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Beyond decreased bowel enhancement: acute abnormalities of the mesenteric and portal vasculature

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Abstract

Acute mesenteric ischemia (AMI) is a potentially life-threatening condition with an associated high mortality. Prompt diagnosis is crucial to achieve a favorable outcome. The radiologist plays a central role in the initial evaluation of a patient with suspected AMI. In this pictorial essay, we review the appropriate imaging evaluation of a patient with suspected AMI, and we review both the common and uncommon etiologies of mesenteric ischemia. With each etiology presented, relevant clinical and imaging findings, as well as potential treatments, are reviewed.

Key words: Mesenteric ischemia—Occlusive mesenteric ischemia—Non-occlusive mesenteric ischemia—Porto-mesenteric venous thrombosis

Acute mesenteric ischemia (AMI) is a potentially life-threatening condition accounting for approximately 1% of acute abdomen hospitalizations and associated with a high mortality rate ranging from 31% to 93% [1–4]. Diagnostic delay contributes to poor patient outcome [1, 2, 5]. Because clinical and laboratory findings, including leukocytosis, elevated lactate, and metabolic acidosis, are nonspecific and insensitive [6], the radiologist plays a crucial role in the initial evaluation of mesenteric ischemia.

The radiologist must recognize both common and uncommon etiologies of AMI. In this pictorial essay, imaging findings on multidetector-row computed tomography (MDCT) and angiography of typical and unusual acute pathologies affecting blood flow in the mesenteric and portal vasculature will be reviewed. This includes occlusive pathologies of the mesenteric arteries, non-occlusive mesenteric ischemia (NOMI), and porto-mesenteric venous thrombosis. The discussion will include clinical findings, imaging appearance, acute complications, and typical management of each entity. The primary imaging findings and potentially differentiating features are summarized in Table 1.

Normal mesenteric vascular anatomy

The superior mesenteric artery (SMA) arises obliquely from the anterior aorta at approximately the L1 level. Its branches supply the distal duodenum, entire jejunum and ileum, and the ascending and transverse colon (Fig. 1). Most commonly, the first branch is the inferior pancreaticoduodenal artery, followed by the middle colic artery. Beyond this level, multiple jejunal and ileal branches arise from the SMA trunk and anastomose via the mesenteric arcades. These arcades give rise to the vasa recta—end arteries supplying the bowel wall. The SMA terminates in the ileocolic artery, supplying the terminal ileum, the cecum and ascending colon, and the appendix.

In addition to the arcades within the SMA distribution, anastomoses between the celiac, SMA, and inferior mesenteric artery (IMA) distributions provide additional collateral flow in most individuals. The inferior pancreaticoduodenal artery from the SMA is continuous with the superior pancreaticoduodenal artery from the celiac axis (the pancreaticoduodenal arcade). The marginal artery of Drummond courses along the mesenteric border of the entire length of the colon and represents an anastomosis around the level of the splenic flexure between the left branch of the middle colic artery (arising from the SMA, as discussed) and the ascending branch of the left colic artery (arising from the IMA). More centrally situated, the arc of Riolan (aka the ''meandering mesenteric artery'') usually accompanies SMA or IMA

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Table 1. Overview of causes of acute mesenteric ischemia

Source	Vascular location	Classic features
Embolic occlusion	3–8 cm distal to SMA origin	Very acute clinical course Risk factors for cardiac source of embolus
Thrombotic occlusion	SMA ostium or within 2–3 cm	Atherosclerotic plaques and calcifications, multivessel stenotic disease, collateral vessels
Secondary mesenteric dissection	Always arises from the aortic lumen, variable length of arterial involvement	Continuous with type A or B aortic dissection
Spontaneous mesenteric dissection	Anterior wall of proximal SMA curve	May be isolated without antecedent trauma or traditional risk factors for dissection Look for aneurysms or other sites of dissection, consider segmental arterial mediolysis
Strangulated bowel obstruction	Intramural vessels	Signs of ischemia include noncontrast bowel wall hyperdensity and decreased bowel wall enhancement Ascites and mesenteric stranding
Volvulus and closed-loop obstruction	Point of convergence/volvulus Veins obstructed before arteries in most cases	Whirl sign, venous cut-off sign U-shaped segment of dilated bowel bracketed by two transition points, triangular cross section of bowel at site of obstruction, upstream bowel distension
Vasculitis or collagen vascular disorders	Depends on type of vasculopathy (large, medium, or small vessel)	Vascular wall thickening and enhancement, perivascular inflammation, stenosis/occlusion without classic features of atherosclerosis, aneurysms
Mesenteric trauma	Variable from proximal to distal arteries and/or veins	Mesenteric hematoma = conservative if only finding Triangular interloop fluid, free fluid in absence of solid organ injury, active mesenteric vascular extravasation, beaded irregularity or abrupt termination of mesenteric vessels = surgical indications Bowel wall thickening and altered mucosal enhancement may be due to direct bowel injury or devascularization/ischemia Flat IVC + diffuse bowel hyperenhancement and mural thickening favors shock bowel
Non-occlusive mesenteric ischemia	No vascular occlusion despite signs of bowel ischemia/infarct	Irregular SMA narrowing (string of sausages), spasm of visceral arcades, and poor mural enhancement in critically ill patients
Porto-mesenteric venous thrombosis	Venous thrombosis, variable involvement of portal vein, SMV, splenic vein, and/or small peripheral venous arcades	Expanded, occluded mesenteric vein(s), hyperdense venous clot Bowel wall thickening and indistinctness, mesenteric infiltration, and ascites from venous congestion Thrombophlebitis more likely if ring enhancement of venous wall, gas within clot, hepatic abscesses Consider intraabdominal inflammatory process as cause of thrombosis/thrombophlebitis
Ischemic colitis	May see IMA atherosclerosis, although often non-occlusive	Segmental (often watershed) or pan-colonic pattern of ischemia, variable appearance by CT

stenosis or occlusion and connects the proximal middle colic artery of the SMA with the proximal left colic artery of the IMA [7]. Incomplete regression of the primitive fetal blood supply accounts for additional variations in the mesenteric vascular supply [8].

The mesenteric venous system is less variable. Venous blood from the jejunum, ileum, and proximal colon flows into the superior mesenteric vein (SMV), which travels vertically to join the splenic vein, forming the portal vein. Blood in the IMA distribution from the midtransverse colon to the rectum flows into the inferior mesenteric vein (IMV), which may drain into the splenic vein or SMV. Additional drainage from the stomach and right colon often forms a common gastrocolic trunk before joining the SMV [8].

Stages of intestinal ischemia

Three stages of intestinal ischemia have been described [9]. The first stage is limited to the mucosa. Its earliest manifestation is increased mucosal permeability to albumin and other macromolecules (including intravenous contrast material), which leak into the bowel wall and lumen and may cause fluid distension of the bowel [10]. This is followed by mucosal and subepithelial edema and epithelial sloughing [10]. Mucosal necrosis, erosions, and ulcerations may develop but will eventually heal completely if the ischemia is reversed. This stage is therefore called *reversible ischemic enteritis*. On CT, bowel will appear thick walled, possibly with dilation or spasm. Edema will cause the bowel wall to be low in

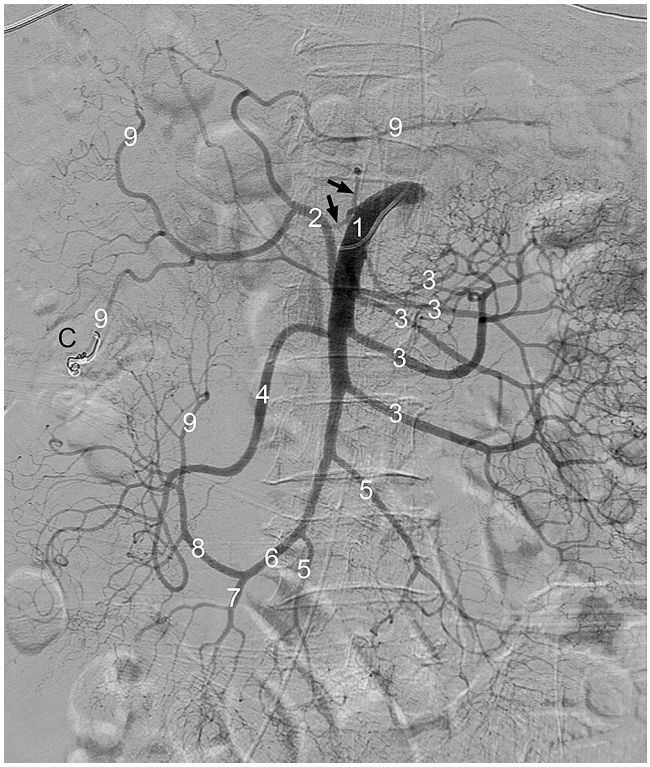

Fig. 1. A 78-year-old man with lower gastrointestinal bleeding. Normal branching anatomy of the SMA is shown on digital subtraction angiography with selective injection of the SMA. Labeled structures include the SMA (*1*), inferior pancreaticoduodenal arcade (*arrows*), middle colic artery (*2*), jejunal branches (*3*), right colic artery (*4*), ileal branches (*5*), ileocolic artery (*6*), with branches to the terminal ileum (*7*), and cecum (*8*). Portions of the marginal artery (*9*) can be seen arising from the ileocolic and middle colic arteries. Active bleeding was treated with coil placement (*C*) of a fourth order branch of the middle colic artery.

