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REVIEW

Mesenteric ischemia: a radiologic perspective

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Abstract
Mesenteric ischemia is a broad term encompassing several clinical conditions leading to impaired vascularity of bowel loops. Absence of specific clinical presentation and a definitive laboratory marker often lead to delayed diagnosis with high morbidity and mortality in the acute setting. Imaging plays a crucial role in the diagnosis and management. Multi-detector CT (MDCT) is the first line imaging modality for the evaluation of patients with suspected mesenteric ischemia and plays an important role for assessing its severity and complications. This review article highlights the causes, pathophysiology, imaging features and possible endovascular treatment options of mesenteric ischemia.

Keywords Mesenteric ischemia · Thrombosis · Embolism · Superior mesenteric artery · Superior mesenteric vein · Infarction · MDCT

Introduction

Mesenteric ischemia is an uncommon condition resulting from inadequate arterial supply in the splanchnic circulation or deficient venous return, leading to bowel ischemia. Acute mesenteric ischemia (AMI) is a potentially life threatening condition. Clinical presentation is non-specific and may mimic other causes of abdominal pain. Currently, no specific laboratory test is available which can accurately detect acute mesenteric ischemia. Owing to these factors, clinical diagnosis is challenging. Delayed diagnosis leads to considerable morbidity and mortality. The mortality rate in AMI has consistently been reported to be as high as 50–69% [1–3]. This review article highlights the causes, pathophysiology, imaging features and possible endovascular treatment options of mesenteric ischemia.

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The splanchnic circulation

The splanchnic arterial circulation consists of the three main ventral branches of the abdominal aorta—the coeliac axis (CA), the superior mesenteric (SMA) and the inferior mesenteric arteries (IMA) (Fig. 1a, b). The coeliac axis divides into the common hepatic, the left gastric and the splenic artery. The common hepatic in turn gives rise to the hepatic artery proper and the gastroduodenal artery (Fig. 1a). The left gastric artery supplies the lesser curvature of the stomach. The gastroduodenal artery gives rise to the right gastric artery (which supplies the greater curvature of the stomach) and the superior pancreaticoduodenal artery. The superior pancreaticoduodenal artery forms an arterial arcade with the inferior pancreaticoduodenal artery, a branch arising from the superior mesenteric artery and supplies the duodenum along with the head of pancreas (Fig. 1a).

The superior mesenteric artery arises about a centimeter below the coeliac axis and is the major contributor to the splanchnic bed (Fig. 1a, b). It supplies the entire small intestine through jejunal and ileal branches (Fig. 1a). It also supplies the cecum, the ascending colon and the entire transverse colon and splenic flexure through ileocolic, right and middle colic arteries. The SMA occasionally have a common

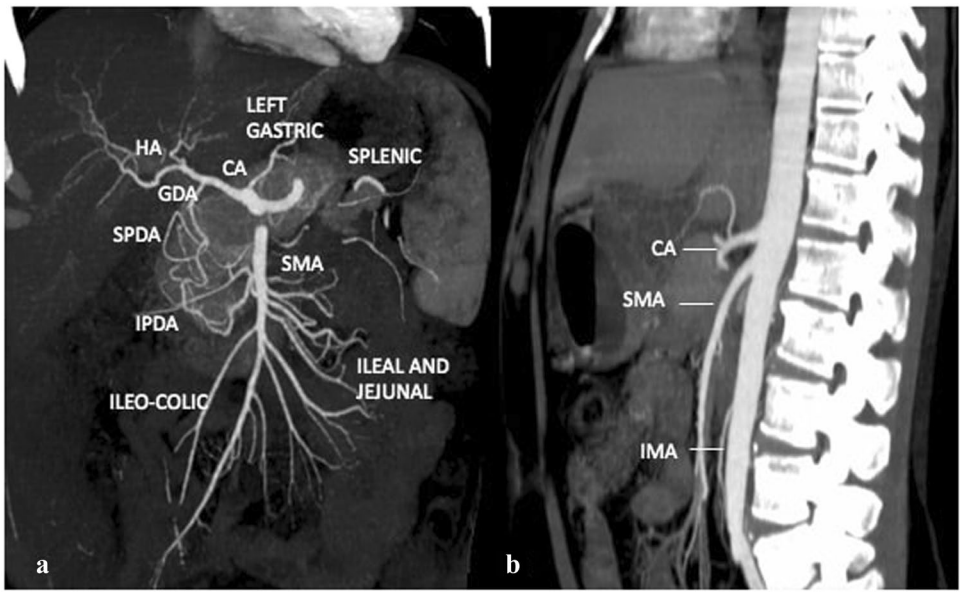

Fig. 1 MDCT angiography of abdomen with **a** Coronal and **b** sagittal MIP images showing major splanchnic arteries and their branches. *CA* coeliac axis, *SMA* superior mesenteric artery, *IMA* inferior mesenteric artery, *HA* hepatic artery, *GDA* gastroduodenal artery, *SPDA* superior pancreaticoduodenal artery, *IPDA* inferior pancreaticoduodenal artery

trunk with the celiac axis or gives rise to a replaced (often right) hepatic artery [4, 5].

Inferior mesenteric artery is the smallest ventral branch arising from the abdominal aorta (Fig. 1b), above the aortic bifurcation and gives rise to the left colic artery and sigmoidal branches and terminates as the superior rectal artery. These branches supply the descending and sigmoid colon and rectum. There is a watershed zone of perfusion in the distal colon created by the overlapping vascular territory of the proximal IMA and the distal SMA. This zone is particularly sensitive for ischemia and infarction [4, 5].

The superior and inferior mesenteric veins receive blood from the small and large gut following similar drainage territory as their arterial counterparts. The superior mesenteric vein and splenic vein form portosplenic confluence and drain into the portal vein (Fig. 2). The inferior mesenteric vein drainage is variable; it drains into the splenic or superior mesenteric vein or their confluence [6].

Classification, clinical features and pathophysiology of mesenteric ischemia

Mesenteric ischemia is classified into acute and chronic forms based on the acuteness of symptoms.

Acute mesenteric ischemia is an uncommon cause of acute abdomen, accounting for 0.9 to 0.2% surgical admissions [7]. Acute mesenteric ischemia is classified as occlusive or non-occlusive. Occlusive forms are more common and account for 70–80% of the cases and result from occlusion of mesenteric arteries or veins.

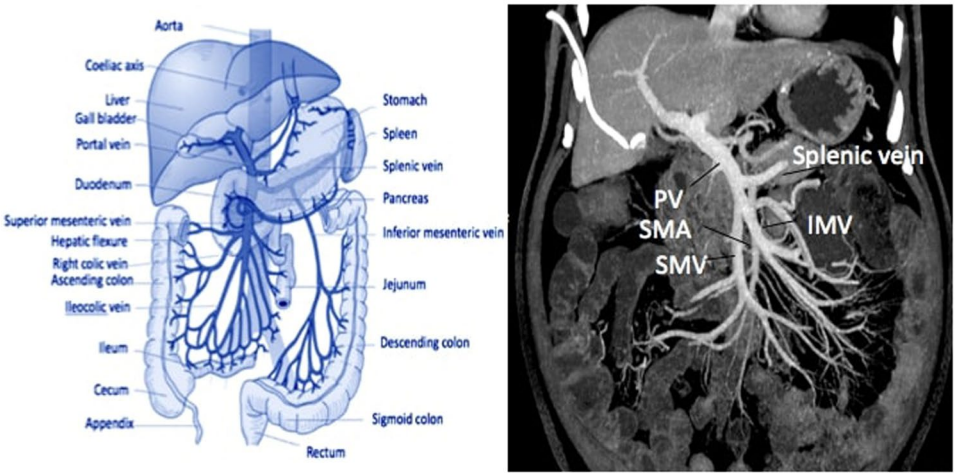

Fig. 2 Diagrammatic representation and CT angiography coronal MIP image of splanchnic venous circulation. *PV* portal vein, *SMV* superior mesenteric vein, *IMV* inferior mesenteric vein, *SMA* superior mesenteric artery

Acute occlusive mesenteric ischemia

Arterial

Clinical features The classical presentation of acute mesenteric ischemia has been described as "pain out of proportion to the clinical exam" [8]. However, it is inconsistently present. The most common presentation is acute non-specific and non-localizing abdominal pain, nausea, vomiting, bloating and sometimes diarrhea with occasional bloody stools [9].

Pathophysiology Acute mesenteric ischemia due to arterial occlusion is a dynamic event. If the acute ischemic is persistent (lasting for more than 6–12 h from the onset of ischemic event) without restoration of arterial supply, it results in an irreversible injury to the entire thickness of the intestinal wall called transmural necrosis with peritonitis and is associated with high mortality. If the initial ischemic event is transient and the blood supply is restored within a short period of time, the intestinal wall becomes thickened due to extravasation of reentered blood components into the interstitial space of the intestinal wall through the damaged micro-vasculature epithelium (from the initial ischemic event).

