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REVIEW

Computed Tomography Imaging of Acute Mesenteric Ischemia for Interventional Radiology

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Abstract:
Acute mesenteric ischemia is a life-threatening condition. A comprehensive approach involving a multidisciplinary team to review patient background, clinical history, physical examination, laboratory data, and imaging examination for respective diagnosis of superior mesenteric arterial occlusion, nonocclusive mesenteric ischemia, and superior mesenteric venous occlusion is essential. The most important imaging modality is computed tomography, which is used for diagnosis and for directing therapeutic strategy (e.g., endovascular revascularization, surgical bowel resection, or conservative management). Computed tomography image findings can support triaging of irreversible transmural bowel necrosis compared with reversible ischemic change with reperfusion. In this review article, the computed tomography imaging findings specifically associated with the pathophysiology of superior mesenteric arterial occlusion, nonocclusive mesenteric ischemia, and superior mesenteric venous occlusion are reviewed.

Keywords:
computed tomography, mesenteric ischemia, acute mesenteric arterial occlusion, acute mesenteric venous occlusion, nonocclusive mesenteric ischemia

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Introduction

Acute mesenteric ischemia (AMI), such as superior mesenteric arterial occlusion (SMAO), nonocclusive mesenteric ischemia (NOMI), and superior mesenteric venous occlusion (SMVO), is a life-threatening condition with a high mortality rate; due to potentially massive bowel necrosis, bacterial translocation, sepsis, and multiple organ failure, AMI dramatically increases with the duration of symptoms [1, 2]. A recently published worldwide survey of the literature found that although diagnosis is relatively straightforward, delayed diagnosis is common [3]. Therefore, prompt and accurate radiological diagnosis is crucial for management of patients with AMI so that the team of emergency physicians, gastroenterologists, surgeons, and radiologists are well informed to discuss therapeutic strategies, including endovascular treatment and surgery by reviewing imaging findings in combination with laboratory data and physical examination.

CT plays a pivotal role in diagnosing AMI by revealing vascular abnormality and bowel ischemia. Although angiography was traditionally regarded as reference standard [4], nowadays, it is performed in a therapeutic procedure [5]. CT is more widely available and has faster scanning time with high sensitivity and specificity [6, 7]. The CT imaging findings specifically related to the pathophysiology of SMAO, NOMI, and SMVO will be reviewed.

Epidemiology, Clinical Manifestation, Physical Examination and Laboratory Tests

AMI is less common than other cardiovascular diseases, such as stroke and myocardial infarction [8]. In Europe, AMI accounts for approximately 1% of acute abdominal pain and 0.05% of hospital admissions and has a prevalence of 6.0-7.3/100,000 person-years [9]. SMAO (65%) is the most common cause of AMI, followed by SMVO (28%) and NOMI (7%) (**Table 1**) [10].

Due to the frequent presence of comorbidities, the incidences of SMAO and NOMI increase with age. Although the development of endovascular therapy to allow bowel re-

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Table 1. Category of Mesenteric Ischemia.

	SMAO	NOMI	SMVO
Causes	Thrombosis, embolism, dissection	Vasospasm	Thrombosis
Onset	Acute	Acute	Subacute
Risk factor	Arrythmia, cardiac infarction, atherosclerosis, etc.	Hypovolemia, postsurgical status, hemodialysis, etc.	Portal hypertension, coagulopathy, etc.
Endovascular treatment	Thrombolysis, thrombectomy	Vasodilator infusion	Thrombolysis, thrombectomy
Surgery	Bowel resection	Bowel resection	Bowel resection

SMAO, superior mesenteric arterial occlusion; SMVO, superior mesenteric venous occlusion; NOMI, non-occlusive mesenteric ischemia

vascularization is expected to increase survival in patients with SMAO [8, 9, 11], the mortality rates remain high (59%-73%).

The nonspecific symptoms of various severity are the main challenges in diagnosing AMI. The sudden onset of severe abdominal pain is a typical clinical manifestation of SMAO [12]. This is consistent with the results of laboratory tests where leukocytosis is detected in over 90% and lactic acidosis in 88% of patients [13]. However, although helpful, the results of laboratory tests are not diagnostic because the findings are nonspecific, in that values may be elevated in other diseases and, in many cases, remain within the normal range despite the patient having AMI [14].