Table 2. Specific and nonspecific MDCT findings in AMI [3, 4, 11, 13–15]

Specific findings on CTA or MDCT venous phase for mesenteric ischemia
Pneumatosis intestinalis[a]
Venous gas[a]
Free intraperitoneal or retroperitoneal gas[a]
SMA occlusion
Celiac and IMA occlusion with distal SMA disease
Arterial embolism
Mesenteric whirl sign, venous cut-off sign
Bowel wall thickening[b] + focally absent bowel wall enhancement
Bowel wall thickening[b] + focally increased bowel wall enhancement
Bowel wall thickening[b] + target sign (submucossal edema + mucosal hyperenhancement)
Bowel wall thickening + solid organ infarction
Bowel wall thickening + venous thrombosis
Bowel wall thinning + focally absent bowel wall enhancement[a]
Other nonspecific findings on CTA or CT venous phase
Superior mesenteric and/or portal venous thrombosis
Stratification of bowel wall (target sign)
Bowel lumen dilation (small bowel >2.5 cm, colon >8 cm)
Transition point between dilated proximal and collapsed distal bowel
Mesenteric stranding
Ascites

[a]Late signs of ischemia suggesting bowel necrosis
[b]Thickness >3 mm of noncollapsed small bowel or colon perpendicular to transverse plane

attenuation, while hyperattenuation suggests superimposed hemorrhage. The mucosa may have decreased or increased enhancement, depending on the presence of reperfusion or outflow obstruction. These findings are nonspecific and do not correlate with the severity of ischemia. The severity of bowel wall thickening will also vary depending on whether the inciting pathology is arterial or venous [9].

The second stage progresses to necrosis of the underlying submucosal and muscular layers, and healing from this stage may create a fibrotic stricture. Dilation is more common once deeper necrosis has occurred, due to reactive cessation of peristalsis or direct involvement of the muscular layer [9].

The third and final stage is complete transmural bowel wall necrosis, which is associated with a high mortality rate without immediate surgery [10]. Findings suggestive of transmural involvement include dilated thin-walled bowel (due to involvement of the intramural musculature and nerves) and perforation [9]. Pneumatosis and portal venous gas indicate an advanced stage of infarction (also considering other causes of pneumatosis), although not necessarily transmural involvement [11]. The imaging appearance at any stage may be complicated by submucosal or intramural hemorrhage or by superinfection of the bowel wall [9].

Techniques for imaging of acute mesenteric ischemia

With increased availability and quality of MDCT, computed tomography angiography (CTA) has replaced conventional angiography in the diagnosis of AMI. American College of Radiology (ACR) Appropriateness Criteria® recommend CTA for rapid, noninvasive diagnosis of suspected AMI [12].

MDCT findings suggestive of AMI are listed in Table 2 [3, 4, 11, 13–15]. A biphasic mesenteric MDCT protocol that includes both arterial and venous phases is ideal [15, 16]. An arterial phase scan performed with 1.25–2.5 mm collimation is most helpful to detect subtle findings. However, patients with nonspecific symptoms not immediately suspicious for ischemia are more likely to undergo venous phase MDCT only—this likely accounts for the lower sensitivity and specificity of MDCT in some retrospective studies [17]. In other centers, a patient with nonspecific symptoms might undergo an initial noncontrast MDCT, followed by biphasic imaging upon discovery of abnormal small bowel loops. An unenhanced MDCT alone may otherwise be obtained in critically ill patients with acute renal failure. The sensi-

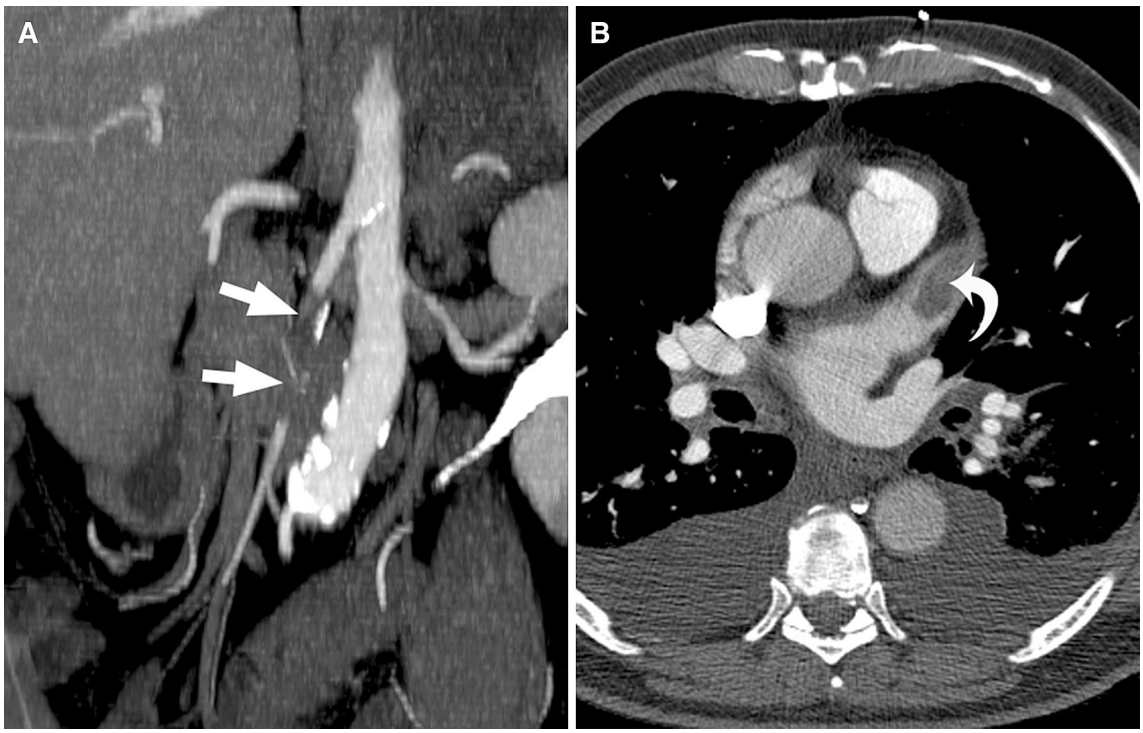

Fig. 2. A 70-year-old patient with cardioembolic SMA occlusion. Mesenteric CTA oblique maximum intensity projection (MIP) reconstruction (**A**) shows embolus (**A** *straight arrows*) occluding the proximal SMA. Axial contrast-enhanced CT image (**B**) through the chest confirms the embolic source, a clot in the left atrial appendage (**B** *curved arrow*).

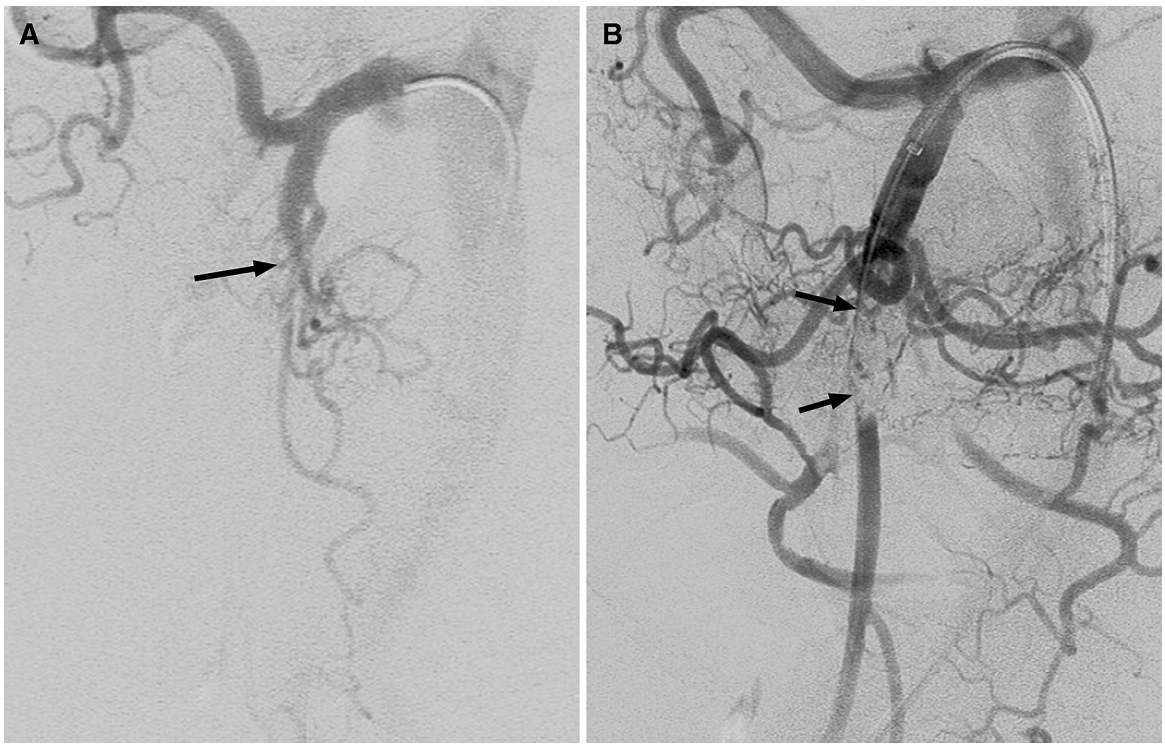

Fig. 3. A 56-year-old patient with embolic SMA occlusion. Mesenteric angiogram before (**A**) and after partial recanalization (**B**) shows embolus (**A**, **B** *arrows*) lodged in the proximal SMA distal to the origin of several jejunal branches. Note the variant, a replaced common hepatic artery originating from the SMA.