The various causes of acute mesenteric arterial occlusion are:

Embolism Acute arterial embolus is the most common cause of acute mesenteric ischemia, accounting for 40–50% of cases [6, 8, 10, 11]. Arterial emboli usually are of either cardiac origin (e.g., atrial fibrillation, myocardial infarction) or arise from aortic atheromatous plaques. Arterial emboli usually lodge about 5–6 cm distal to the arterial ostium. The superior mesenteric artery is commonly involved.

Thrombosis Acute thrombosis of SMA is a less common cause of acute mesenteric ischemia in 15–30% of cases and have worse prognosis [6, 7]. Acute arterial thrombosis involves the arterial ostium leading to large territory of ischemic bowel. Atherosclerotic changes predisposes to thrombosis of mesenteric arteries [7, 10].

Dissection Mesenteric artery dissection can be divided into two groups: spontaneous isolated and combined. Combined aortic dissection with secondary involvement of SMA and celiac axis is relatively more common. Intestinal ischemia may result due to narrowing or occlusion of the vessel lumen by the dissection flap if the SMA arises from the false lumen of an aortic dissection [12] [L]. Spontaneous isolated dissection of SMA(SIMAD) is a rare entity [13]. It has been found to be associated with hypertension, connective tissue disorders, atherosclerosis, vasculitis, and trauma.

Venous

Clinical features The onset of mesenteric vein thrombosis (MVT) is characterized by sub-acute abdominal pain that may manifest over 2–4 weeks, with symptoms of nausea and vomiting.

Pathophysiology It accounts for 5–15% of cases of acute mesenteric ischemia. MVT secondary to various underlying diseases such as portal hypertension, hypercoagulable states (e.g., protein C and protein S deficiencies, polycythemia, or factor V Leiden mutation), right-sided heart failure, abdominal trauma, abdominal infection, acute pancreatitis, malignancies, nephrotic syndrome, cirrhosis are more common, accounting for 50–75% of all MVT cases [14–16]. Use of oral contraceptives, pregnancy, and puerperium are risk factors in young women. Primary MVT without an underlying disease are less common in occurrence, accounting for less than 30% cases. [15].

With the onset of venous outflow obstruction, the blood volume in the vascular territory increases due to continuous inflow from relatively higher pressure arterial inflow. This results in an elevation of the intravascular hydrostatic pressure. Progressive oxygen desaturation of the stagnated blood gradually disrupts the capillary wall and increases its permeability. Raised intravascular hydrostatic pressure with increased capillary permeability leads to leakage of intravascular contents into the extravascular interstitial space of the villus, resulting in mucosal edema. Lymphatic vessels drain this fluid into the submucosa and further into the mesentery, resulting in bowel wall and mesenteric edema respectively. With prolonged venous occlusion, there is sloughing of mucosal epithelium with passage of RBCs in stool resulting in bloody diarrhea. Progressive submucosal edema eventually leads to impairment of arterial inflow and transmural necrosis of bowel wall ensues [17].

Acute non-occlusive mesenteric ischemia (NOMI)

Non-occlusive mesenteric ischemia (NOMI) is defined as intestinal hypoperfusion in the absence of vascular occlusion. It accounts for 5–15% (25%) of all cases of acute mesenteric ischemia with a mortality rate of the order of 50% [8, 10, 18].

Clinical features Usually presents with sudden onset of abdominal pain, abdominal distension and, in the advanced stage, signs of peritonitis ensue.

Pathophysiology This condition is usually seen in patients with debilitating comorbid conditions such as shock, cardiac disorders, post-operative stress, pancreatitis, burn, dehydration, and hypovolemia [18]. Cardiac surgery, dialysis and long-term extracorporeal circulation are risk factors for the condition. The pathogenesis of NOMI is thought to arise by a combination of low cardiac output and vasoconstriction. A

decrease of 50% of the blood flow through the superior mesenteric artery triggers the autoregulatory mechanism with a compensatory vasodilatation in the splanchnic circulation. If the hypoperfusion is prolonged for several hours, this compensation ceases to be effective and begins a mesenteric vasoconstriction. The vasoconstriction is reversible if the decline in flow through SMA is corrected rapidly. However, if the splanchnic vasoconstriction lasts more than half an hour, it becomes irreversible, even on 100% reestablishment of blood flow through the artery [19]. NOMI has the worse prognosis as the symptoms are usually masked due to sedation/analgesia. A high index of suspicion is required for early diagnosis. In addition to small bowel ischemia, low-flow states also contribute to non-occlusive forms of ischemic colitis. In a case series by Iacobellis et al. NOMI accounted for 20/32 patients (62.5%) of ischemic colitis [20].

Strangulating/closed-loop obstruction

Strangulating obstruction is a mechanical bowel obstruction resulting in ischemia, seen in approximately 10% of patients with small bowel obstruction. It is usually seen with closed-loop obstruction with obstruction at two different points along the bowel segment at the same level in a C- or U-shaped configuration [14]. The examples of closed-loop obstruction include volvulus, internal hernias and adhesions. Typically, ischemia in a closed-loop bowel obstruction is caused initially by impairment of venous outflow followed by compromise of the arterial supply leading to congestion or hemorrhage in the bowel wall and mesentery. The affected bowel loops are distended and filled with fluid.

Chronic mesenteric ischemia

It is a relatively rare condition accounting for less than 5% of cases and has a more indolent course due to the development of collaterals [21]. These mesenteric collaterals are embryonic remnants of vessels connecting the celiac artery, superior mesenteric artery, and inferior mesenteric artery. Collaterals can develop between two mesenteric arteries or between mesenteric and parietal or body wall vessels in the presence of chronic single or double mesenteric arterial stenosis. The most common collateral pathways found between the CA and the SMA are the pancreaticoduodenal arcades and occasionally the arc of Buhler (a persistent communication between embryonic ventral segmental arteries). Common connections between the SMA and the IMA include the marginal artery of Drummond and the more centrally located arc of Riolan [22] (Fig. 3).

Clinical features Patients with atherosclerotic arterial stenosis present typically with weight loss and post-prandial abdominal pain [8]. Younger patients with vasculitis as underlying cause may present with systemic symptoms like fever, weight loss, weakness, malaise, headache and myalgia along with specific gastrointestinal symptoms such as abdominal pain, nausea and vomiting.

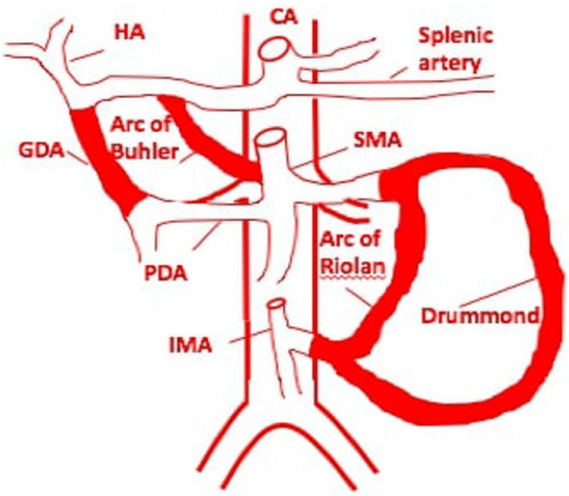

Fig. 3 Diagrammatic representation of collateral pathways in splanchnic circulation between coeliac axis, the SMA and the IMA. *SMA* Superior mesenteric artery, *IMA* inferior mesenteric artery

Pathophysiology Most patients with chronic mesenteric ischemia have ostial atherosclerotic lesions involving at least two of the three mesenteric arteries, affecting the elderly population. Other cases of chronic mesenteric ischemia are vasculitis and median arcuate ligament syndrome [23, 24]. Mesenteric vasculitis accounts for < 5% of all cases of mesenteric ischemia. But the diagnosis should be entertained in young patients without other features of atherosclerotic disease (wall calcifications/plaques). Primary vasculitis is a rare disease characterized by inflammation of the vessel wall [25]. Polyarteritis nodosa, Takayasu's arteritis, microscopic polyangiitis, Wegener's granulomatosis, systemic lupus erythematosus (SLE) and Churg-Strauss syndrome are the major vasculitis subtypes affecting the mesenteric arteries and bowel [21]. Various forms of vasculitis usually affect either the large and medium-sized arteries (Takayasu's) involving aorta and major mesenteric arterial trunk or medium-sized arteries such as branches of coeliac axis, SMA, IMA (polyarteritis nodosa, Wegener's and SLE). Smaller terminal intramural arterioles, venules and capillaries are affected in microscopic polyangiitis. Chronic inflammation of the vessel wall can cause intimal-medial thickening resulting in stenosis and occlusions as in Takayasu's arteritis. Polyarteritis nodosa, Wegener's granulomatosis and SLE lead to fibrinoid necrotizing angitis with weakening of the media and thinning of the arterial wall causing an aneurysm. The median arcuate ligament is a fibrous arch that unites the diaphragmatic crura on either side of the aortic hiatus. The ligament usually passes superior to the origin of the celiac axis. In some individuals it is lower in position

causing indentation on the origin of celiac axis with focal narrowing and significant hemodynamic ostial stenosis of the celiac axis with resultant ischemia in gastroduodenal circulation [24]. Chronic NOMI is a less recognized entity and is characterized by low-grade ischemia, possibly due to insufficient mesenteric circulation [26]. It is associated with cardiac forward failure, pulmonary hypertension, severe chronic obstructive pulmonary disease, vasospasms of the mesenteric arteries, low-flow states (e.g., patients with chronic kidney disease on dialysis) and severe anemia. In contrast to acute NOMI, this does not progress to transmural necrosis or bowel infarction.

