without oral contrast has been described by multiple authors for detection of small bowel ischemia associated with bowel obstruction. Frager *et al* (1996) found the sensitivity of CT for SBO-related ischemia to be 100% but with only 61% specificity.⁶⁴ Ha *et al* (1997) found that poor bowel wall enhancement, presence of a serrated beak, large ascites, unusual course of mesenteric vessels, and diffuse engorgement of mesenteric vessels detected 85% of strangulated obstructions from adhesions, hernia, or volvulus.⁶⁵ Balthazar *et al* (1997) found CT to be 83% sensitive and 93% specific for ischemia in small bowel obstruction.⁶⁶ Zalcman *et al* (2000) reported CT to be 96% sensitive and 93% specific, with several signs having diagnostic significance, including reduced bowel wall enhancement, mural thickening, mesenteric fluid, congestion of mesenteric veins, and ascites.⁶⁷ Mallo *et al* (2005) reviewed 11 studies (including those described above) totaling 743 patients for CT in assessing SBO-related bowel ischemia and found an aggregate sensitivity of 83% with a specificity of 92%.⁶⁸ However, Sheedy *et al* (2006) found that prospective interpretation of CT by radiologists was only 14.8% sensitive, though 94.1% specific, for ischemia. Decreased segmental enhancement of bowel was a specific finding associated with bowel ischemia. The small bowel feces sign (the presence of gas bubbles mixed with particulate matter in dilated small bowel segments) was also strongly associated with the presence of small bowel ischemia from SBO.^{69, 70}

Pearls and Pitfalls

- Pneumatosis is a rare but potentially dire sign of advanced bowel infarction and should prompt rapid surgical consultation
- Bowel ischemia and infarction can complicate bowel obstruction
- CT with IV contrast can demonstrate ischemic changes in the setting of small bowel obstruction, though the test may be more specific than sensitive. Bowel ischemia may be present despite an absence of CT evidence.

Case 6

A 39 year-old female presents from rheumatology clinic with acute right and left lower abdominal pain. She is being treated with high-dose steroids (prednisone 60mg daily and methylprednisolone 32 mg orally twice daily) for severe rheumatological disease including dermatomyositis and systemic lupus erythematosus. She has a history of pulmonary embolism, deep venous thrombosis, and inferior vena

cava filter placement and is under treatment with enoxaparin and Coumadin. On ED arrival, the patient's vital signs show blood pressure 129/72, heart rate 113, respiratory rate 22, temperature 36.0C, SaO2 97 % on room air. Her abdominal exam shows tenderness which the emergency department physicians ascribe to abdominal wall skin lesions, consistent with the patient's prior panniculitis. X-rays of the abdomen are obtained to evaluate for pneumoperitoneum from suspected perforated ulcer, given her steroid therapy (**figure 6.1**). Instead, they reveal pneumatosis of small and large bowel. The radiologist states that "benign" pneumatosis is sometimes seen with steroid use. Lactate is elevated at 3.1mmol/L (upper limit 1.6) with a venous pH of 7.27. White blood cell count is normal (9.6k). An abdominal CT is performed to evaluate further. CT reveals severe pneumatosis intestinalis within the ascending colon and proximal half of the transverse colon but without bowel wall thickening, portal venous gas, free intraperitoneal air, or adjacent mesenteric stranding (**figure 6.2**). No large arterial occlusion is seen. The patient is admitted for serial exams and is treated with IV fluids, bowel rest, total parenteral nutrition, and antibiotics. Her steroids are discontinued and she improves with these treatments and does not undergo surgery. She is discharged 35 days later.



Figure 6.2 Pneumatosis, steroid-induced

Same patient as prior figure. Bone windows have been selected for this image, as these highlight air by making all other tissues brighter. On standard abdominal windows, fat appears quite dark and may make detection of small amounts of air difficult. Bone and lung windows both make air more evident. So much mural gas is present in the hepatic flexure of the colon that the wall is quite indistinct. Compare with normal colon in the left upper quadrant.

Discussion

Pneumatosis intestinalis does rarely occur in the absence of mesenteric ischemia. Steroid use has been implicated, though the precise mechanism is not understood. An increase in mucosal permeability to gas has been suggested.⁷¹ Other causes including Salmonella infection have been described.⁷² From the emergency physician standpoint, because pneumatosis can be an indication of frank bowel infarction, immediate surgical consultation should be obtained when pneumatosis is recognized on x-ray or CT. A benign etiology of pneumatosis should never be assumed until thorough evaluation for ischemia has been performed. Some patients with this condition can be managed nonsurgically.^{73, 74}

As the name implies, pneumatosis cystoides intestinalis presents with discrete cystic gas bubbles on x-ray or CT, without other CT findings of ischemia (described earlier in this article).⁷⁵ Originally described based on x-ray findings, the implication that cystic morphology of gas implies a harmless cause may be incorrect. Olson *et al* (2009) examined features of pneumatosis in children which might predict a serious or benign cause.⁷⁶ A cystic appearance of gas, as opposed to a linear pattern, did not discriminate benign from serious causes. Associated small bowel wall thickening, free peritoneal fluid, and peri-intestinal soft-tissue stranding were concerning findings linked with serious causes. Ironically, a more extensive distribution of gas was associated with a benign cause.⁷⁶

Pearls and Pitfalls

- Pneumatosis intestinalis can result from causes other than mesenteric ischemia, but ischemia should be assumed until proven otherwise.