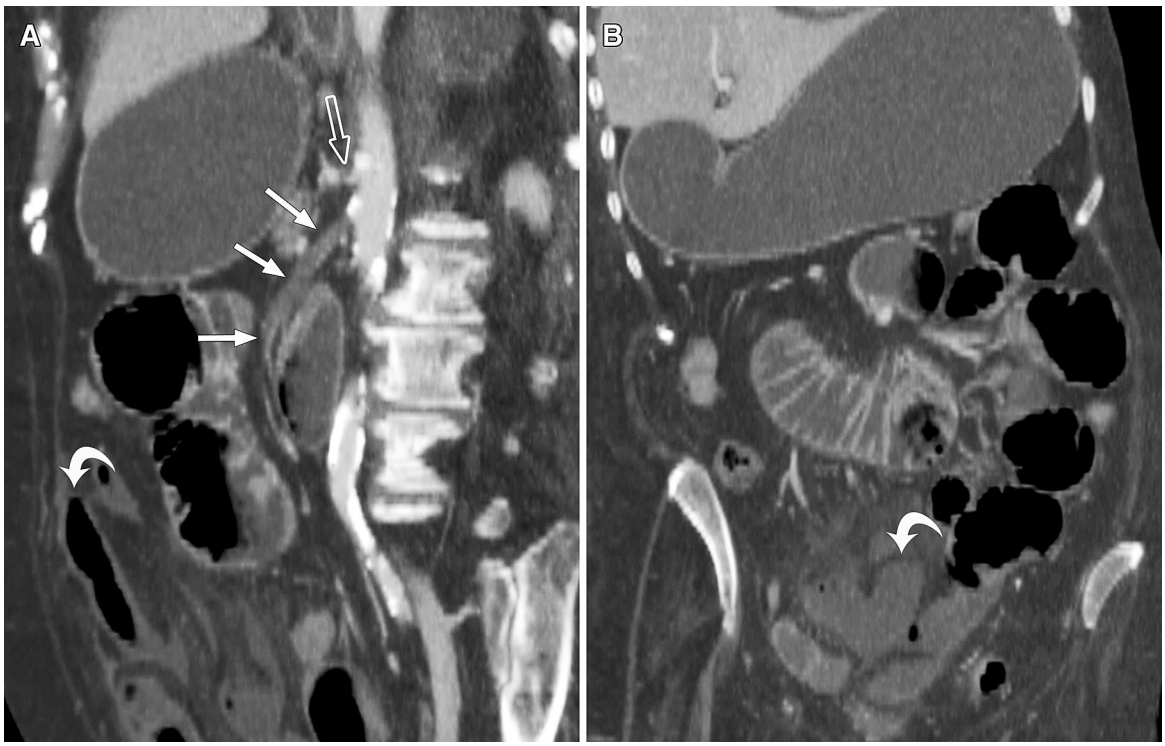

Fig. 4. A 67-year-old patient with bowel necrosis from in situ SMA thrombosis. Sagittal MPR image (**A**) demonstrates no contrast material filling the thrombosed SMA (**A** *white arrows*) and a high-grade celiac artery ostial stenosis (**A** *open arrow*). Non-enhancing necrotic ileum can be seen on both sagittal (**A** *curved arrow*) and coronal (**B** *curved arrow*) MPR images, compared with normally perfused jejunum in the mid abdomen.

tivity of MDCT is 0.66–0.96 and specificity is 0.67–0.98 [15–18]. Maximum intensity projections, multiplanar reconstructions, and volume-rendered images may also aid in the diagnosis [13]. Dual-energy MDCT may have additional advantages over conventional MDCT by increasing the conspicuity of ischemic segments [19] but is not yet widely available.

Magnetic resonance angiography (MRA) with gadolinium is another imaging alternative that does not rely on radiation; however, its utility is limited by longer duration of scanning, lower spatial resolution, and inability to visualize vascular calcium (in atherosclerotic disease). Furthermore, the risk of nephrogenic systemic fibrosis must be considered before administration of gadolinium to those with impaired renal function. MRA without a contrast agent has lower sensitivity and specificity than MRA with gadolinium and is usually not appropriate [12].

Mesenteric arterial thromboembolism

Acute embolic SMA occlusion is usually cardiogenic in origin (Fig. 2) and may coexist with infarcts in other organs from multiple emboli [20]. The MDCT appearance depends on the location of the thrombus, the degree and duration of vascular occlusion, and whether reperfusion has occurred. The embolus most commonly lodges at a branch point 3–8 cm distal to the SMA ostium (Fig. 3) [21]. This is often distal to the first jejunal branches and middle colic artery and consequently may spare the jejunum and transverse colon from ischemia [22]. A convex proximal surface of the thrombus suggests embolus rather than in situ thrombosis. All patients with peritoneal signs or threatened bowel by imaging (including dilation with thin wall, lack of enhancement, pneumatosis, porto-mesenteric gas, and free air) require surgical exploration. Otherwise, treatment varies between surgical embolectomy and catheter-directed thrombolysis or thromboaspiration depending on whether the embolus partially or completely occludes the vessel and how proximal or distal it is located [1, 23].

Mesenteric arterial thrombosis

In situ thrombosis of the SMA is usually superimposed on pre-existing atherosclerotic plaque. The imaging appearance of SMA thrombosis can be similar to that of thromboembolism. The key in differentiating thrombosis of the SMA from thromboembolism lies in the location and appearance of the occlusion. SMA thrombosis tends to superimpose on ostial atherosclerotic plaque, consequently occurring within 2–3 cm of the SMA origin and proximal to the middle colic and early jejunal arteries

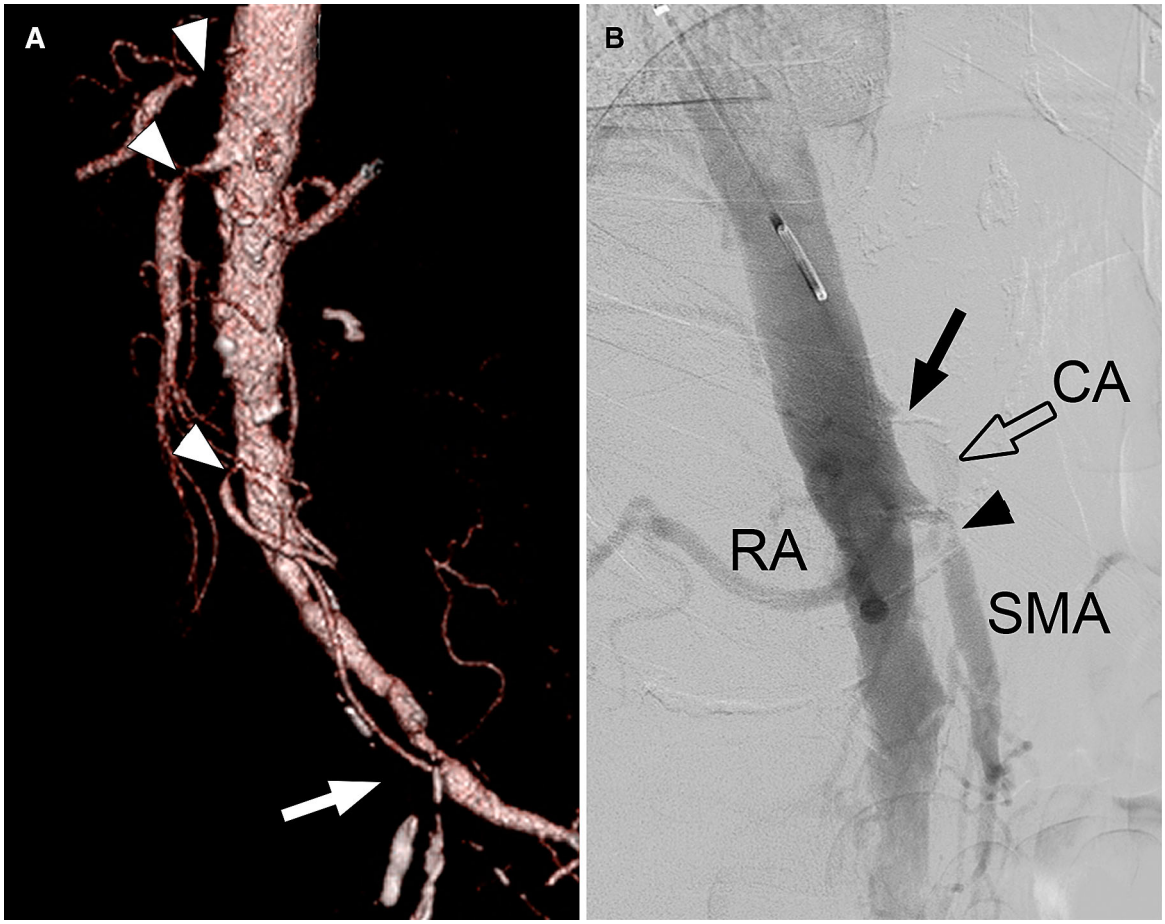

Fig. 5. A 64-year-old woman with mesenteric ischemia from multivessel stenosis. 3-Dimensional reconstruction (**A**) from abdominal CTA shows high-grade stenoses (**A** *white arrowheads*) at the origins of the celiac axis, SMA, and IMA, as well as occlusion of the right common iliac artery (**A** *white arrow*). No CT findings of bowel ischemia were detected, although pain was ascribed clinically to ischemia. Aortogram from subsequent digital subtraction angiogram (**B**) shows subtotal stenosis at proximal SMA (**B** *black arrowhead, SMA*) and critical stenosis (**B** *black arrow*) of the celiac axis origin, resulting in delayed opacification of the celiac branches (**B** *open arrow*, *CA*), compared with the SMA and right renal artery (**B** label *RA*). The celiac axis was successfully stented.