Imaging Modalities and Strategy for Diagnosing Acute Mesenteric Ischemia

Imaging is an important addition to physical examination and laboratory data for diagnosing AMI. The timely and accurate diagnosis allows potential rescue of ischemic bowel, avoidance of short bowel syndrome, and improved quality of life for the patient. CT imaging can generally be obtained promptly due to wide availability, is convenient to perform, and has excellent diagnostic performance; thus, it is a highly suitable modality for diagnosing AMI [15]. In addition to allowing diagnosis of vascular and bowel abnormalities, CT can also allow exclusion of other life-threatening conditions such as bowel obstruction, gastrointestinal perforation, intraabdominal abscess, and aortic dissection.

MRI is an alternative modality that can be used to delineate ischemic bowel [16]. However, MR examinations are less likely to be performed in acute settings due to the longer examination time, and limited image resolution may mean that distal arterial emboli are not shown [15, 17].

A study of a small series of patients reported that evaluation of flow velocities in the SMA using duplex ultrasound had a high negative predictive value for diagnosing proximal occlusive AMI. However, ultrasound is highly operator-dependent and cannot be used to detect distal emboli [18].

Although conventional angiography has long been the reference standard in diagnosing AMI and is still considered a gold standard for diagnosing NOMI [4], it has nowadays been supplanted by CT angiography (CTA) for diagnosis of AMI. Thus, angiography is frequently performed along with endovascular treatment.

CT Scanning Technique

Obtaining both nonenhanced and biphasic contrast-enhanced CT images is essential for diagnosing AMI. To obtain the latter, a bolus of 100-150 ml (600 mgI/kg) of iodine contrast media is injected at a rate of 2.5-4 ml/s via the cubital vein, and the arterial phase is scanned at a delay of 25-30 s and the phase at a delay of 60-70 s. The abdominopelvic CT scanning is performed from the level of the diaphragm to the ischial tuberosity to allow checking for other urgent abdominal conditions. In addition, it is common for embolism to occur in other organs in patients with SMAO (i.e., splenic and renal infarction, among others) [6], and other arteries may be affected with vasospasm in NOMI (i.e., the celiac and renal arteries) [19]. To be able to detect small thrombus and embolism and allow MPR and MIP or volume rendering (VR), the imaging slice thickness below 1 mm is preferable. Recently, iterative or deep-learning-based reconstruction is often used to remove image noise and allow reduction of the radiation dose [20]. MPR, MIP, and VR images are helpful for the assessment of vascular anatomy, emboli, and vasospasm in a manner similar to conventional angiography [21, 22]. There are several different techniques—dual source, kVp switching, dual-layer spectral detector, and split filter—available for performing dual-energy CT imaging to produce high- and low-energy datasets. The low keV images are particularly helpful wherein there will be increased contrast enhancement to assist in the evaluation of the bowel wall and abdominal vessels, or alternatively for the purposes of performing CTA, the amount of contrast agent can be reduced. Furthermore, the dual-energy dataset can be used to generate parametric images, particularly iodine map, which allows a quantitative measurement of bowel wall enhancement [23].

CT Imaging Findings

CT imaging findings in AMI comprise primary mesenteric vessel abnormalities and secondary subsequent bowel ischemia. The vessel abnormalities depend on each etiology (**Table 2**), and imaging findings regarding the bowel are different between arterial and venous ischemia (**Table 3**).

Vascular abnormalities

On noncontrast-enhanced CT, acute emboli, thrombus, and coagulated false lumen in dissection demonstrate higher

Table 2. Vascular Abnormalities in Mesenteric Ischemia on CT Imaging.