Laboratory markers

Currently there is no specific laboratory marker for acute or chronic mesenteric ischemia. Metabolic acidosis, elevated lactate and D-dimer, leukocytosis, raised amylase or liver enzymes are non-specific findings which are seen in AMI as well as other acute abdominal emergencies [10, 27, 28].

Diagnostic modalities and imaging features

MDCT is the gold standard procedure in the evaluation of suspected cases of mesenteric ischemia. It has high diagnostic performance for detection of AMI, with a sensitivity of 64–96% and a specificity of 92–100% [29–31]. It not only confirms the diagnosis but also assesses the severity and complications of the condition. In addition, it is helpful in ruling out other causes of abdominal pain.

Protocol We follow a triple-phase protocol in our institute, including non-contrast images followed by dual-phase contrast-enhanced CT study using IV non-ionic contrast. The utility of non-contrast phase is debatable in view of increased radiation dose to the patient. Nevertheless, the non-contrast phase helps in differentiating intramural hemorrhage or hemorrhagic infarction from hyperemia or hyperperfusion as the cause of high bowel wall attenuation in the setting of ischemia, which may not be possible when assessed on contrast CT alone. With the help of dual-energy CT scanners, the need for a separate non-contrast phase acquisition can be overcome by generating it synthetically using iodine subtraction, thereby reducing radiation dose to patients. A recent study has highlighted the potential benefits of dual-energy CT iodine maps and 40-keV mono-energetic dual-energy CT scanners in the evaluation of mesenteric ischemia and bowel injury, because its iodine sensitivity permits reduction of iodine dose and concentration, reducing the risk of contrast-enhanced acute kidney injury in patients at risk [32, 33]. In the acute settings, oral contrast is not given to avoid unnecessary delay in the diagnosis. Moreover, the bowel loops are usually dilated due to ischemic event and fluid in the lumen act as intrinsic contrast. In chronic setting, the bowel may be distended with neutral oral contrast such as water. Positive oral contrast should be avoided in CT angiography as it can obscure mucosal changes occurring in the setting of ischemia. About 100–150 mL of non-ionic iodinated contrast material is administered via IV injector at a rate of 4–5 mL per second using bolus tracking method with ROI placed in the descending aorta. The arterial phase is acquired at a scanning delay of 25–30 s and venous phase at 60–70 s. Arterial phase images are used for mesenteric arterial evaluation and venous phase images for the assessment of splanchnic venous circulation and bowel loops. Recently, in a retrospective study, researchers have reported a low yield of venous phase images in the evaluation of mesenteric ischemia [34]. But, in our opinion, venous phase images are important to assess the status of bowel wall and mucosa and are indispensable for the diagnosis of venous occlusion. Rapid contrast administration and volume acquisition with thin sections allows the assessment of tiny distal mesenteric branches. Initial assessment is made using the axial source images followed by the post-processed images. Multi-planar reconstruction with evaluation of the vessel lumen in all three planes axial, coronal and sagittal images are pertinent to avoid missing small intraluminal filling defects. Maximum intensity projections (MIP) images are equally important to evaluate the tiny distal branches. Volume rendered images are helpful in quick analysis of the vessel ostia, presence of any anomalous arterial origin and collateral circulation.

Imaging features on MDCT

Acute mesenteric ischemia

The imaging features on CT angiography depends on many factors such as the type of the vascular structure involved (arterial/venous), patency of vessel (occlusive/non-occlusive), lapse of time passed from the onset of ischemic event to imaging, and finally the presence or absence of a reperfusion process.

Arterial

Vascular

Embolus appears as intraluminal filling defects, usually located 5–6 cm away from the ostium in the main arterial trunk or within distal branches (Fig. 4). Arterial thrombosis usually occurs in the setting of previous severe atheromatous disease. Vessel wall abnormalities in the form of mural calcifications and wall thickening with intraluminal

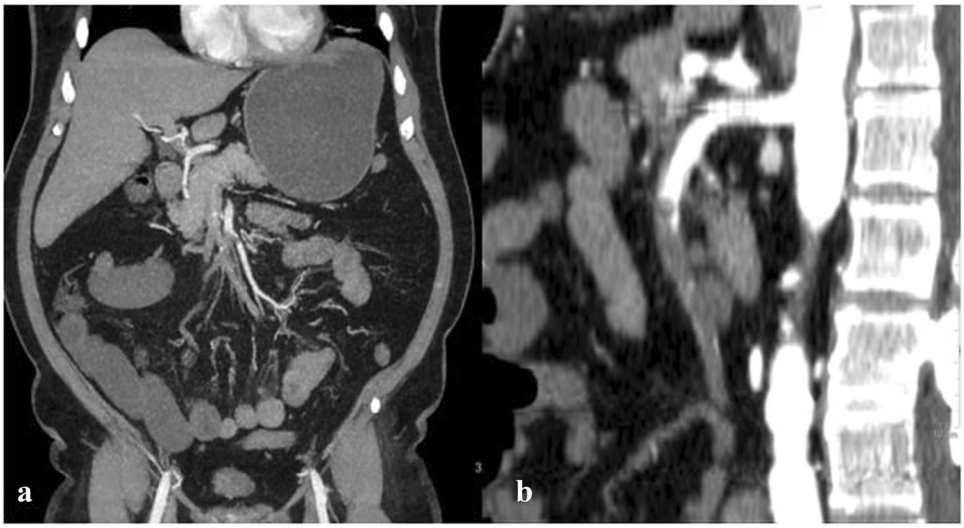

Fig. 4 Acute mesenteric ischemia in a 70-year-old man with a history of atrial fibrillation presenting with acute pain and bloating of abdomen. **a** Coronal CT angiography (MIP) image shows intraluminal filling defect in the mid and distal SMA representing embolus extending into the ileal and colonic branches. Dilated ileal loops are seen in the right lower quadrant with markedly thinned out hypoenhancing wall suggesting ischemic changes. **b** Sagittal MIP CT angiography image shows intraluminal filling defect in the mid and distal SMA with coincidental atherosclerotic plaque and luminal narrowing in abdominal aorta. *SMA* superior mesenteric artery

filling defects at the ostium of splanchnic arteries are seen (Fig. 5a). SMA dissection with resultant luminal thrombosis is an uncommon cause of AMI. It can occur as a continuation of aortic dissection (Fig. 6) or in isolation (Fig. 7). Dissection flap with distal luminal filling defect representing thrombosis is seen on arterial phase images.

Bowel wall

When normally perfused, the thickness of bowel wall ranges from 3 to 5 mm with uniform, homogeneous mural enhancement on venous phase images. Diminished or absent post-contrast enhancement of the bowel mucosa is a specific but not sensitive finding for acute arterial MI (Fig. 4a). Arterial occlusion without restoration of blood flow leads to intramural microvascular capillary damage and loss of muscle tone with luminal dilatation typically causing paper thin wall (Figs. 4, 5b). Frequently, intramural air foci (pneumatosis) are seen (Fig. 5c), presumably due to entry of gas into bowel via a disrupted mucosa. Pneumatosis may not necessarily indicate irreversible injury [35]. The presence of portomesenteric venous gas and intraperitoneal free air are regarded as ominous signs of irreversible injury and transmural bowel necrosis with bowel perforation. In cases with

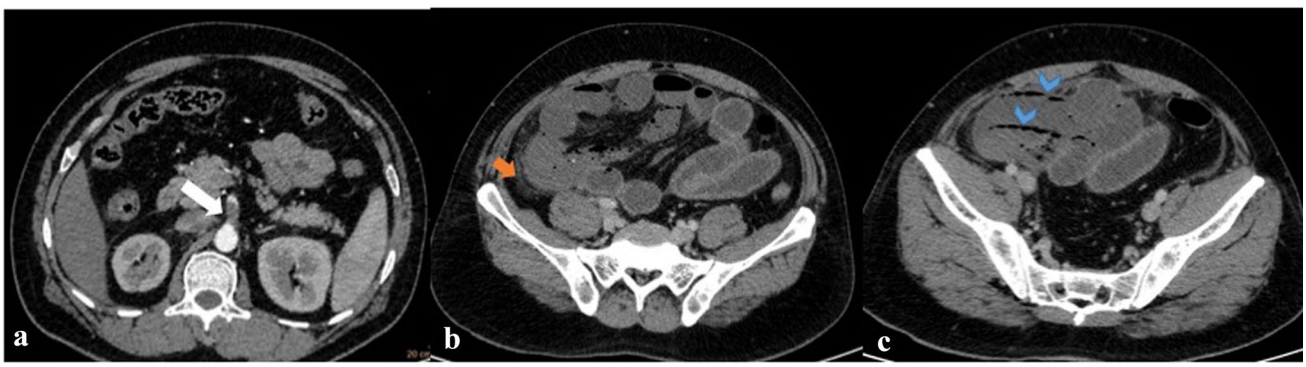

Fig. 5 Acute SMA thrombosis in a 56-year-old male with history of diabetes and dyslipidemia presenting with sudden onset abdominal pain, vomiting and diarrhea. **a** Axial CT angiography image of upper abdomen showing intraluminal filling defect at the origin of the SMA (white arrow). Venous phase CT image of lower abdomen shows **b** dilated ileal loops with non-enhancing paper thin wall in right lower quadrant and adjacent mesenteric fat stranding (orange arrow) suggesting transmural bowel necrosis and **c** intramural air foci suggestive of pneumatosis (blue arrow head). *SMA* superior mesenteric artery