(Fig. 4). The extent of intestinal infarct from thrombosis tends to be greater because the jejunum and colon are typically not spared [20, 21].

Collateral vessels suggest a chronic thrombosis or pre-existing high-grade stenosis. Acute symptoms usually require stenotic or occlusive disease of multiple visceral arteries with sudden blockage of a critical site (Fig. 5). Treatment of SMA thrombosis is historically surgical [1] and may include thrombectomy, mesenteric bypass grafting, patch angioplasty, reimplantation, and endarterectomy [2]. Increasingly, endovascular means of reperfusion are being used for arterial occlusive disease [23, 24], usually in combination with laparotomy for direct inspection of the bowel.

Mesenteric arterial dissection

Dissection of the SMA, either isolated [25, 26] or contiguous with aortic dissection [27], is a rare cause of AMI. Mesenteric ischemia worsens the prognosis in patients with aortic dissection [28]. Isolated visceral arterial dissection, most often of the SMA, is a rare condition not associated with aortic dissection. It usually begins along the anterior wall of the SMA curve 1.5–3 cm beyond the SMA origin (Fig. 6) [25]. While most cases of spontaneous visceral dissection are described as idiopathic [25], some might be attributed to dissecting aneurysms of segmental arterial mediolysis (SAM) (discussed later) [29]. Treatment of SMA dissection is medical with rou-

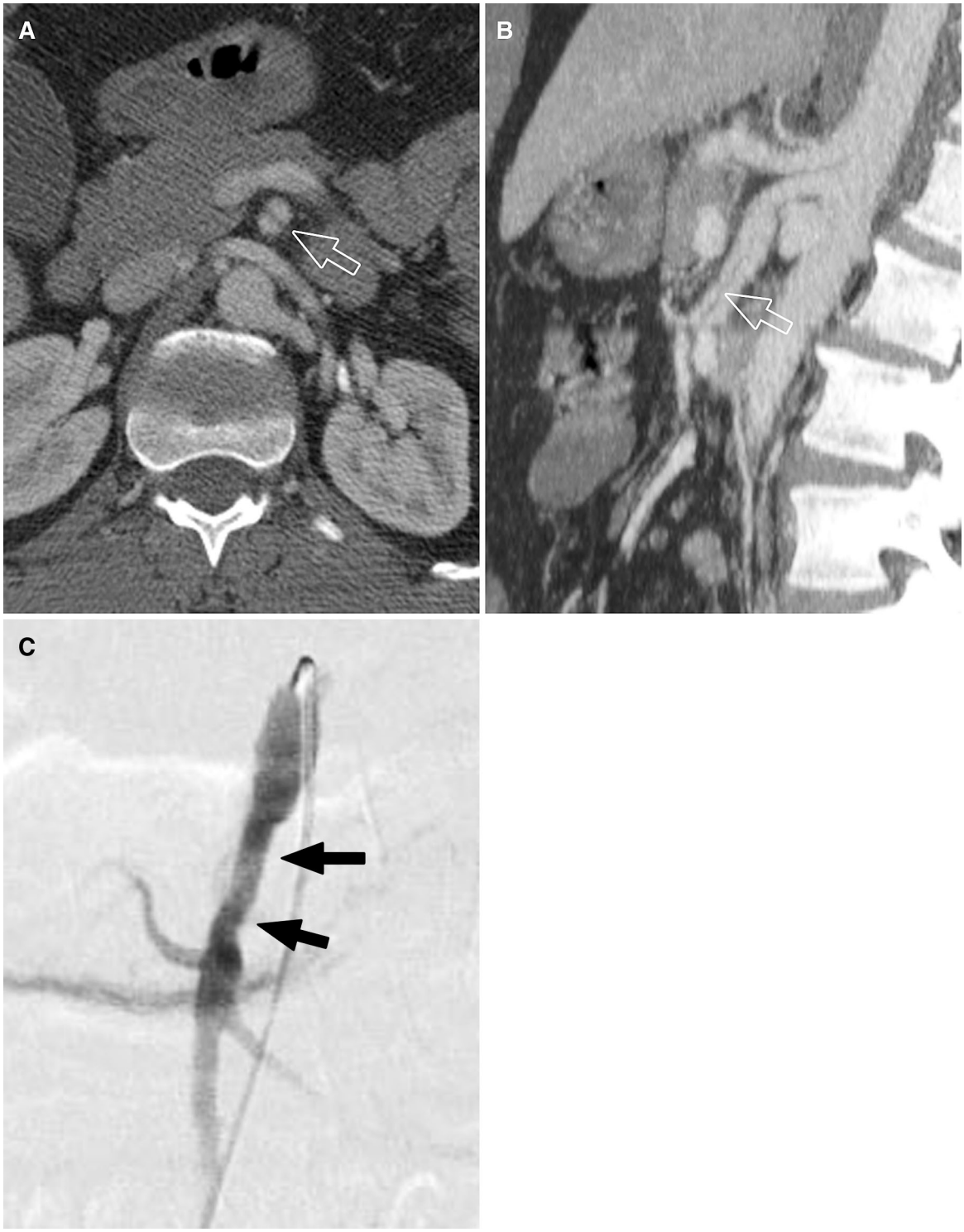

Fig. 6. A 47-year-old man diagnosed with SMA dissection after sustaining minor fall-related trauma. Axial (**A**) and sagittal (**B**) CT images demonstrate an SMA dissection flap (**A**, **B** *open arrows*) beginning proximally at the SMA curve. Early phase SMA injection during mesenteric angiogram (**C**) shows narrowing (**C** *black arrows*) of the proximal SMA from the dissection. It is unclear whether the dissection was pre-existing in this otherwise healthy patient or related to trauma.

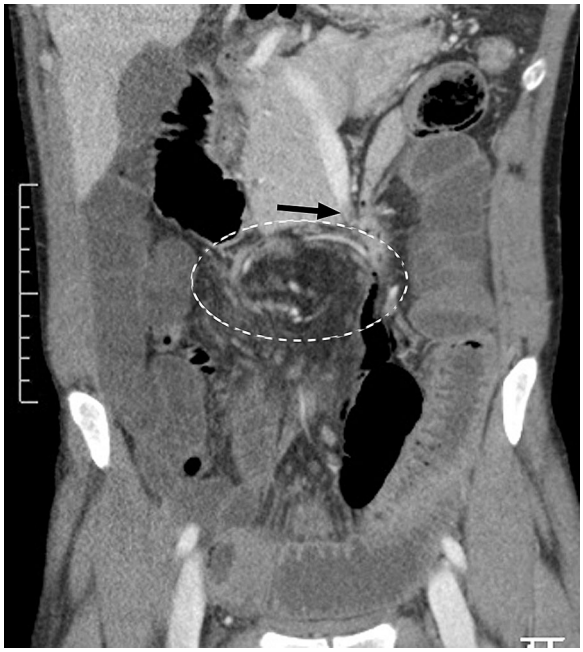

Fig. 7. A 39-year-old man with mesenteric volvulus. Coronal CT image shows "whirl sign" of the mesentery (*dashed circle*) with dilated, hypoenhancing small bowel. A subtle thrombus in the SMV (*arrow*) formed upstream of the torsed mesentery due to poor venous inflow.

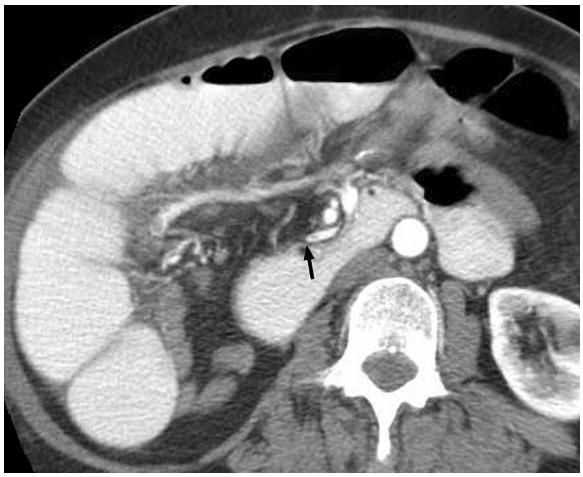

Fig. 8. A 49-year-old patient with mesenteric volvulus. The "venous cut-off" sign (*arrow*) identifies the mesenteric vein occluded by the volvulus. Note also the dilated bowel from obstruction.

tine surveillance if the patient remains asymptomatic and if any associated visceral aneurysm measures less than 2 cm in diameter [25]. Symptomatic dissection or larger aneurysmal dilation may be treated by endovascular or surgical revascularization [25].

Strangulating small bowel obstruction, mesenteric volvulus, and closed-loop obstruction

MDCT findings that are most suggestive of ischemia in the setting of mechanical small bowel obstruction (regardless of cause) include decreased bowel wall mucosal enhancement [30, 31] and increased noncontrast attenuation of the bowel wall [31]. The latter sign is due to intramural hemorrhage resulting from venous congestion. The absence of mesenteric fluid is a reassuring sign that strangulation is not present [30].