	Non-contrast	Contrast
SMAO	Smaller SMV sign	Luminal filling defect of the SMA (thrombosis and embolism)
	High dense in the SMA (thrombosis and embolism)	Intraluminal flap in the SMA (dissection)
	Enlarged SMA diameter and fat stranding surrounding SMA (dissection)	
NOMI	Reduced SMA diameter (comparing previous baseline CT images are helpful)	In addition to non-contrast scan, peripheral branches evaluation, MIP, and VR is available.
SMVO	High dense in the SMV	Luminal filling defect of SMV surrounded by rim-enhanced venous walls

MIP, maximum intensity projection; SMA, superior mesenteric artery; SMAO, superior mesenteric arterial occlusion; SMV, superior mesenteric vein; SMVO, superior mesenteric venous occlusion; NOMI, non-occlusive mesenteric ischemia; VR, volume rendering

Table 3. Bowel Wall Abnormalities in Mesenteric Ischemia on CT Imaging.

	Imaging findings	Clinical significance
Wall thickness		
Thickening	Disproportionate larger thickness of the bowel wall compared with bowel distension	Reperfusion in SMAO, NOMI Congestion in SMVO
Thinning	Too thin to identify the normal bowel wall and evaluate attenuation or enhancement (paper-thin wall)	Loss of tissue volume, muscular tone due to injured intramural nerves in SMAO, NOMI
Attenuation on non-contrast CT		
Increased	High attenuating bowel wall compared to the normal bowel segments (10–20 HU)	Hemorrhagic necrosis
Decreased	Low attenuating submucosal between high attenuating mucosa and muscularis propria commonly accompanied with wall thickening (target sign)	Reperfusion in SMAO, NOMI Congestion in SMVO Others (e.g., inflammation, autoimmune disease, etc.)
Gas	Presence of gas in the bowel wall (pneumatosis intestinalis)	Transmural bowel necrosis in some cases Other benign causes (e.g., pulmonary disease, medication, etc.)
Enhancement on contrast-enhanced CT		
Increased	Strong enhancement compared with the normal bowel	Reperfusion in SMAO, NOMI Congestion in SMVO
Decreased or absent	Weak or lack of enhancement compared with the normal bowel	Decreased or interrupted blood supply indicating various degrees of bowel necrosis

SMAO, superior mesenteric arterial occlusion; SMVO, superior mesenteric venous occlusion; NOMI, non-occlusive mesenteric ischemia

density than the circulating blood due to condensed red blood cells giving rise to higher iron concentration [24]. Narrowing window width improves visualization of high-density intravascular thrombus (**Fig. 1**). Moreover, observation of a smaller SMV is known to be an indirect imaging sign suggesting a diagnosis of SMAO with 70% sensitivity and 99.2% specificity (**Fig. 2**). However, the phenomenon is observed in other conditions (i.e., shock, hypovolemia, end-stage cancer, heart failure, etc.) [25]. In some cases of solitary SMA dissection, an enlarged SMA diameter and increased density around the SMA are observed on noncontrast CT [26].

Contrast-enhanced CT images are essential for demonstrating intraarterial pathologies that provide higher confidence in diagnosing SMAO when present (**Fig. 2** and **3**). Additionally, in isolated SMA dissection, a flap may be identified in the lumen of the SMA.

In NOMI, arterial bowel ischemia arises due to vasospasm of the SMA rather than arterial occlusion, and therefore, there is no imaging evidence of mechanical obstruction. SMA diameter is significantly smaller in the patients with NOMI than in the control group (6.0 mm vs. 7.6 mm; $P < 0.01$) [27]. If comparison with previous CT images is available, they can provide a useful baseline for assessing whether there has been a reduction in the diameter of SMA (**Fig. 4**). The diameter of SMA is significantly smaller in the onset of NOMI (1.93 ± 1.1 mm; $P < 0.001$) [28]. Although the diameter of the SMA can be reasonably well evaluated even on noncontrast CT images, contrast-enhanced CT provides clear delineation even in the more peripheral branches and also allows MIP and VR reconstructions to be produced of the vascular anatomy that can be helpful to review (**Fig. 5**) [21, 29].