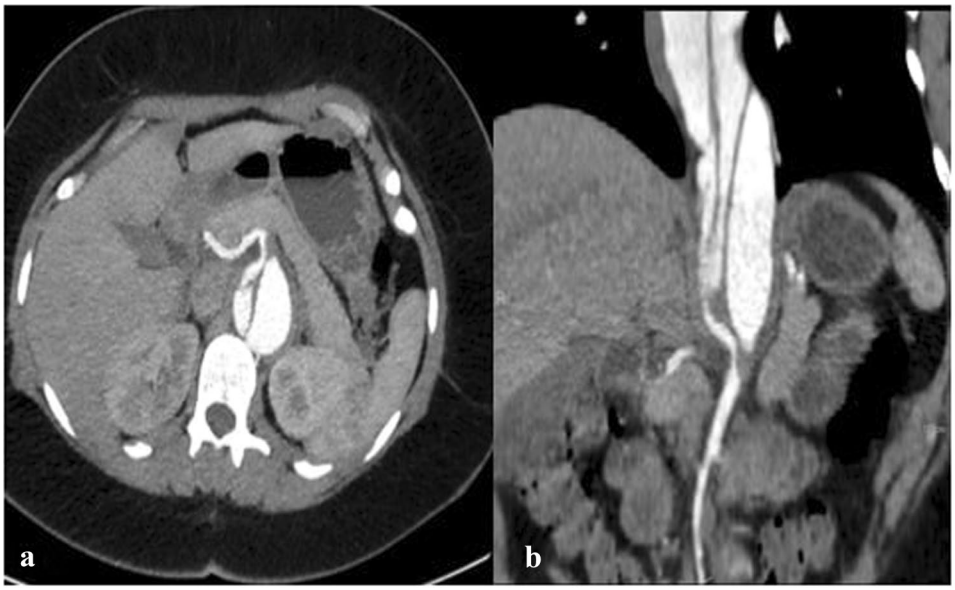

Fig. 6 Aortic dissection in a 63-year-old female with a chronic history of hypertension presenting with acute onset chest and abdominal pain CT angiography(MIP) image of upper abdomen shows aortic dissection extending into the origin of **a** celiac axis and **b** the SMA. Notice both the arteries are arising from the true lumen with secondary thrombosis and ostial narrowing. *SMA* superior mesenteric artery

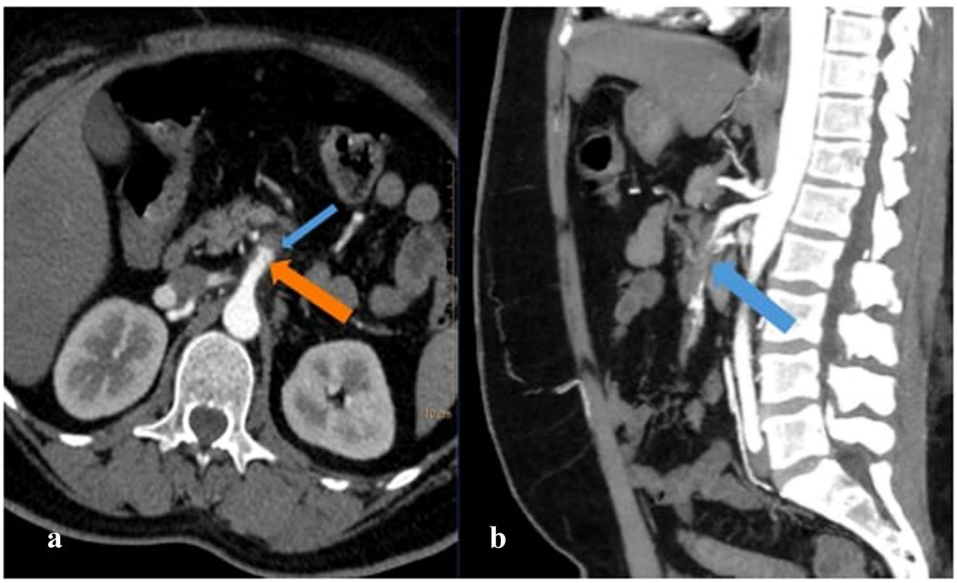

Fig. 7 Acute spontaneous isolated dissection of the SMA in a 59-year-old male with history of chronic hypertension presenting with acute onset abdominal pain and distension. **a** Axial and **b** sagittal CT angiography images shows the dissection flap in the proximal SMA (orange arrow) with associated distal intraluminal filling defect suggesting thrombosis (blue arrow). SMA superior mesenteric artery

transient arterial occlusion with restoration of blood flow, bowel wall thickening with target appearance, intramural hemorrhage/edema and mucosal hyperenhancement indicate the presence of reperfusion.

Mesentery

Mesenteric strands and ascites are rare in the early course of the disease. Focal mesenteric stranding appears adjacent to the involved bowel segment in prolonged arterial occlusion with transmural infarction (Fig. 5); however it is less pronounced in comparison to venous occlusion or NOMI.

Venous

Vascular

Thrombi are seen as intraluminal filing defects in the portomesenteric venous system, best visualized on the venous phase images (Fig. 8a, b).

Bowel wall

Bowel loops show circumferential wall thickening with target or stratified enhancement pattern due to enhancing mucosal and serosal layers and intervening edematous

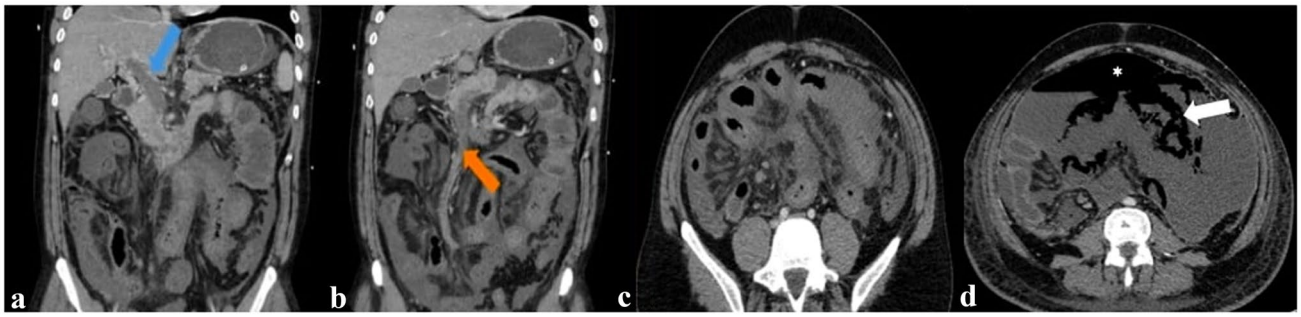

Fig. 8 Acute mesenteric venous thrombosis in a 36-year-old male with protein C deficiency presenting with 2 weeks history of abdominal pain, nausea, distension. Contrast-enhanced CT coronal venous phase images of abdomen show intraluminal filling defect in the **a** portal (blue arrow) and **b** the superior mesenteric veins (orange arrow). **c** Contrast-enhanced CT axial venous phase image of abdomen shows diffuse mesenteric congestion and target appearance of edematous small bowel loops. Patient was managed conservatively with systemic anticoagulation. **d** Follow-up axial CT after 4 days shows transmural necrosis of bowel wall with diffuse intramural air foci (pneumatosis intestinalis) (white arrow) and free intraperitoneal air (asterisk) suggesting bowel perforation

submucosal and muscular layers (Fig. 8c). Impeded venous return leads to capillary congestion causing mucosal hyperenhancement on contrast-enhanced CT scans. Untreated severe cases of venous thrombosis, may lead to bowel infarction. Absent bowel wall enhancement indicates transmural infarction, particularly when it is associated with pneumatosis, portomesenteric venous gas and intraperitoneal free air (Fig. 8d). The extent of bowel wall thickening, mesenteric fat stranding, or ascites does not correlate with the severity of ischemic bowel damage in venous occlusion.

Mesentery

Diffuse congestion and edema is noted in mesenteric fat in the form of haziness on CT (Fig. 8a).

NOMI

Vascular

Segmental, non-consecutive vasoconstriction/spasm is seen in the splanchnic arteries, most commonly in the SMA [9]. However, the arteries remain patent and no intraluminal filling defect is observed (Fig. 9a). The vasoconstriction is reversible and the vascular caliber returns to normal when the underlying cause is removed (Fig. 9b). It usually involves a wider area of splanchnic circulation. IVC luminal narrowing known as flat IVC and hyperenhancing adrenals are other associated findings [9, 18].