Mesenteric volvulus and closed-loop obstruction may result in secondary bowel ischemia from extrinsic compression of mesenteric arteries or veins. Volvulus can be idiopathic or secondary to post-operative adhesions, hernia, or congenital malrotation [32, 33]. Twisting of the bowel in volvulus may result in a closed-loop bowel obstruction. Closed-loop obstruction may also occur without volvulus due to adhesions or small-necked hernia. By MDCT, the twisted mesentery can be identified by swirling strands of soft tissue/vessels and mesenteric fat—the *whirl sign* (Fig. 7) [33]. The whirl sign is appreciated best perpendicular to the axis of rotation of the volvulus and may be most easily recognized on coronal or sagittal images and MIP reconstructions. Obstructed patients with the whirl sign are 25 times more likely to need an operation than those without the sign [34]. As the mesenteric twist tightens, the mesenteric venous supply, followed by the mesenteric arterial supply, becomes compressed, resulting in ischemia. If unrelieved, infarct of the affected bowel loops can ensue [35]. The *venous cut-off sign* reflects occlusion of the SMV at the point of torsion or compression (Figs. 8, 9) [36].

Other than the whirl sign and venous cut-off sign, CT signs that distinguish volvulus and strangulating obstruction from other causes of mesenteric ischemia include a U-shaped segment of fluid-dilated bowel terminated at each end by two adjacent loops of decompressed bowel, fusiform tapering and a triangular configuration of bowel in cross section at the site of obstruction, and upstream bowel dilation (Fig. 10) [32]. While a minority of cases of closed-loop obstruction may be treated conservatively, any case with bowel compromise requires urgent surgical decompression.

Mesenteric arterial vasculitis and vasculopathy

Vasculitides and collagen vascular disorders rarely cause mesenteric ischemia (Table 3) [37–45]. Presentation is usually of chronic ischemia rather than acute infarction. General MDCT signs of vasculopathy include stenosis, often over a long segment without calcification or

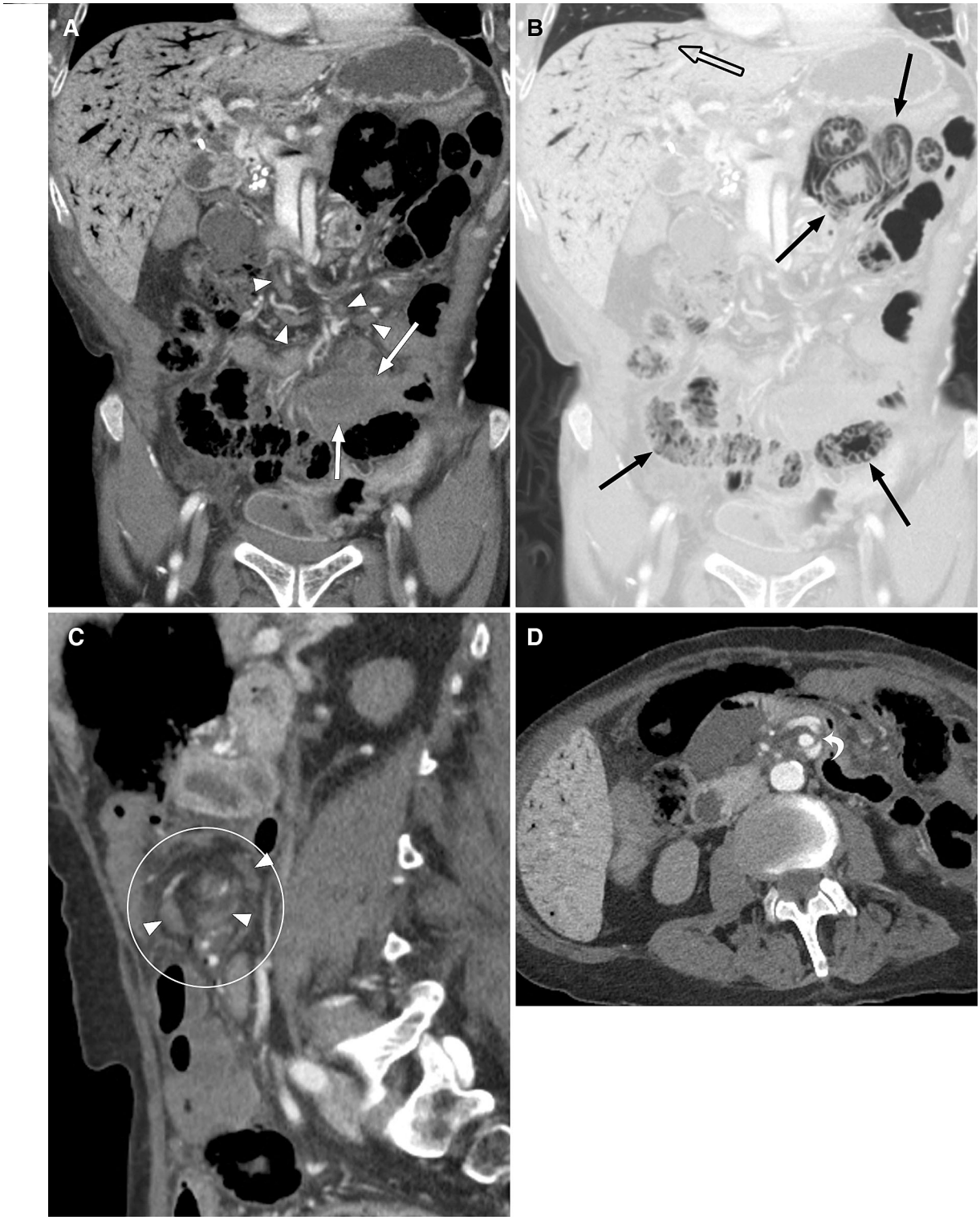

Fig. 9. A 54-year-old man with prior Roux-en-Y surgery presenting with acute mesenteric volvulus. Coronal image in soft tissue (**A**) and lung (**B**) windows shows thrombosis of multiple mesenteric arteries (**A** *arrowheads*), hypoenhancement of small bowel loops (**A** *white arrows*), extensive small bowel pneumatosis (**B** *black arrows*), and intrahepatic portal venous gas (**B** *black open arrow*). Sagittal (**C**) reconstruction showed mesenteric "whirl sign" (**C** *circle*) composed of thrombosed mesenteric arteries (**C** *arrowheads*), suggesting mesenteric volvulus as the cause. Axial image (**D**) also shows abrupt occlusion of the SMV, the "venous cut-off" sign (**D** *curved arrow*). Immediate laparotomy confirmed volvulus of the entire small bowel mesentery without internal hernia. Although 60 cm of infarcted small bowel had to be resected, the patient recovered fully.

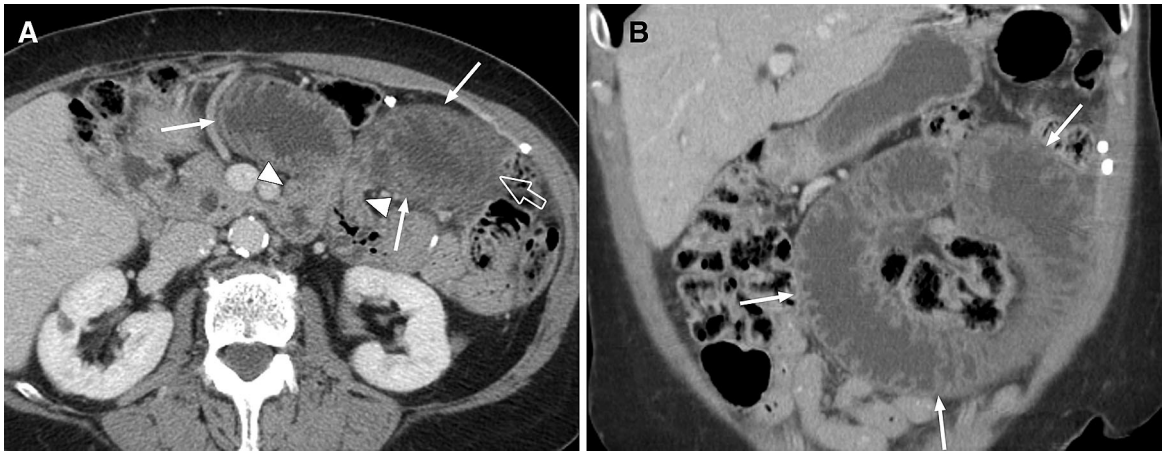

Fig. 10. A 62-year-old woman with worsening abdominal pain and history of Roux-en-Y gastric bypass surgery. Axial (**A**) and coronal (**B**) images show a dilated loop of jejunum (**A**, **B** *white arrows*). Findings suspicious for closed-loop obstruction include the proximity of the proximal and distal transition points giving the dilated bowel a conspicuous U-shape (**B**) and evidence of extrinsic constriction (**A** *white arrowheads*) at the transition points. Bowel wall is edematous with areas of poor mucosal enhancement (**A** *open arrow*), suggesting ischemia. Closed-loop obstruction secondary to internal hernia through an iatrogenic transverse mesocolon defect was confirmed at surgery.