In SMVO, it is rare for thrombosis to be limited to the superior mesenteric vein and is likely to extend massively to the intrahepatic portal and splenic veins [9]. Similar to the

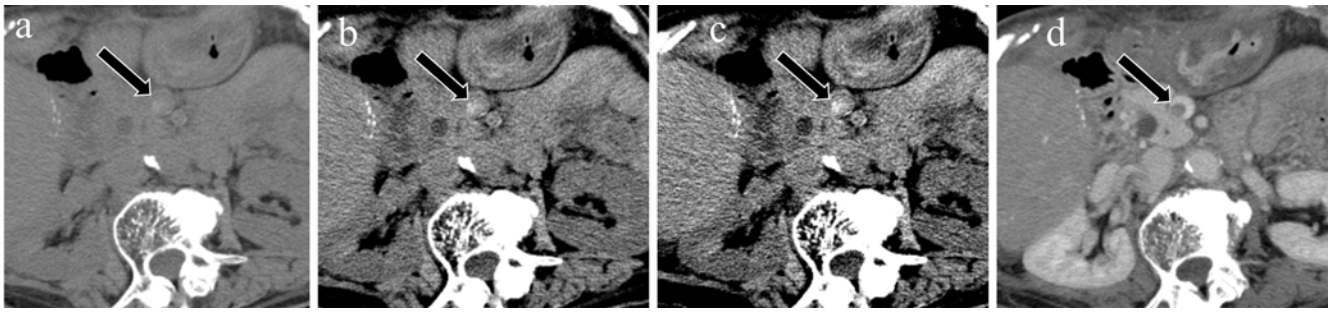

Figure 1. Optimal window setting to identify thrombus.
A 77-year-old female with mesenteric venous thrombosis. Noncontrast CT with default setting (window level [WL]: 40, window width [WW] 350) shows the subtle high dense lesion in the superior mesenteric vein (a: arrow). A narrow window setting (WL: 40, WW: 120) improves visualization of high attenuating thrombosis (b: arrow), and a narrower window setting (WL: 40, WW: 80) boosts contrast (c: arrow). Contrast-enhanced CT at the portal venous phase shows thrombosis as a filling defect in the superior mesenteric vein (d: arrow).