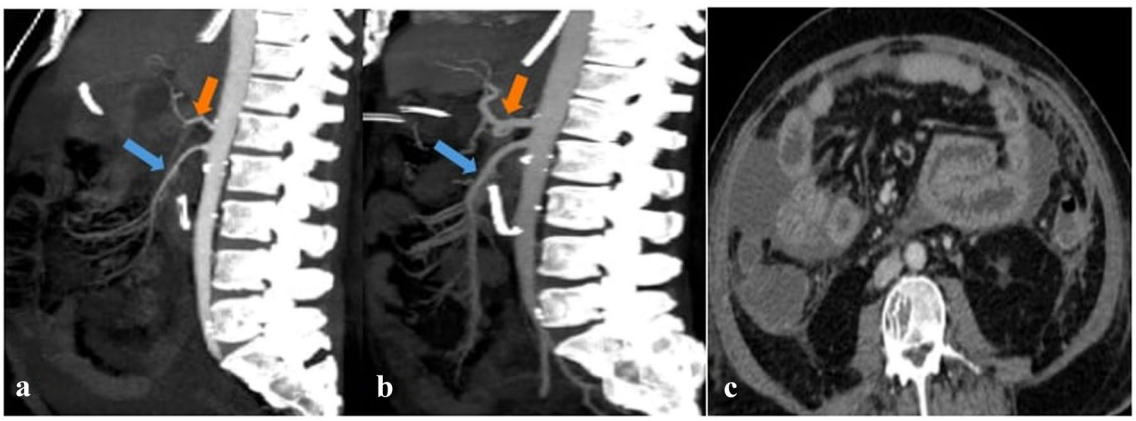

Fig. 9 NOMI in a 40-year-old male with 1-week history of acute severe pancreatitis, hypovolemia and abdominal distension with raised serum lactate levels. **a** CT angiography (MIP) image in sagittal plane shows marked narrowing of the coeliac axis (orange arrow) and SMA(blue arrow) without intraluminal filling defect. Notice the presence of gross ascites. Post-hypovolemia correction CT angiography. **b** Sagittal MIP image shows reversal of normal coeliac axis (orange arrow) and SMA (blue arrow) luminal diameter. **c** Axial sections of the lower abdomen shows mural thickening with mucosal hyperenhancement of the small bowel loops suggesting reperfusion. SMA superior mesenteric artery

Bowel wall

The characteristic CT appearance resembles that of classic arterial occlusion, i.e., thinning and lack of enhancement of bowel wall with luminal dilatation. Porto venous gas and free intraperitoneal air foci may be seen if transmural necrosis ensues. Diffuse bowel wall thickening with mucosal hyperenhancement are seen in the setting of reperfusion (Fig. 9c).

Mesentery

Mesenteric fat stranding and/or with ascites may be observed depending on the underlying disease or if reperfusion sets in (Table 1).

Strangulating obstruction

Bowel

On CT, a closed-loop obstruction is identified by a unique configuration of C- or U-shaped distended loops with the mesenteric vessels converging toward the site of obstruction. The affected bowel is filled with fluid. In cases with strangulation, the bowel wall is thickened and shows absent or diminished enhancement, hyperenhancement, or a halo or target pattern of enhancement on contrast-enhanced CT examination (Fig. 10).

Table 1 Summary of imaging features of mesenteric ischemia

	Arterial		Venous	Nomi
	Acute	Chronic		
Vessel	Intraluminal filling defect in SMA, coeliac axis or IMA in thrombosis/embolism Dissection flap with luminal occlusion Aneurysmal dilatation of splanchnic artery	Ostial narrowing with intimal-medial plaques in atherosclerosis Circumferential narrowing in vasculitis Collateral arterial circulation	Intraluminal filling defect in SMV and portal vein	No filling defects in arteries or veins Small caliber arteries
BOWEL	Early Ischemia Thinning with diminished enhancement of the wall Late infarction Hypotonic dilatation, non-enhancing bowel wall with pneumatosis intestinalis	No significant abnormality in atherosclerotic stenosis Circumferential wall thickening with submucosal edema may be seen in vasculitis	Early-Mucosal hyperenhancement, bowel wall thickening, Bowel wall dilatation Late Infarction Pneumatosis and intravenous air	Segmental hypoenhancing, dilated bowel loops Wall thickening with mucosal hyperenhancement after restoration of vascularity/reperfusion
MESENTERY	Normal in early stages Focal mesenteric fat stranding and occasional ascites due to transmural infraction	Normal	Hazy/fat stranding due to venous congestion and resultant edema with ascites	Usually normal Occasional fat stranding or ascites may be seen related to predisposing condition

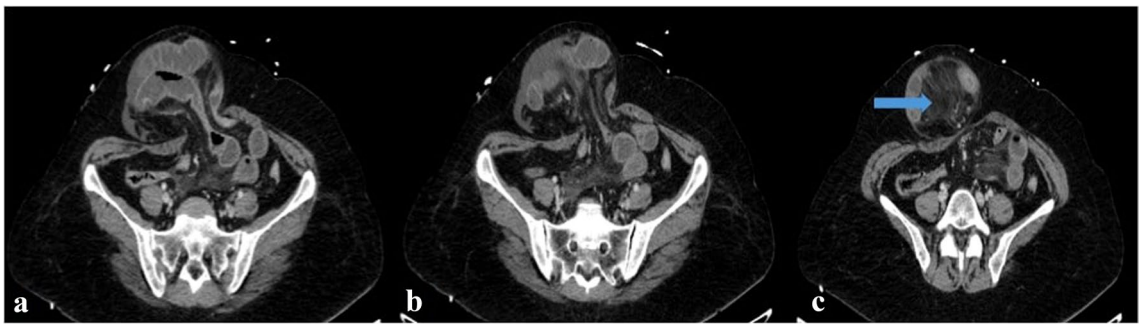

Fig. 10 Ventral hernia with closed-loop obstruction in a 50-year-old woman presenting with acute abdominal pain and vomiting. Axial contrast-enhanced CT image through the mid abdomen shows **a** C-shaped obstruction with dilated fluid filled small bowel loops in the hernia sac, entering through ventral abdominal wall defect. **b** Congested veins seen in the adjacent mesentery. **c** Notice the typical fan-shaped mesentery (Blue arrow)

Mesentery

The affected mesentery typically shows a fan shape. The affected mesentery shows infiltration and stranding associated with engorged mesenteric veins (Fig. 10). Ascites may also be observed.

Chronic mesenteric ischemia

Vascular

Chronic mesenteric ischemia is usually associated with atherosclerotic disease of aorta in elderly population. The typical imaging finding is diffuse atherosclerotic plaques (calcified or non-calcified) at the origin and/or distally causing significant luminal narrowing in the splanchnic arteries (Fig. 11). As explained earlier, there is always presence of a well developed collateral circulation. In case of celiac axis stenosis, the flow is maintained through prominent pancreaticoduodenal arcade around pancreatic head from SMA to gastroduodenal artery, a branch of celiac axis and rarely via arc of Buhler (Figs. 3, 12). SMA stenosis leads to establishment of flow from IMA via marginal artery of Drummond and arc of Riolan (Fig. 3). Mesenteric arterial vasculitis is an uncommon cause of chronic ischemia of bowel. The imaging features differ depending upon the underlying type. Irregular vessel walls, stenosis, post-stenotic dilatation, aneurysm formation, occlusion, and evidence of increased collateral circulation are the hall marks of vascular changes in vasculitis (Figs. 12, 13). Median arcuate ligament syndrome on CT angiography presents as a soft tissue external indentation on the proximal celiac artery leading to stenosis and characteristic hooked appearance on the sagittal reformatted image (Fig. 14).

Bowel wall/mesentery

Bowel appearance may be normal in atherosclerotic stenosis. Bowel wall is usually abnormal in vasculitis and shows concentric thickening with target appearance. Mesenteric congestion with or without ascites may be seen.

CT mimickers of mesenteric ischemia

Luminal dilatation is a non-specific finding in acute mesenteric ischemia and can be seen in bowel obstruction and paralytic ileus. Bowel wall thickening with target appearance is also seen in acute stage of Crohn's disease, infectious enteritis, acute radiation enteritis, shock bowel mimicking veno-occlusive disease. The pattern of bowel involvement (segmental with skip areas in Crohn's disease), normal portomesenteric venous contrast opacification with specific clinical or prior treatment history may help in differentiating these entities from veno-occlusive mesenteric ischemia. Pneumatosis intestinalis, although considered a sign of acute ischemic damage to bowel mucosa, it may be associated with several benign conditions like connective tissue disease, emphysema, asthma, and intra-abdominal procedures. Patients are usually asymptomatic in these cases with otherwise normal bowel wall thickness and enhancement pattern and absence of portomesenteric gas.