Table 3. Vasculitides and collagen vascular disorders associated with mesenteric ischemia

Vasculitis and collagen vascular disorder	Incidence
Polyarteritis nodosum	7 cases in series of 81 patients with vasculitis of the gastrointestinal tract
Takayasu's arteritis	0.3 cases/100,000 (USA); 36% involve mesenteric artery
Giant cell arteritis	18% of patients with giant cell arteritis involve mesenteric artery
Buerger disease	26 case reports as of 2003
Systemic lupus erythmatosis	0.2–9.7% of SLE pts develop mesenteric involvement
Behçet's disease	34 cases in series of 81 patients with vasculitis of the gastrointestinal tract; less then 25% have vascular involvement
Inflammatory bowel disease-related vasculitis	Rare
Henoch–Schönlein purpura	10 cases/100,000
Dermatomyositis	Rare
Scleroderma	Rare

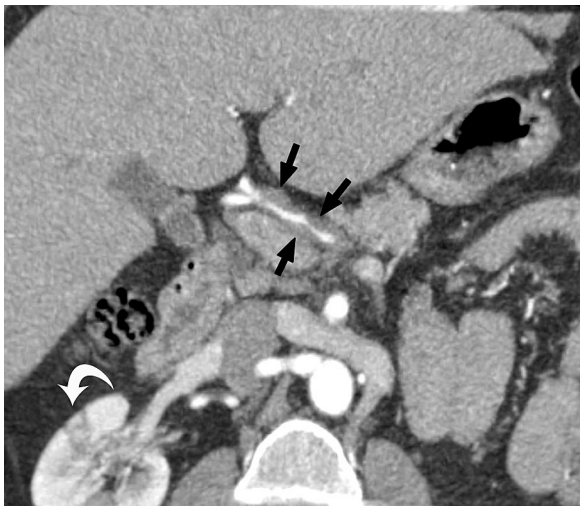

Fig. 11. A 44-year-old methamphetamine user with mesenteric vasculitis. Curved planar CT reformation shows inflammatory rind narrowing the hepatic artery (*black arrows*). Note the small kidney infarct (*curved arrow*) from renal artery involvement. The SMA was spared.

irregularity characteristic of atherosclerosis, vascular occlusion, vascular wall thickening and enhancement, perivascular inflammation, and aneurysm (Fig. 11) [39]. Surgical or endoluminal revascularization may be combined with medical treatment of the underlying inflammatory process. Unfortunately, steroids, which are frequently administered for treatment of these inflammatory disorders, may increase the risk of gastrointestinal complications and may mask peritoneal symptoms following an acute vascular event.

Noninflammatory, nonatherosclerotic vasculopathies may also affect the mesenteric arteries, including fibromuscular dysplasia (FMD) and SAM. These two distinct entities have overlapping features on imaging and histology but are differentiated by clinical factors. FMD is most common in young females and may present with occlusive disease, most often of the renal and internal carotid arteries. Mesenteric involvement in FMD is unusual.

In comparison, SAM tends to present with spontaneous hemorrhage or acute luminal occlusion in late

middle-age and elderly patients with slight male predilection [46]. Despite being a rare diagnosis, abdominal visceral involvement is the most common manifestation of SAM [29]. Renal arteries and iliac arteries may also be affected. The distinct hallmark of the diagnosis is the presence of dissecting aneurysms (Fig. 12) [29]. Cavitation within the outer portion of the arterial wall media leads to mural weakening, dissection, and pseudoaneurysm formation [29, 46]. MDCT may reveal multifocal involvement with a segmental, skip pattern, alternating stenotic and aneurysmal portions, and circumferential or partial arterial wall involvement. SAM may be under recognized and may underlie an apparently spontaneous SMA dissection [29].

Ischemia from mesenteric trauma

Traumatic SMA injuries are rare but highly lethal, resulting more often from penetrating than blunt trauma [47]. A paucity of literature on these uncommon injuries exists, but mortality has been shown to correlate with location of the SMA injury (Table 4) [47–49]. The high rate of mortality is in part related to the difficulty in obtaining control of the injured artery or valveless porto-mesenteric vein and also from compromised bowel perfusion [47, 50]. Despite technical advances in the past decade, overall mortality from SMA traumatic injuries remains high (33%, not broken down by Fullen zone) [51], compared with prior overall rate of 39% [49].

A moderate to large volume of free fluid on MDCT following abdominal trauma is one of the most sensitive findings of either bowel or mesenteric injury and could represent extraluminal bowel contents and/or extravasated blood products. Beaded irregularity or abrupt termination of mesenteric vessels, even without active bleeding, is a relatively specific finding of mesenteric injury [52]. The absence of free fluid excludes bowel or mesenteric injury [52], whereas active hemorrhage into the mesentery indicates significant mesenteric injury that requires laparotomy (Fig. 13) [53].

Bowel wall thickening and abnormal enhancement can be seen with transmural or partial thickness bowel injuries as well as with devascularization or ischemia from mesenteric vascular injury and is therefore not specific [52]. Both, however, necessitate surgical exploration for resection and/or reperfusion of compromised bowel loops. Diffuse hyperenhancement and bowel wall thickening arising from hypoperfusion (and accompanied by other imaging signs such as a flattened inferior vena cava and adrenal and renal hyperenhancement)

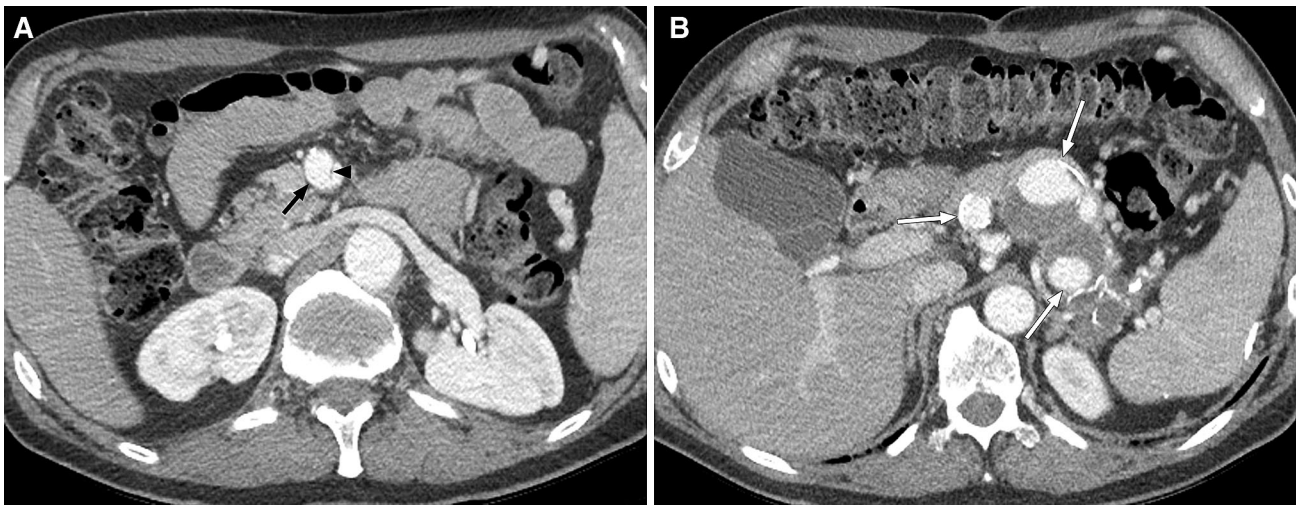

Fig. 12. A 56-year-old man with head and neck cancer. Staging CT axial images (**A**, **B**) revealed asymptomatic aneurysmal dilatation of multiple mesenteric arteries including the SMA (**A** *black arrow*) and splenic artery (**B** *white arrows*), with short segment dissection of the SMA (**A** *arrowhead*), consistent with SAM. The splenic arterial aneurysm was later embolized (not shown).

Table 4. Fullen's anatomic classification of SMA injury by zone [48] with mortality as shown in a multiinstitutional study [47]

Zone	SMA segment	Bowel segments affected	Mortality (%) [49]
I	SMA trunk proximal to first major branch (inferior pancreaticoduodenal)	Jejunum, ileum, right colon	76.5
II	SMA trunk between inferior pancreaticoduodenal and middle colic	Major segment of small bowel and/or right colon	44.1
III	SMA trunk distal to middle colic	Minor segment of small bowel or right colon	27.5
IV	Segmental jejunal, ileal or colic branches	No ischemic bowel	23.1

should be differentiated from mesenteric or bowel wall injuries, which tend to be more focal [53].

Non-occlusive mesenteric ischemia

NOMI is disproportionate mesenteric vasoconstriction that can progress to bowel infarction. NOMI accounts for approximately 16% of cases of mesenteric ischemia [20]. It is most commonly implicated in the critically ill patient with a low-flow state from a variety of causes. Splanchnic vasoconstriction to maintain perfusion pressure is the normal physiologic response to a reduction in blood pressure [10], but this autoregulation eventually fails when blood pressure falls below a certain threshold [54]. Septic shock, hypovolemic or hemorrhagic shock, cardiogenic shock, vasoconstrictor administration, and vasoactive drugs (digoxin and ergotamine) have all been reported as causes of NOMI [55, 56]. The high mortality rate of NOMI is attributed to the conundrum of further mesenteric circulation compromise resulting from vasoactive drugs needed to support systemic circulation. Intra-arterial infusion of papaverine is the treatment of choice for reversing the vasoconstriction, combined with surgery when peritoneal signs are present [1].