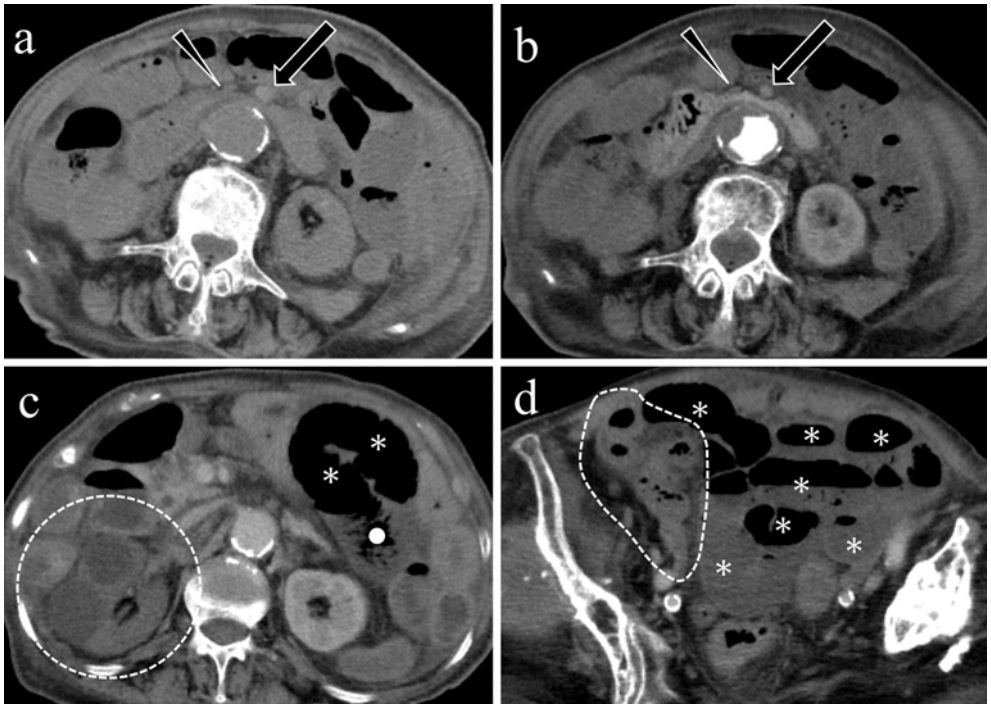

Figure 2. Superior mesenteric arterial occlusion due to thrombosis.
A 91-year-old female complained of abdominal pain and discomfort for over 3 days. Physical examination shows rebound tenderness in the whole abdomen. Noncontrast CT shows high dense area in the superior mesenteric artery (SMA) (a: arrow) and the superior mesenteric vein (SMV) with smaller diameter than that of SMA (a, b: arrowheads). Contrast-enhanced CT at the arterial phase shows lack of enhancement of SMA (b: arrow). Contrast-enhanced CT at delayed phase demonstrates lack of enhancement in the right kidney (c: circle). distal ileum (d: circle), paper-thin bowel wall appearance (c, d: asterisks), and pneumatosis (c: dot) in the ileum.

situation with SMAO, acute thrombus in the SMV and other veins is shown as a high-density lesion on noncontrast-enhanced CT (**Fig. 1**), and luminal filling defects surrounded by rim-enhanced venous walls can be seen on contrast-enhanced CT [5, 30]. Venous enlargement around the clot is suggestive of acute SMVO, whereas venous atrophy is suggestive of chronic SMVO. Furthermore, increased venous pressure can lead to engorgement of the upstream venous tributaries [31].

Bowel abnormalities

There are three stages by which acute bowel ischemia is categorized in terms of pathology [32]. Stage I is characterized by necrosis, erosions, ulcerations, edema, and hemorrhage localized to the mucosa and is reversible. In Stage II, necrosis extends into the submucosal and muscularis propria. This stage is potentially related to a risk of bowel stricture in late phase in a case of nonsurgical management [33]. In Stage III, there is transmural necrosis involving all layers