Summary

Mesenteric ischemia encompasses a family of related conditions including acute or chronic thrombosis or embolic occlusion of major mesenteric arteries, portal vein thrombosis, ischemic colitis due to systemic hypoperfusion, and bowel ischemia in the setting of physical bowel obstruction. Just as emergency physicians have come to recognize atypical presentations of myocardial ischemia, we must broaden our paradigm for mesenteric ischemia to include less "textbook" presentations. When the diagnosis is suspected, a very rapid evaluation should be completed to ensure the best possible patient outcome. The diagnosis should be suspected despite normal vital signs and laboratory values. Leukocytosis is common with mesenteric ischemia and should be recognized as consistent with ischemia, although other causes including infection should be considered. Delays for unnecessary diagnostic testing should be minimized by eliminating x-rays and oral contrast for CT. IV contrast for CT should be administered whenever possible to allow recognition of vascular filling defects. Communication with the radiologist about the diagnostic concern is essential to allow a tailored CT protocol with special attention to mesenteric vessels and secondary findings of ischemia by the radiologist. Surgical consultation should occur early in the course, and when the diagnosis is confirmed, aggressive surgical therapy should be considered, as bowel infarction can be fatal or debilitating.

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Table 2. Some common biases and errors in studies of diagnostic tests		
Bias/error	Description	Effect
Spectrum bias	The severity of disease in the study population is not representative of that in a real population. A form of selection bias.	The test performance will likely be different in a real population – leading to poor external validity
Selection bias	A non-representative sample is chosen for the study. Many different forms of selection bias occur, but the result is a study population which differs from an expected real population in one or more ways.	The test performance will likely be different in a real population – leading to poor external validity
Verification (confirmation) bias	Not all subjects undergo the same degree of verification of results with a definitive test. For example, only patients with a positive or a negative test may undergo definitive testing.	Sensitivity and specificity calculations may be wrong, as these require an objective determination of the presence or absence of disease, which was not performed in all cases
Inadequate statistical power	The study is not large enough to determine the performance characteristics of the test with precision. The reported point estimates of sensitivity and specificity may wildly misestimate the real test performance, unless 95% confidence intervals are considered.	The sensitivity and specificity of the test may be much worse (or better) than reported.

(Back to Article)

Table 3. Levels of evidence, American College of Cardiology/American Heart Association Practice Guidelines²⁸⁻³⁰
A: Data derived from multiple randomized clinical trials or meta-analyses
B: Data derived from a single randomized trial or nonrandomized studies
C: Only consensus opinion of experts, case studies, or standard-of-care

(Back to Article)

We'll start with just a few questions and definitions before delving into cases. First, what evidence do we need to determine the common presentation of mesenteric ischemia? Second, what kind of evidence would we want to determine the accuracy of a diagnostic test for mesenteric ischemia? Third, what form of evidence would we desire to determine the effect of a treatment? The answer to all these questions is that the optimal research study to provide reliable evidence is unavailable in the case of mesenteric ischemia. A study to determine the common presentation of mesenteric ischemia should involve prospective data collection and a large patient population, with and without mesenteric ischemia proven by a definitive test in all patients. Given the rarity of mesenteric ischemia, such a study will likely never be performed. Instead, we must rely on retrospective case-control studies in which the clinical presentation of patients with mesenteric ischemia is compared with that of patients without ischemia. Unfortunately, several biases and inaccuracies likely come into play in such a study. For example, it is possible that some patients with mesenteric ischemia present in a manner quite unlike the classic textbook description, and these patients may never be diagnosed. As a consequence, the cases of mesenteric ischemia identified for the study would likely be skewed in their presentation toward the familiar medical school description, even if this reflects a minority of actual presentations. Such a study might falsely reinforce our preconceived notions about the presentation of mesenteric ischemia. Moreover, we would be forced to rely upon retrospective chart review to identify the presenting features, including vital signs, patient age, past medical history, history of present illness, and physical exam. While some features such as vital signs and age might be reliably determined from the chart, the history and physical exam would rely on the whim of the documenting physician. Perhaps the chart was documented after the final diagnosis was made, and thus describes the history and examination and biased by knowledge of the final outcome. If the chart does not document that the patient has "pain out of proportion to exam," does this mean that such pain was absent, or that the physician simply neglected to document it?