Some of the imaging signs of NOMI classically described for digital subtraction angiography (Table 5) [57, 58] may be seen on CTA [59]. A retrospective study of the diagnosis of NOMI by MDCT (compared with surgically confirmed mesenteric ischemia) in patients after cardiac surgery found MDCT had high sensitivity but low specificity [60]. MDCT signs are primarily those of bowel ischemia (i.e., wall thickening, luminal dilation, pneumatosis, intravenous gas, free air, and ascites) without vessel cut-off to indicate an occlusive etiology (Fig. 14) [60]. In a small series of patients with NOMI, CTA demonstrated findings classically seen on angiog-

Table 5. Classic angiographic signs of NOMI [57, 58]

Narrowing and vasoconstriction of multiple SMA branches
"String of sausages" appearance (alternating dilatation and narrowing of SMA)
Spasm of mesenteric arches
Slowed flow with delayed filling of peripheral vessels and mesenteric veins
Impaired filling or occlusion of intramural vessels
Reflux of contrast into abdominal aorta
Splayed segmental arteries due to bowel distention

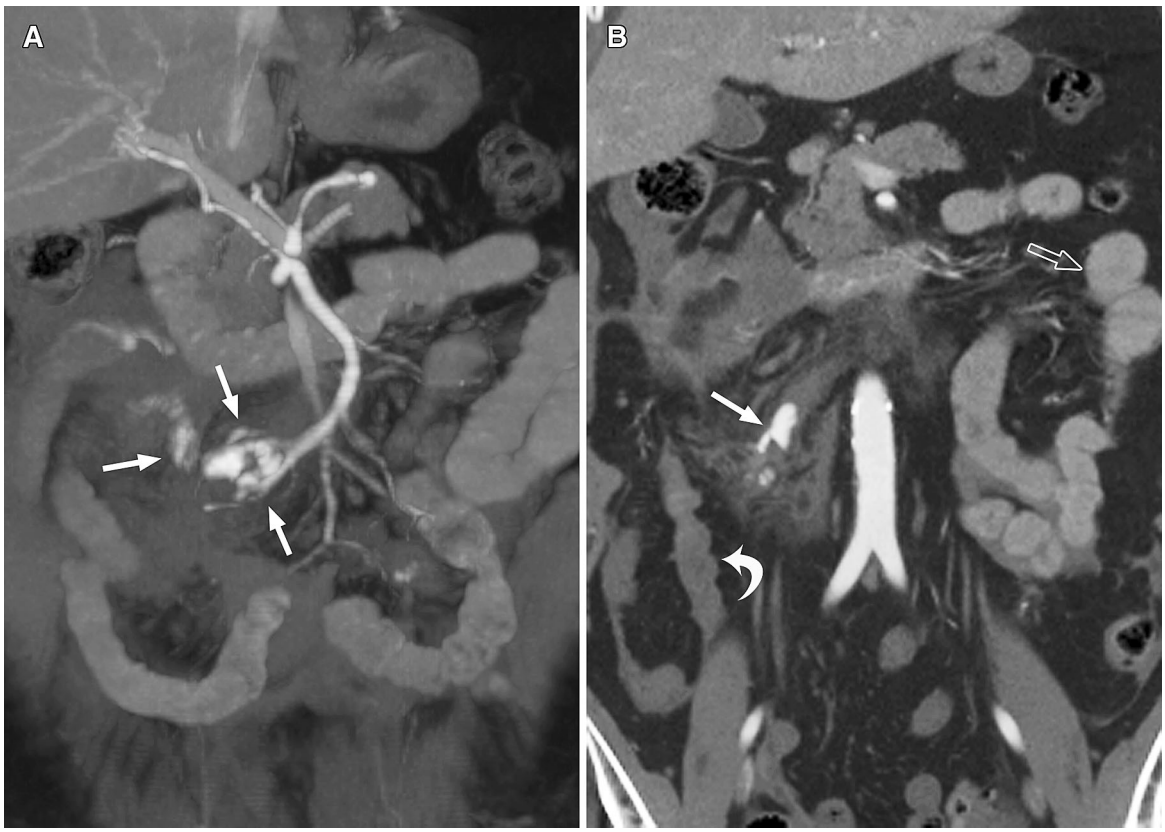

Fig. 13. A 48-year-old with traumatic transection of the SMA resulting from motor vehicle crash. Coronal MIP (**A**) from contrast-enhanced abdominopelvic CT shows transection of the SMA with active extravasation (**A** *straight arrows*). Coronal MPR image (**B**) demonstrates extravasation (**B** *straight arrow*) with devitalized ileum in the right lower quadrant (**B** *curved arrow*). Compare to hyperemic shock bowel in the left upper quadrant (**B** *open arrow*).

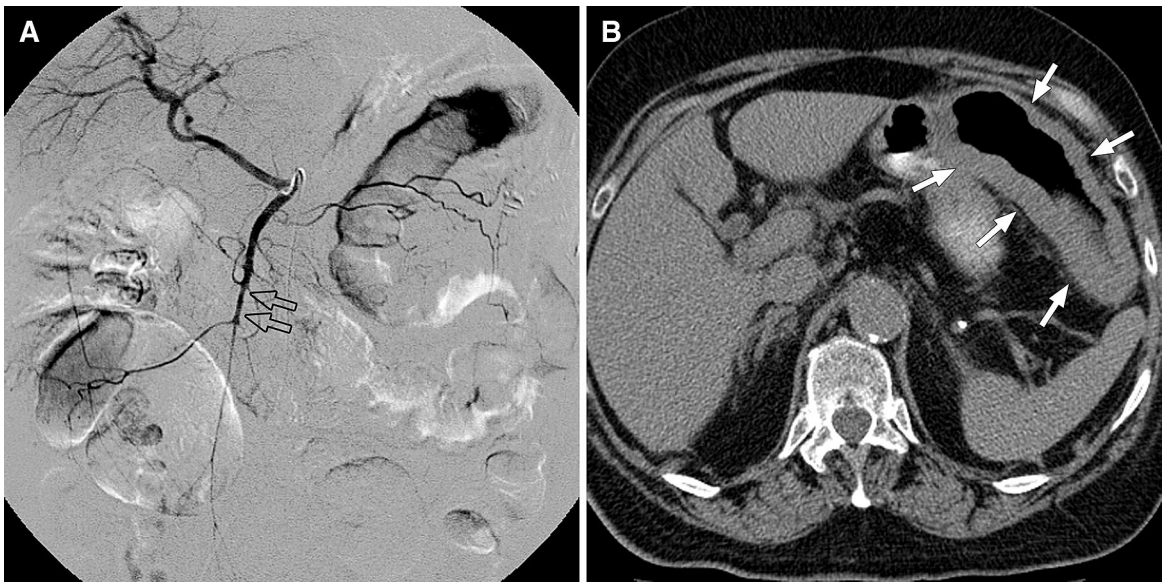

Fig. 14. A 70-year-old woman in the intensive care unit with NOMI. Digital subtraction angiogram (**A**) of the SMA shows diffuse vasospasm and subtle "string of sausages" appearance (**A** *open arrows*). Incidental variant of replaced hepatic artery. Subsequent noncontrast axial CT with positive oral contrast (**B**) demonstrates thickening of transverse colon and splenic flexure (**B** *straight arrows*) worrisome for ischemia. At exploratory laparotomy, the entire bowel was mildly ischemic but not overtly infarcted.

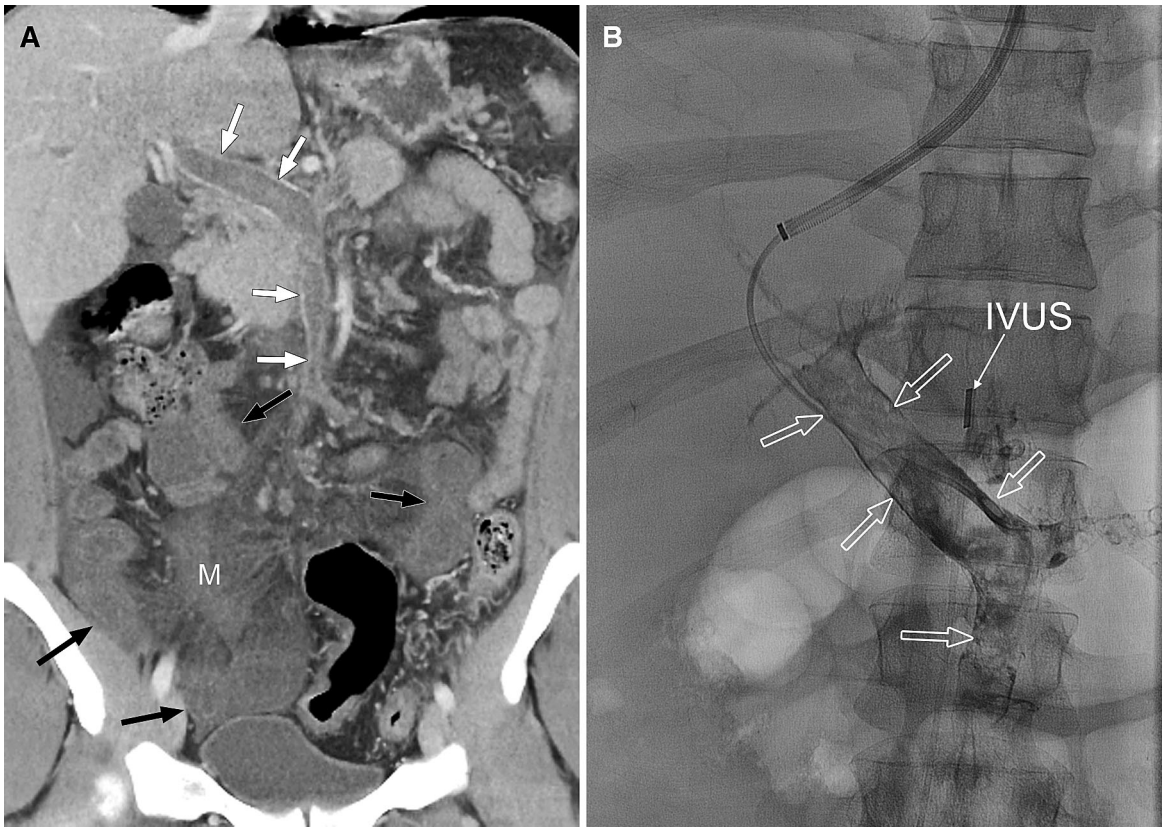

Fig. 15. A 34-year-old man with extensive porto-mesenteric venous thrombosis. Coronal CT MIP shows mesenteric vein thrombus extending into the portal vein (**A** *white straight arrows*). Note the edematous, hypoenhancing ischemic bowel (**A** *black straight arrows*) and mesenteric infiltration (**A** label *M*) from venous congestion. Corresponding image from transhepatic mesenteric thrombolysis (**B**) shows injected contrast material outlining thrombus cast (**B** *open arrows*) within the portal and superior mesenteric veins. Intravenous ultrasound (**B** label *IVUS*) device can be seen.