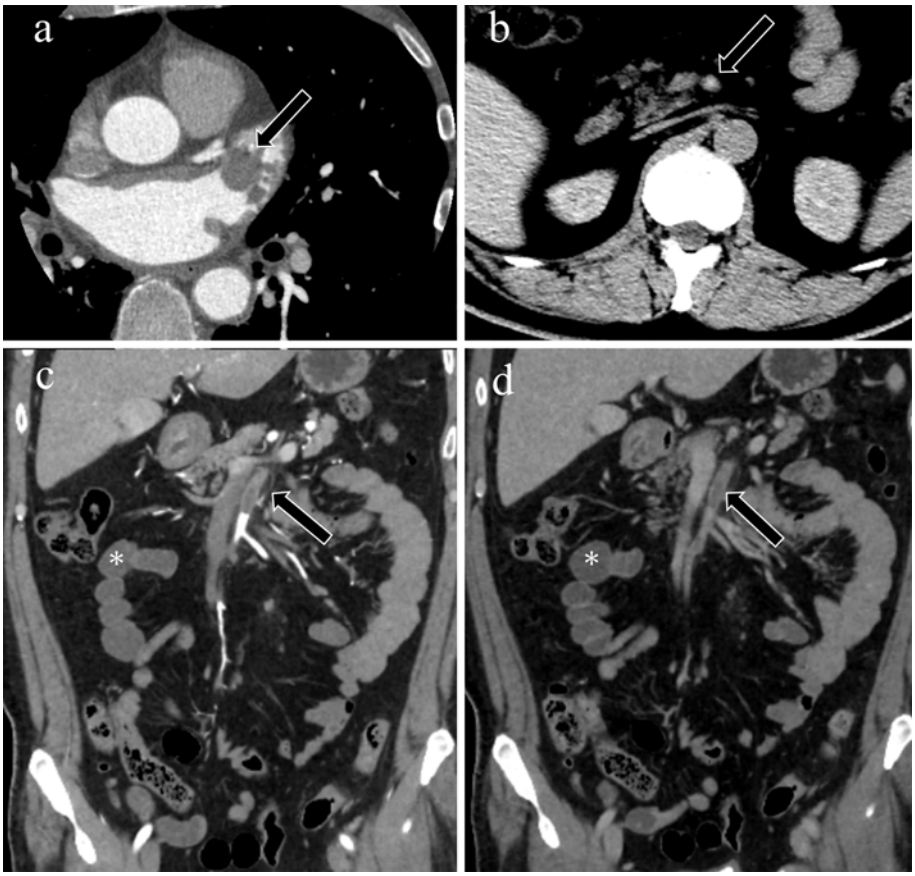

Figure 3. Superior mesenteric arterial occlusion due to thromboembolism deriving from intracardiac thrombus.
A 50-year-old male was treated with anticoagulation therapy for thrombus in the left atrial appendage. He complains of soreness in his abdomen and left lower extremity. Contrast-enhanced CT shows unenhanced mass, likely thrombus, in the left atrial appendage (a: arrow). Noncontrast CT (narrow window setting, WL: 40, WW: 120) shows high attenuating thrombus in the superior mesenteric artery (SMA) (b: arrow). Coronal contrast-enhanced CT images show filling defect in the SMA (c, d: arrows) and suspected decreased bowel wall enhancement (c, d: asterisks).

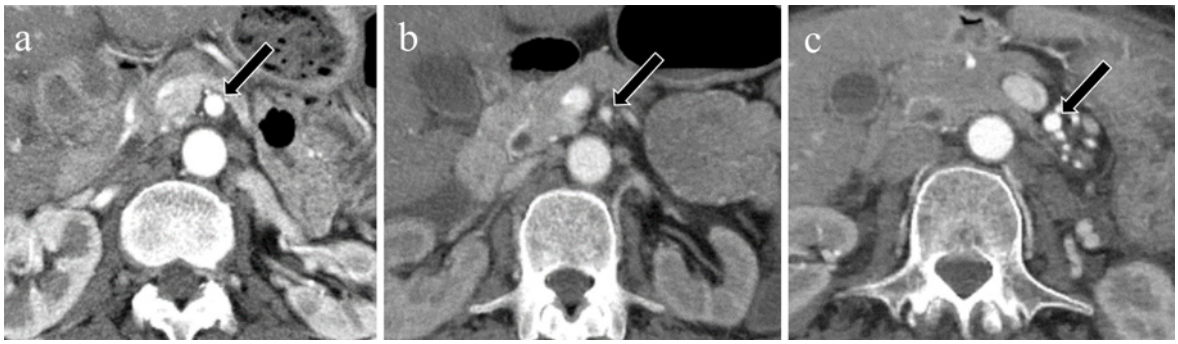

Figure 4. Diameter of the superior mesenteric artery in nonocclusive mesenteric ischemia.
Nonocclusive mesenteric ischemia in a 53-year-old female. The baseline diameter of the superior mesenteric artery was 8 mm on contrast-enhanced CT 2 years ago (a: arrow). The superior mesenteric artery diameter reduced to 5 mm at the onset of nonocclusive mesenteric ischemia (b: arrow). The diameter of the superior mesenteric artery recovered to 7 mm 1 week later (c: arrow).

and high associated mortality.

CT imaging findings of ischemic bowel wall can be categorized as changes in thickness, attenuation on noncontrast-enhanced images, and degree of bowel enhancement on contrast-enhanced images (**Table 3**). However, image appearance may be drastically changed in individual patients depending on duration from onset, severity, and the success of reperfusion treatment (**Fig. 6**).