With regard to the accuracy of diagnostic tests for mesenteric ischemia, the optimal study design would involve prospective assessment of all patients using the experimental test in question, and comparison of that test result with a gold standard (also called a criterion standard) such as surgical findings, pathology reports, autopsy, or 30 day clinical follow-up. Instead, most studies are retrospective, and do not uniformly work up all patients regardless of the results of the experimental test. This **workup bias** (also called **verification bias**) likely leads to an over-estimation of the sensitivity of diagnostic tests such as CT scan. For example, if patients with negative CT scans for mesenteric

ischemia are not evaluated further and are lost to follow-up (eg, dying without definitive diagnosis), false negative CT scans may not be recognized. If patients with positive CT scans for mesenteric ischemia are not confirmed by some definitive test (eg surgical pathology or autopsy), false positive CT scans might not be recognized. This might occur in an elderly patient wrongly diagnosed by CT with mesenteric ischemia and dying without surgery or port-mortem exam. Unfortunately, large trials meeting a strict methodological standard do not exist.

Considering the optimal treatment of mesenteric ischemia, a reliable design requires a prospective randomized controlled trial, directly comparing surgical therapy, angiographically-directed therapy such as thrombolysis or stenting, and systemic medical therapies such as intravenous heparin or thrombolytics. But again due to the rarity of mesenteric ischemia, such trials do not exist. As an example, a PubMed search using the terms "acute mesenteric ischemia treatment" and the limits "randomized controlled trial" finds no correct matches, only a retrospective study in 26 patients with no control group.¹¹

(Back to Article)

CT timing. Although CT protocols vary from institution to institution, a generic abdominal CT protocol typically times the acquisition of images to coincide with a late arterial phase of enhancement. This means that the aorta and its branches still show some contrast enhancement, though not as much as would be seen with images acquired sooner after contrast administration. End-organs such as the liver, spleen, and kidneys show significant enhancement with this timing. Structures that take longer to enhance, including the portal venous system, are not yet enhanced with contrast on images acquired with this timing. CT scan performed specifically for mesenteric ischemia usually uses two separate image acquisitions: an aortic or early arterial phase, designed to maximize contrast in the aorta and its major mesenteric branches, and a portal venous phase acquisition, designed to capture contrast (and vascular filling defects) within the portal venous system. Only a single IV contrast dose is administered; the initial acquisition visualizes the bolus in the aorta and its branches, and the delayed images capture that contrast bolus as it arrives in the portal venous system. In one published protocol, the delay for arterial phase images was 25 seconds, and 60-70 seconds for portal venous phase images.²³

Slice thickness. Modern CT scanners can acquire and display image data using a variety of slice thicknesses. The acquired slice thickness may be as low as 0.625mm with commonly available commercial CT scanners (see manufacturer data, RT Image website³³). Because image slices this thin would result in a large number of viewable images on the resulting CT scan (for example, a 60cm abdomen imaged contiguously at 0.625mm slice thickness would result in approximately 96 slices), the original acquired data is usually reconstructed at a somewhat thicker slice thickness for review by the radiologist. The reconstructed slice thickness is selected based on the expected pathology, with 3 or 5mm slices being typical values for abdominal pathology such as appendicitis.³⁴ Thick slices (10mm) have been shown to have limited diagnostic accuracy for appendicitis and competing diagnoses.³⁵ In the case of mesenteric ischemia, where small abnormalities such as a focal stenosis or occlusion of an aortic branch might suggest the diagnosis, thin slice acquisition and reconstruction are beneficial. For more generic abdominal CT protocols, some institutions routinely acquire the original data set at a relatively thick slice thickness (eg 3mm), limiting the reconstructed slice thickness to this thickness or greater. Informing the radiologist of the concern for mesenteric ischemia allows the original slice acquisition and the reconstructed slice thickness to be optimized for detection of mesenteric ischemia.

In a published protocol specifically for evaluation of mesenteric ischemia, the collimation (slice thickness) was 1.25mm for the arterial phase images and 5mm for the portal venous phase images. The authors reported a sensitivity of 96% and a specificity of 94% for bowel ischemia using these parameters.²³ A second published protocol used 2.5mm slice thickness for both arterial and portal venous phases, with a reported sensitivity of 92% and specificity of 100%. The reported sensitivity and specificity must be taken with some skepticism from these small studies, which included a total of only 122 patients.²⁴

Multiplanar reconstructions. Historically, abdominal CT images have been viewed using axial slice reconstructions only. Modern CT scanners can acquire a three-dimensional volume of original image data, and the reconstructed slices can be viewed in axial, coronal, sagittal, or other planes, depending on the pathology of interest. Curved planar reconstructions can be created to track tortuous blood vessels, which may not lie in a single cardinal anatomic plane. Specialized slices called Maximum

Intensity Projection (MIP) images can be created to bring more anatomic territory into view in a single image (see figure 1.3). These reconstructions may not be routinely performed for standard CT protocols but can be performed on demand by the radiologist or radiology technician if a diagnosis for which these reconstructions might be beneficial (such as mesenteric ischemia) is suspected. These reconstructions require a high quality original image data set with thin slice acquisition, so the radiologist must be alerted of the suspected diagnosis before the CT scan is performed.

(Back to Article)