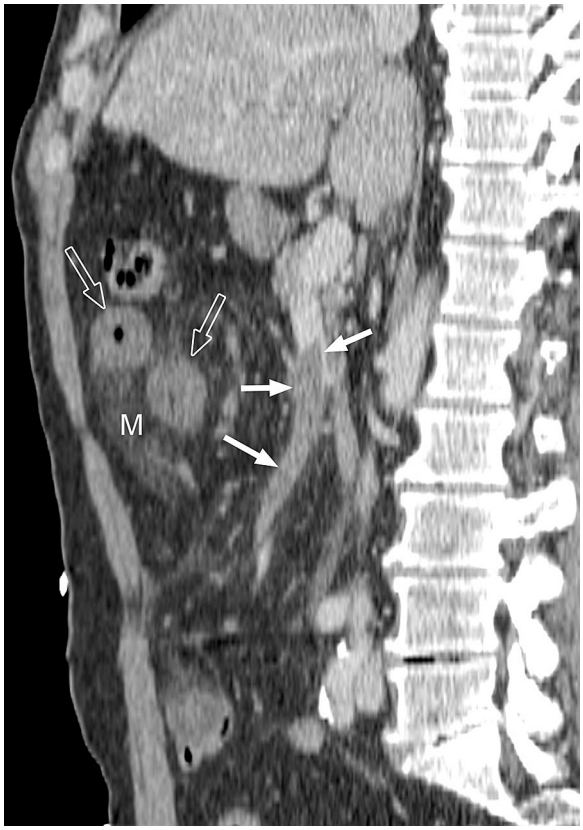

Fig. 16. A 62-year-old man with ischemia from SMV thrombosis. Sagittal image demonstrates occlusive thrombus within the SMV (*white arrows*) with hypoenhancing bowel (*open arrows*) in the anterior abdomen and associated venous congestion within the mesentery (label *M*). The patient had 70 cm of necrotic bowel resected at surgery.

raphy including irregular narrowing of the SMA, spasm of visceral arcades, and poor opacification of intramural vessels [59]. The diameter of the SMA was also significantly narrower (mean of 3.4 ± 1.1 mm) compared with normal controls (6.0 ± 1.5 mm) [59]. Although these findings show promise, they require validation in a larger number of patients—in general, NOMI remains a diagnosis of exclusion in at-risk patients undergoing MDCT.

Porto-mesenteric vein thrombosis

Porto-mesenteric venous thrombosis accounts for approximately one-sixth of cases of AMI [20], although the presentation is more often subacute than with arterial occlusions. The degree to which intestinal perfusion is affected depends on the location, extent, and speed of thrombus formation. The SMV, splenic vein, and portal vein are frequently involved, whereas IMV thrombosis is rare [61]. Thrombosis of small peripheral veins is more likely to cause ischemia than isolated portal vein or SMV thrombosis, which is more frequently subclinical and presents with complications of portal hypertension from chronic venous thrombosis [61]. Conditions predisposing to mesenteric venous thrombosis include inherited and acquired hypercoagulable states (including thrombophilia, primary or metastatic malignancy, and oral contraceptive use), direct venous injury (such as from pancreatitis, abdominal trauma, or iatrogenic), and local venous stasis or congestion [20]. Other associations include extramesenteric venous thromboembolic disease (concurrent or prior) and morbid obesity [20]. Still, 10–49% of cases are idiopathic [61, 62].

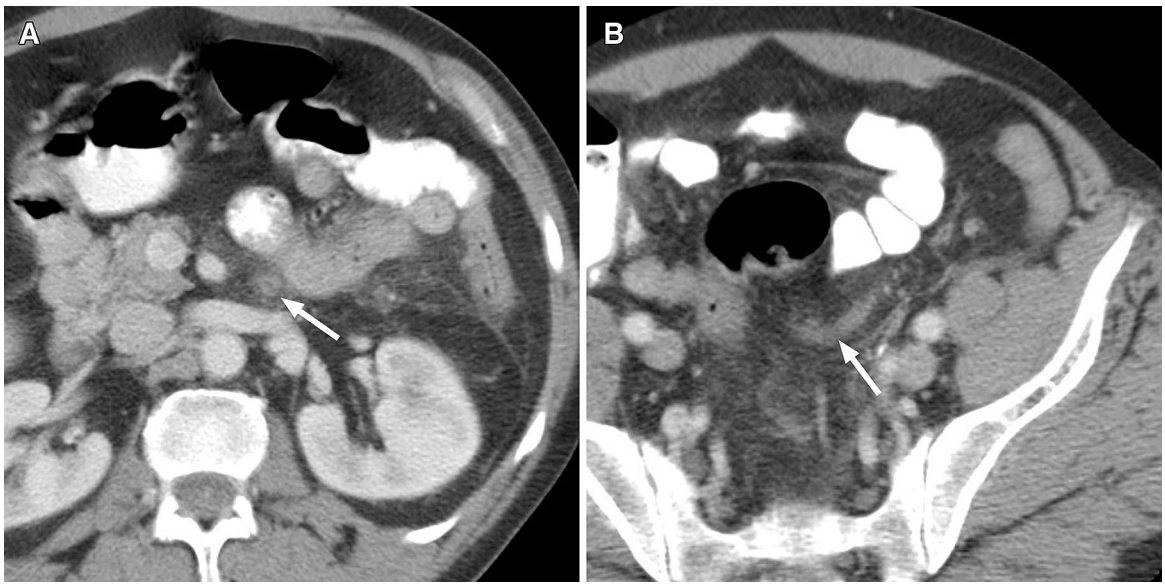

Fig. 17. A 66-year-old man with IMV thrombosis. Axial CT images (**A**, **B**) show complete IMV thrombosis to its junction with the unaffected splenic vein (**A**, **B** *white arrows*). Since the sigmoid was also mildly thickened and inflamed (not shown), it remained clinically uncertain whether sigmoid diverticulitis or primary IMV thrombosis was the original causative etiology.

The venous occlusion is identified as a tubular hypodensity along the expected course of a mesenteric vein (Fig. 15). The vein lumen is often expanded. MDCT findings of ischemia from mesenteric venous thrombosis will frequently include wall thickening and luminal distension, thickened and hazy mesentery, ill-defined bowel wall margins, and ascites due to venous congestion (Fig. 16) [61]. Hyperdensity of venous segments can be seen with venous thrombosis on noncontrast MDCT, with greater sensitivity and diagnostic confidence when narrow windows are used [63].

If the patient demonstrates peritoneal signs on exam, laparotomy should be performed and infarcted bowel resected. Goals of treatment otherwise include prevention of new or worsening intestinal ischemia with immediate heparinization and possibly thrombectomy [64] or transcatheter thrombolytics [64, 65] (although thrombolytics should be avoided if intestinal infarction is present). Also, long-term anticoagulation is initiated if any underlying hypercoagulable condition is diagnosed.

Mesenteric venous thrombosis or thrombophlebitis may rarely complicate acute inflammatory conditions of the bowel, including appendicitis and diverticulitis (Fig. 17) [66, 67]. In such cases, it may be difficult to differentiate thrombosis-related bowel ischemia from thrombus-inducing bowel inflammation, both of which may have bowel wall thickening, adjacent fat stranding, and mucosal hyperenhancement. Thrombophlebitis of the portal system (also known as pylephlebitis) is differentiated from bland thrombosis by ring enhancement of the vein wall, gas in or adjacent to the thrombus, and intrahepatic seeding and abscess formation [67].

Conclusion

AMI is a rare but potentially life-threatening condition in which the radiologist plays a key diagnostic role. CTA and venous phase abdominal MDCT can often differentiate the various etiologies of AMI and appropriately triage the patient to surgical or endovascular therapies, if indicated. Embolic obstruction of the SMA is the most common cause of AMI and should prompt evaluation for a cardiogenic source. In situ arterial thrombosis typically complicates severe atherosclerotic ostial disease, and symptomatic disease is usually accompanied by critical stenosis of celiac and/or IMA. NOMI is common in critically ill patients and should be considered when MDCT findings of ischemia are detected in this at-risk population. Some of the classic angiographic findings may be detectable on CTA. Dissection, volvulus, mesenteric vascular trauma, and vasculitis are uncommon causes of AMI that are each treated differently and that can often be diagnosed confidently by MDCT. Porto-mesenteric venous thrombosis may occur in hypercoagulable patients or as a complication of abdominal inflammatory processes.

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