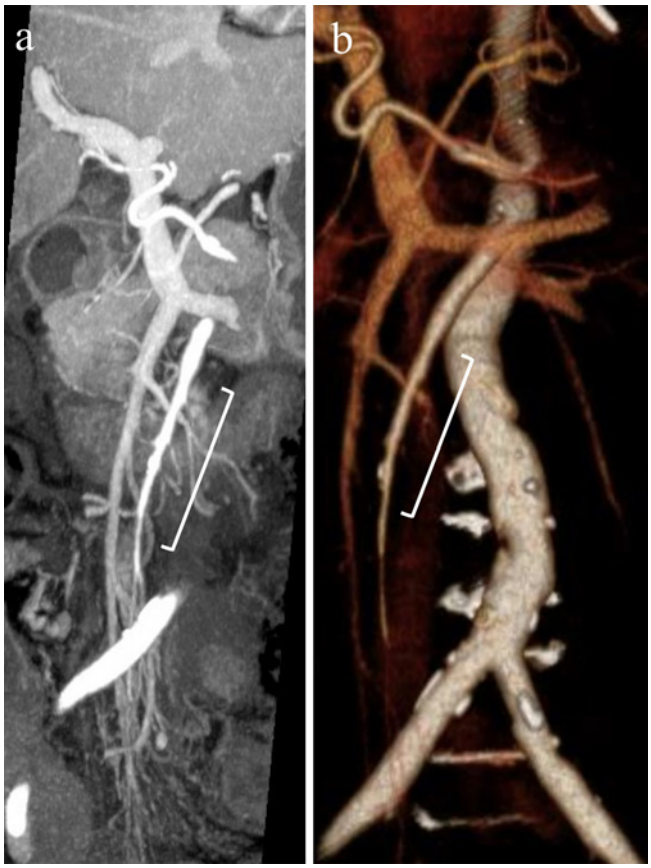

Figure 5. Maximum intensity projection (MIP) and volume rendering (VR) images.
Nonocclusive mesenteric ischemia in a 77-year-old female. MIP image demonstrates irregular margin of the superior mesenteric artery (SMA) (a). Similarly, VR image shows the spastic SMA (b).

Decreased enhancement indicates reduced or interrupted blood inflow to the bowel wall. A complete absence of enhancement indicates more severe ischemia (**Fig. 2** and **3**). Since evaluation of the degree of enhancement is subjective with only a moderate level of interobserver agreement reported [34], evaluation is best performed by comparison with the enhancement of the normal bowel. In the case of dual-energy CT, the monoenergetic image provides a useful reference to increase confidence level and a map of iodine concentration affords more objective analysis [35].

In evaluating the perfusion status of the bowel wall in patients with SMAO and NOMI treated with medication, endovascular treatment, or collateral blood supply, a thickened bowel wall with stratification formed by high-density inner layer indicating mucosa, low-density middle layer indicating edematous submucosa, and high-density outer layer indicating muscularis propria on contrast-enhanced CT is referred to as a target sign and may indicate favorable prognosis [36]. In the case of SMAO, if following treatment ischemia is limited to the mucosa, it is likely that cure has been achieved, whereas if ischemic injury reaches the submucosal layer and muscularis propria, bowel stricture caused by severe fibrosis is the likely sequelae in the late phase of the condition.

If there is a failure of reperfusion in treatment of SMAO and NOMI, then ischemia of the bowel wall progresses to transmural necrosis. Particularly, hemorrhagic necrosis, paper-thin bowel wall, and pneumatosis are indicative imaging findings for penetrating bowel necrosis. Hemorrhagic bowel necrosis will appear as increased density in the bowel wall on images obtained without contrast [37]. Therefore, it is essential to obtain noncontrast images in diagnosing AMI, because when CT images are obtained with contrast, it is not possible to distinguish between high-density bowel wall and contrast enhancement. Using dual-energy CT, it is possible to obtain a virtual noncontrast CT and iodine density map to diagnose hemorrhagic infarction [38]. If the bowel wall appears paper-thin, then this is highly suggestive of irreversible transmural bowel necrosis requiring surgical resection [38].

Pneumatosis intestinalis is defined as the presence of gas in the bowel wall and can be caused by benign and life-threatening conditions such as bowel ischemia (**Fig. 7**). Decreasing the window level and widening window width when viewing the CT images can assist in diagnosing whether pneumatosis intestinalis is present (**Fig. 7**). When pneumatosis intestinalis is due to a benign cause, such as pulmonary disease, patients usually do not complain of having any symptoms or report only mild abdominal discomfort, whereas severe abdominal pain is common in AMI [39]. Pneumatosis intestinalis and mesenteric venous and portal venous gas are predictors of transmural ischemic necrosis in AMI [40, 41]. Pneumatosis may indicate irreversible bowel necrosis, but some patients in whom bowel loops show these imaging findings may recover without surgery, especially in the case of NOMI. Due to these reasons, a comprehensive approach in which clinical history, physical examination, and laboratory test results are reviewed in combination with imaging findings is required for the diagnosis and management of AMI.