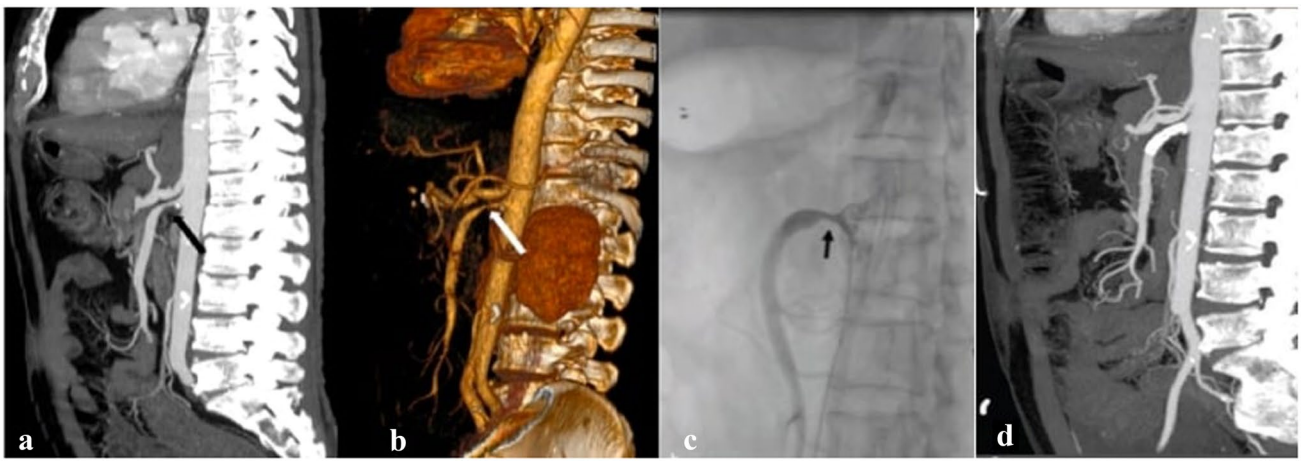

Fig. 11 Chronic mesenteric ischemia in a 71-year-old man with 6 months history of chronic post-prandial abdominal pain. **a** Pre-stenting sagittal MIP CT angiography image of the abdomen shows atherosclerotic plaque with speck of calcification at the SMA ostium (long black arrow). **b** Volume rendered CT angiography of the abdomen (white arrow). **c** Digital subtraction angiography images (small black arrow) show marked ostial narrowing of the SMA. **d** Post-stenting sagittal MIP CT angiography image showing restoration of luminal diameter in the proximal SMA. *SMA* superior mesenteric artery

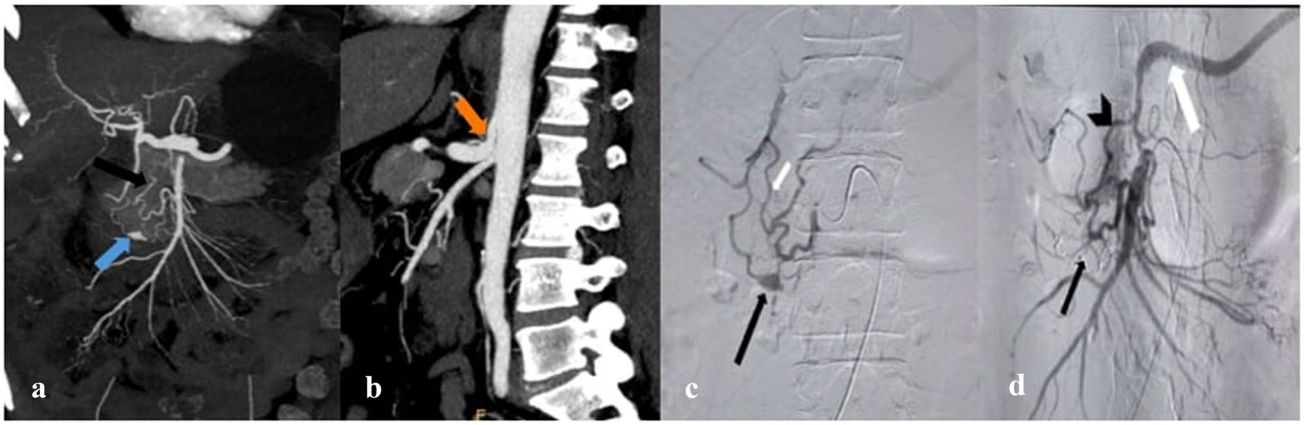

Fig. 12 Polyarteritis nodosa in a 40-year-old woman with a history of intermittent non-specific abdominal pain. CT angiography (MIP) images in **a** coronal plane shows presence of a small aneurysm along the pancreaticoduodenal artery (blue arrow) and in **b** sagittal plane shows focal narrowing of the proximal coeliac axis with post-stenotic dilatation (blue arrow). Notice the presence of arc of Buhler (black arrow), a communication between celiac axis and SMA. Digital subtraction angiogram images with selective run **c** through the arc of Buhler (white arrow) show small aneurysm along pancreaticoduodenal artery (black arrow). **d** Through the SMA (short black arrow) shows filling of splenic artery (white arrow), a branch of coeliac axis via the arc of Buhler (black arrow head). The aneurysm was embolized with coil (Black arrow). *SMA* superior mesenteric artery

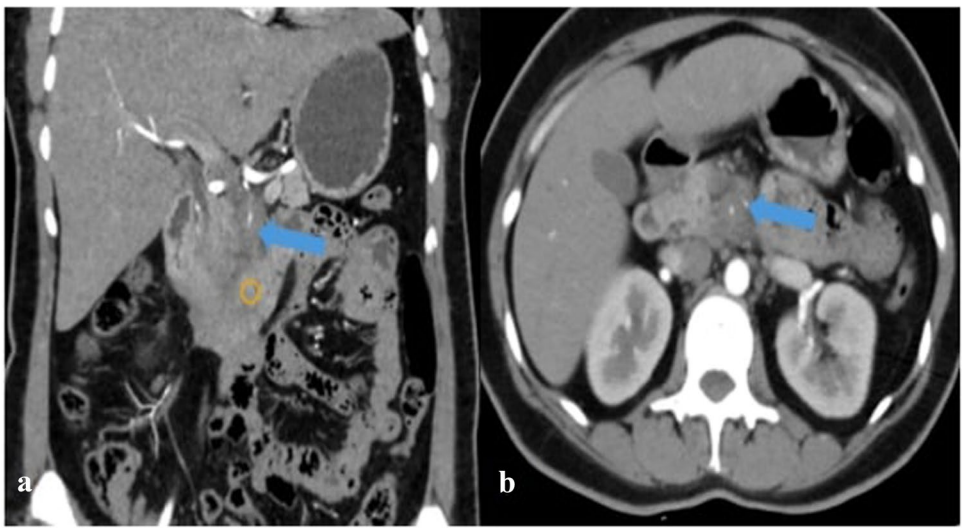

Fig. 13 Imaging features consistent with the diagnosis of vasculitis in a 28-year-old female with 2 months' history of abdominal pain, nausea and generalized myalgia and arthralgia. **a** Coronal and **b** axial CT angiography image of abdomen shows long segment, concentric wall thickening with marked luminal narrowing of the SMA. *SMA* superior mesenteric artery

Role of other imaging modalities

Conventional X-ray

Radiography has a limited role in the evaluation of acute abdominal pain because of its non-specific findings and low diagnostic yield [36]. Approximately 25% patients of AMI have normal radiographs at initial presentation [37]. Positive findings on radiography in patients with acute mesenteric ischemia usually appear late in the course of illness after occurrence of bowel infarction and are, therefore, associated with a high mortality rate. Bowel dilatation, pneumatosis intestinalis, portal venous gas are important radiological findings suggesting advanced mesenteric ischemia and poor prognosis [37, 38].

Color Doppler

Role of B-mode ultrasound and color Doppler is limited in the diagnosis of acute mesenteric ischemia as the performance depends upon operator's expertise and patient factors such as body habitus and presence or absence of gaseous distension of bowel loops. Color Doppler may be used for follow-up after endovascular procedure or post-anticoagulation to monitor treatment response. It is used for initial screening of chronic mesenteric ischemia (Fig. 15a–c) as well as follow-up after stenting (Fig. 15d). Duplex study is usually

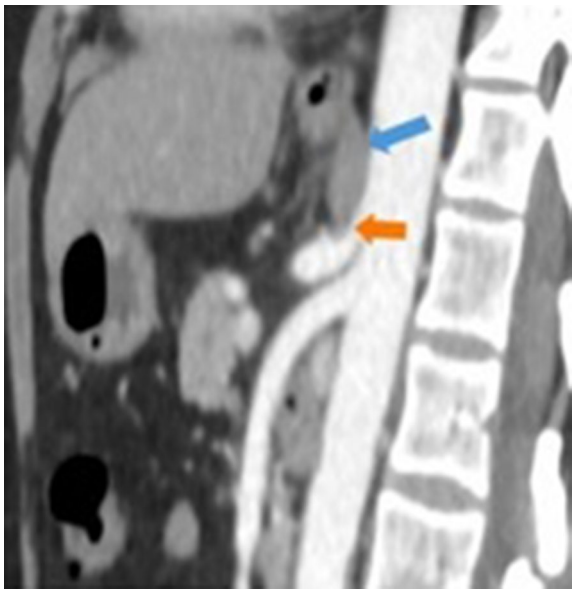

Fig. 14 Median arcuate ligament syndrome in a 53-year-old man with history of intermittent abdominal pain. CT angiography of abdomen sagittal image shows oblong soft tissue indentation (blue arrow) at the celiac axis origin with ostial narrowing (orange arrow) and post-stenotic dilatation giving rise to characteristic hook-shaped configuration

performed in the fasting state to avoid bowel gas which may obscure visibility of the mesenteric vasculature. Out of all color Doppler indices, peak systolic velocity is the most accurate parameter for diagnosing stenosis [39]. According to study done by Ali et al. (2012), the PSV threshold with highest overall accuracy (OA) for detecting ≥ 50% SMA stenosis was ≥ 295 cm/s and for detecting ≥ 70% SMA, it was ≥ 400 cm/s. For celiac arteries, the PSV threshold that provided the highest OA for ≥ 50% stenosis was ≥ 240 cm/s and for ≥ 70% stenosis was ≥ 320 cm/s [39].

Contrast-enhanced magnetic resonance angiography (CEMRA)

CEMRA performs better in grading mesenteric vessel stenosis compared to DSA. However, the overall accuracy and inter-observer agreement are lower compared to CTA. MRA has lower spatial resolution as compared to CTA, making it unreliable in the assessment of distal branches and the IMA [40]. Moreover, longer acquisition time and lack of availability at all centers make it unsuitable in acute settings. Non-contrast MRA has low spatial resolution which often leads to overestimation of vascular stenosis. CEMRA can be used for the diagnosis of ostial stenosis in suspected chronic mesenteric ischemia with reported sensitivity and specificity up to 95% to 100% [41].

Digital subtraction catheter angiography

CT angiography has virtually replaced digital subtraction angiography as the diagnostic modality of choice in the

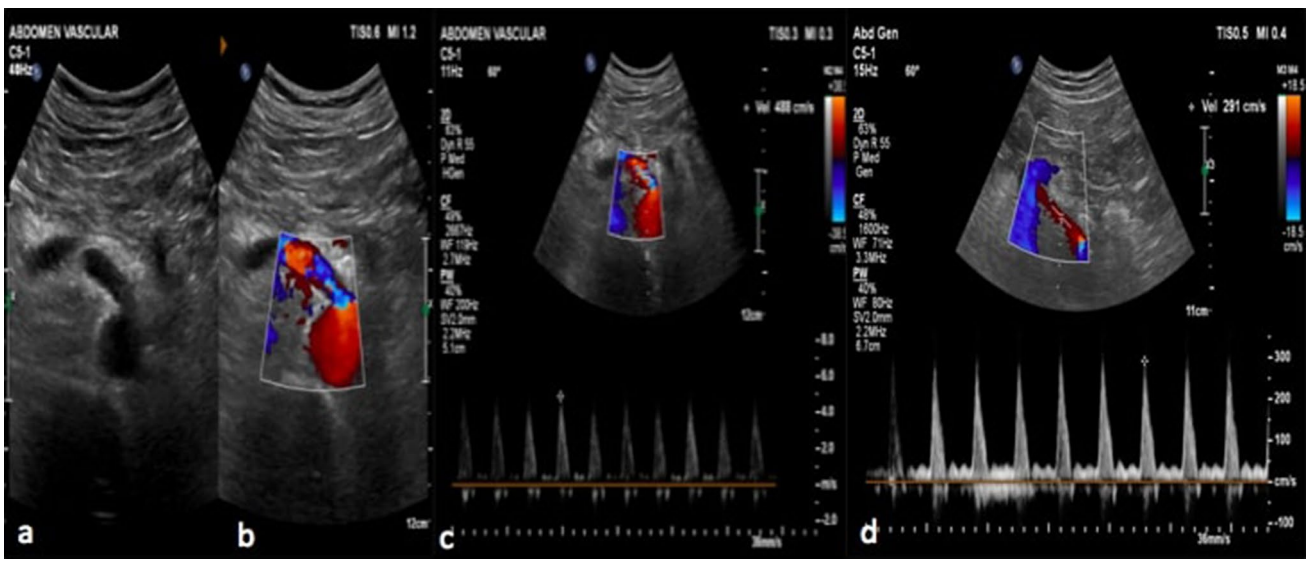

Fig. 15 Chronic mesenteric ischemia in a 71-year-old man with 6 months history of chronic post-prandial abdominal pain. **a** B-mode and ultrasound images of the SMA shows atherosclerotic plaque at the ostium causing marked stenosis. **b** Color Doppler image shows aliasing at the origin. **c** Spectral waveform at the stenotic segment shows peak systolic velocity(PSV) 488 cm/s with spectral broadening suggesting > 70% stenosis. **d** Color Doppler image of the SMA post-stenting shows good flow across the stent with reduction in the PSV to 291 cm/s. SMA superior mesenteric artery

evaluation of mesenteric ischemia. Its role is now mainly limited to endovascular treatments.

Role of interventional radiology in the management of mesenteric ischemia

Earlier, management of AMI was primarily surgical, centered at restoration of mesenteric blood flow and resection of necrotic bowel. However, in recent times, endovascular management has emerged as an alternative, less invasive targeted treatment option for early cases of reversible vascular bowel ischemia [42]. The key to a favorable prognosis in cases of AMI is early detection and prompt initiation of management. The mortality rate ranges from 0 to 10% with immediate management, increases to 50–60% with a treatment delay of 6–12 h, and increases further to 80–100% with a delay of more than 24 h after symptom onset [43]. Several non-randomized trials have shown a benefit for endovascular therapy compared to open surgery in terms of lower bowel resection rates and lower 30 day mortality rates [44–46].

Acute mesenteric ischemia

The endovascular management is aimed at effective thrombolysis and restoration of blood flow through the occluded vessel.

Arterial Mechanical embolectomy, thrombus fragmentation and aspiration followed by transcatheter thrombolysis are the preferred procedures for revascularization in acute mesenteric artery embolism/thrombosis [42]. Since the underlying vessel is usually stenosed in thrombotic occlusions, balloon expandable stent is deployed at the stenotic segment of the involved mesenteric artery following thrombolysis/thrombectomy. Arterial dissections are treated with stent placement to maintain the patency of lumen with/without catheter guided thrombolysis.

Venous Endovascular treatment for mesenteric vein thrombosis includes direct thrombolysis either by percutaneous transhepatic approach or by a surgically placed SMV catheter in combination with thrombectomy and indirect thrombolysis through the SMA [47].

NOMI Correction of hypovolemia and normalization of cardiac output are the mainstay steps in the management of NOMI. However, sometimes the vascular spasm persists even after correcting the inciting factor. In those cases, transcatheter infusion of vasodilators into the SMA is used to reverse the vasoconstriction. The vasodilator drugs used are prostaglandin E1 and papaverine [18].

Chronic mesenteric ischemia

Percutaneous transluminal angioplasty (PTA) is an effective alternative, less invasive endovascular treatment option for chronic ischemia in atherosclerotic or vasculitic stenosis. It involves transcatheter balloon dilatation of the stenosed artery with restoration of normal luminal diameter [48]. It is a less invasive technique as compared to surgical revascularization bypass procedures. Endovascular stent placement should be considered in cases of higher grades of stenosis of CA or SMA or if the residual stenosis is > 30% or a persistent pressure gradient of > 15 mmHg is seen after angioplasty (Fig. 11d) [49]. Stents also help in reducing post-procedural complications of PTA like arterial dissection and restenosis.

Conclusion

Mesenteric ischemia is an uncommon entity with high morbidity and mortality in the acute setting. A high degree of clinical suspicion with imaging correlation is pertinent for early diagnosis to improve clinical outcome. MDCT is the first line imaging modality for the diagnosis of mesenteric ischemia due to its wide spread availability, faster acquisition and great spatial resolution. CT angiography allows for a comprehensive non-invasive assessment of the abdominal vasculature as well as the bowel and mesentery. It not only detects the cause of ischemia but also helps in differentiating reversible and irreversible ischemic event based on the bowel wall and mesenteric findings, thereby predicting outcome. Endovascular catheter thrombolysis is the preferred treatment option for early reversible acute ischemia with better patient outcome and lesser morbidity. Percutaneous transluminal angioplasty with stenting is a less invasive, effective treatment option for chronic mesenteric stenosis as compared to surgical revascularization.

References

1. Haghighi PH, Lankarani KB, Taghavi SA et al. Acute mesenteric ischemia: causes and mortality rates over sixteen years in southern Iran. Indian J Gastroenterol 2008;27(6):236–238.
2. Wadman M, Syk I, Elmståhl S. Survival after operations for ischaemic bowel disease. Eur J Surg 2000;166(11):872–877.
3. Tsai MS, Lin CL, Chen HP et al. Long-term risk of mesenteric ischemia in patients with inflammatory bowel disease: a 13-year nationwide cohort study in an Asian population. Am J Surg 2015;210(1):80–86.
4. Horton KM, Fishman EK. CT angiography of the mesenteric circulation.Radiol Clin North Am 2010;48:331-45, viii.

5. Rosenblum JD, Boyle CM, Schwartz LB. The mesenteric circulation. Anatomy and physiology. Surg Clin North Am 1997;77:289-306.
6. Costa AF, Chidambaram V, Lee JJ, et al. Multidetector computed tomography of mesenteric ischaemia. Insights Imaging 2014;5:657-666(2014).
7. Herbert GS, Steele SR. Acute and chronic mesenteric ischemia. Surg Clin North Am 2007;87:1115-34, ix.
8. Clair DG, Beach JM. Mesenteric Ischemia. N Engl J Med 2016;374:959-68.
9. Gore RM, Yaghmai V, Thakrar KH, et al. Imaging in intestinal ischemic disorders. Radiol Clin North Am 2008;46:845-75, v.
10. Bobadilla JL. Mesenteric ischemia. Surg Clin North Am 2013;93:925-40, ix.
11. Dhatt HS, Behr SC, Miracle A, et al. Radiological Evaluation of Bowel Ischemia. Radiol Clin North Am 2015;53:1241-54.
12. Wasnik A, Kaza RK, Al-Hawary MM, et al. Multidetector CT imaging in mesenteric ischemia—pearls and pitfalls. Emerg Radiol 2011;18:145-56.
13. Jia Z, Tu J, Jiang G. The classification and management strategy of spontaneous isolated superior mesenteric artery dissection. Korean Circ J 2017;47(4):425–431.
14. Furukawa A, Kanasaki S, Kono N, et al. CT diagnosis of acute mesenteric ischemia from various causes. AJR Am J Roentgenol 2009;192:408-16.
15. Goldberg MF, Kim HS. Treatment of acute superior mesenteric vein thrombosis with percutaneous techniques. AJR Am J Roentgenol. 2003;181 (5): 1305-7.
16. Warshauer DM, Lee JK, Mauro MA et al. Superior mesenteric vein thrombosis with radiologically occult cause: a retrospective study of 43 cases. AJR Am J Roentgenol. 2001;177 (4): 837-41.
17. Watson DW, Sodeman WA. The small intestine. In: Sodeman WA, Sodeman TM, editors. Pathologic physiology-Mechanisms of disease. Philadelphia: WB Saunders; 1985. p. 813-51.
18. Trompeter M, Brazda T, Remy CT et al. Non-occlusive mesenteric ischemia: etiology, diagnosis, and interventional therapy. Eur Radiol 2002;12(5): 1179–1187.
19. Wilcox MG, Howard TJ, Plaskon LA et al. Current theories of pathogenesis and treatment of nonocclusive mesenteric ischemia. Dig Dis Sci 1995; 40: 709–16.
20. Iacobellis F, Berritto D, Fleischmann D et al. CT findings in acute, subacute, and chronic ischemic colitis: suggestions for diagnosis. Biomed Res Int. 2014;2014:895248.
21. T.G. Walker Mesenteric vasculature and collateral pathways Semin Intervent Radiol, 26 (2009), pp. 167-174
22. J.H. van Bockel, R.H. Geelkerken, M.N. Wasser Chronic splanchnic ischaemia Best Pract Res Clin Gastroenterol, 15 (2001), pp. 99-119
23. Ha HK, Lee SH, Rha SE et al. Radiologic features of vasculitis involving the gastrointestinal tract. RadioGraphics 2000;20(3):779–794.
24. Horton K.M.,Talamini M.A., Fishman E.K. Median arcuate ligament syndrome: evaluation with CT angiography. RadioGraphics. 2005; 25: 1177-1182
25. M.D. Morgan, C.O. Savage.Vasculitis in the gastrointestinal tract Best Pract Res Clin Gastroenterol, 19 (2005), pp. 215-233
26. Terlouw LG, Moelker A, Abrahamsen J et al. European guidelines on chronic mesenteric ischaemia - joint United European Gastroenterology, European Association for Gastroenterology, Endoscopy and Nutrition, European Society of Gastrointestinal and Abdominal Radiology, Netherlands Association of Hepatogastroenterologists, Hellenic Society of Gastroenterology, Cardiovascular and Interventional Radiological Society of Europe, and Dutch Mesenteric Ischemia Study group clinical guidelines on the diagnosis and treatment of patients with chronic mesenteric ischaemia. United European Gastroenterol J. 2020;8(4):371-395.
27. Umphrey H, Canon CL, Lockhart ME. Differential diagnosis of small bowel ischemia. Radiol Clin North Am 2008;46:943-52, vi-vii.
28. Cudnik MT, Darbha S, Jones J, et al. The diagnosis of acute mesenteric ischemia: A systematic review and meta-analysis. Acad Emerg Med. 2013;20(11):1087-1100.
29. Kirkpatrick ID, Kroeker MA, Greenberg HM. Bipahsic CT with mesenteric CT angiography in the evaluation of acute mesenteric ischemia: initial experience. Radiology 2003; 229 (1) : 91-98.
30. Lee R, Tung HK, Tung PH et al. CT in acute mesenteric ischaemia. Clinical Radiol 2003; 58 (4): 279-287.
31. Chou CK, Mak CW, Tzeng WS et al.CT of small bowel ischemia. Abdom Imaging 2004;29(1):18–22.
32. Lourenco PDM, Rawski R, Mohammed MF et al. Dual-Energy CT Iodine Mapping and 40-keV Monoenergetic Applications in the Diagnosis of Acute Bowel Ischemia. AJR Am J Roentgenol. 2018;211(3):564-570.
33. Mazzei MA, Gentili F, Volterrani L. Dual-Energy CT Iodine Mapping and 40-keV Monoenergetic Applications in the Diagnosis of Acute Bowel Ischemia: A Necessary Clarification. AJR Am J Roentgenol. 2019;212(3):W93-W94.
34. Gopee-Ramanan, Prasaanthan & Patlas, Michael & Pindiprolu, Bharadwaj & Katz, Douglas. (2019). Utility of biphasic multidetector computed tomography in suspected acute mesenteric ischemia in the emergency department. Emergency Radiology. 26. https://doi.org/10.1007/s10140-019-01698-9.
35. Lily Y Kernagis, Marc S. Levine, Jill E. Jacobs. Pneumatosis intestinalis in patients with ischemia: correlation of CT findings with viability of the bowel. AJR Am J Roentgenol 2003 180:3, 733-736
36. Gans SL, Stoker J, Boermeester MA. Plain abdominal radiography in acute abdominal pain; past, present, and future. Int J Gen Med. 2012;5:525-533.
37. Oldenburg WA, Lau LL, Rodenberg TJ. Acute mesenteric ischemia: a clinical review. Arch Intern Med. 2004;164(10):1054-1062.
38. Wolf EL, Sprayregen S, Bakal CW. Radiology in intestinal ischemia. Plain film, contrast, and other imaging studies. Surg Clin North Am. 1992;72(1):107-124
39. AbuRahma AF, Stone PA, Srivastava M, et al. Mesenteric/celiac duplex ultrasound interpretation criteria revisited. J Vasc Surg. 2012;55(2):428-436 e426; discussion 435-426.
40. Laissy JP, Trillaud H, Douek P. MR angiography: noninvasive vascular imaging of the abdomen. Abdom Imaging. 2002;27(5):488-506.
41. Meaney JF, Prince MR, Nostrant TT, Stanley JC. Gadolinium-enhanced MR angiography of visceral arteries in patients with suspected chronic mesenteric ischemia. J Magn Reson Imaging. 1997;7(1):171-176.
42. Acosta S,Björck M.Modern treatment of acute mesenteric ischaemia.Br J Surg 2014;101(1):e100–e108.
43. Klar E, Rahmanian PB, Bücker A, Hauenstein K, Jauch KW, Luther B. Acute mesenteric ischemia: a vascular emergency. Dtsch Arztebl Int 2012;109(14):249–256.
44. M.L. Schermerhorn, K.A. Giles, A.D. Hamdan, et al. Mesenteric revascularization: management and outcomes in the United States, 1988-2006 J Vasc Surg, 50 (2) (2009), pp. 341-348
45. Z.M. Arthurs, J. Titus, M. Bannazadeh, M.J. Eagleton, S. Srivastava, T.P. Sarac, et al. A comparison of endovascular revascularization with traditional therapy for the treatment of acute mesenteric ischemia J Vasc Surg, 53 (3) (2011), pp. 698-704 discussion-5
46. R.J. Beaulieu, K.D. Arnaoutakis, C.J. Abularrage, et al 3rd Comparison of open and endovascular treatment of acute mesenteric ischemia J Vasc Surg, 59 (1 2014), pp. 159-164
47. Hollingshead M,Burke CT,Mauro MA,Weeks SM,Dixon RG,Jaques PF. Transcatheter thrombolytic therapy for acute

mesenteric and portal vein thrombosis.J Vasc nterv Radiol 2005;16(5):651–661.
48. Y. Rits, G.S. Oderich, T.C. Bower, D.V. Miller, L. Cooper, J.J. Ricotta 2nd, et al. Interventions for mesenteric vasculitis J Vasc Surg, 51 (2) (2010), pp. 392-400 e2
49. Kougias P, Huynh TT, Lin PH. Clinical outcomes of mesenteric artery stenting versus surgical revascularization in chronic mesenteric ischemia. Int Angiol 2009;28:132-7.