The imaging findings and clinical course are different in the case of venous occlusion. In particular, bowel wall thickening, mesenteric congestion and fluid, pneumatosis, and ascites indicate a diagnosis of SMVO [42]. Furthermore, mesenteric congestion and fluid may be more frequent in venous than in arterial occlusion due to the congestive nature of the disease [6, 32].

Superior Mesenteric Arterial Occlusion (SMAO)

The etiologies of SMAO are embolism, thrombosis, and dissection. Patient background, clinical history, and imaging findings all need to be considered in making diagnosis, which is important because treatment strategy is dependent on etiology.

Embolism commonly originates from an intracardiac blood clot associated with atrial fibrillation and valvular disease (**Fig. 3**), whereas an oncotic embolism due to cardiac myxoma and sarcoma or a mycotic embolism due to septic vegetation in the heart is uncommon [43, 44]. Embolism might also derive from aortic atheromatous plaques, and hypercoagulable status in diabetic ketoacidosis [45] and coro-

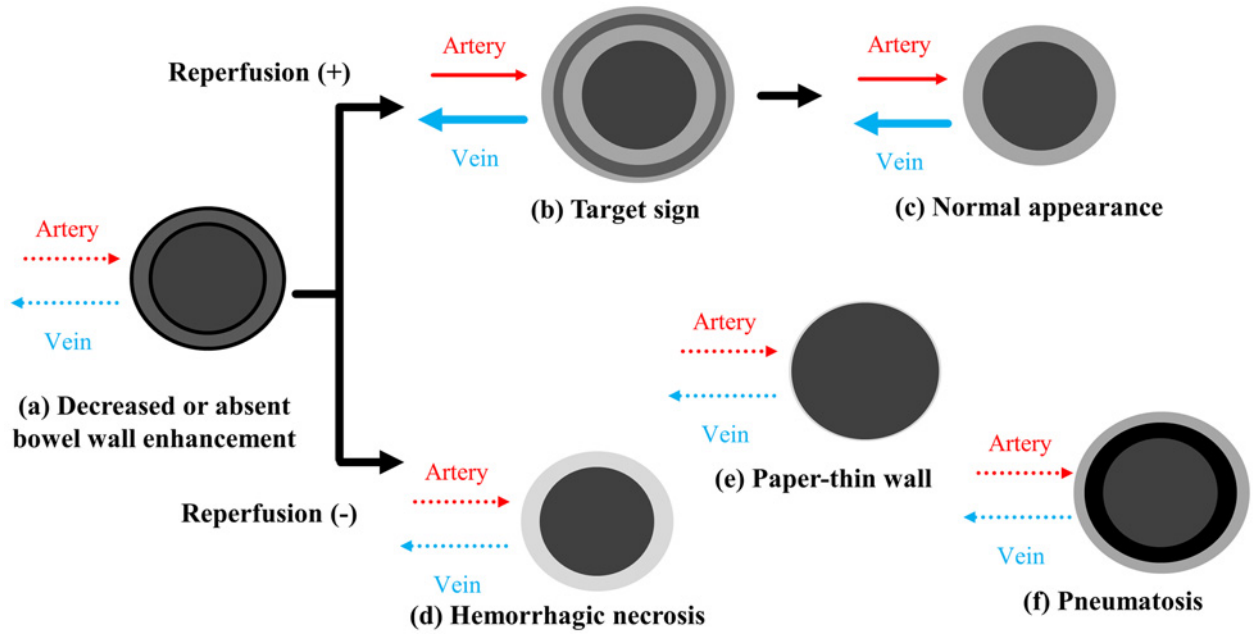

Figure 6. Clinical course of the arterial bowel ischemia.
Decreased or absent enhancement neither thickening nor thinning are observed in the ischemia bowel in early stage (a). In case of reperfusion, stratified bowel wall thickening occurs by increased permeability resulting in submucosal edema (b), the ischemic bowel is normalized if ischemia is mild (c) but bowel obstruction due to stricture if ischemic change involves muscularis propria in distant period. Without reperfusion, ischemic bowels fall into transmural necrosis showing worrisome imaging findings including hemorrhagic necrosis (d), paper-thin wall (e), and pneumatosis (f).

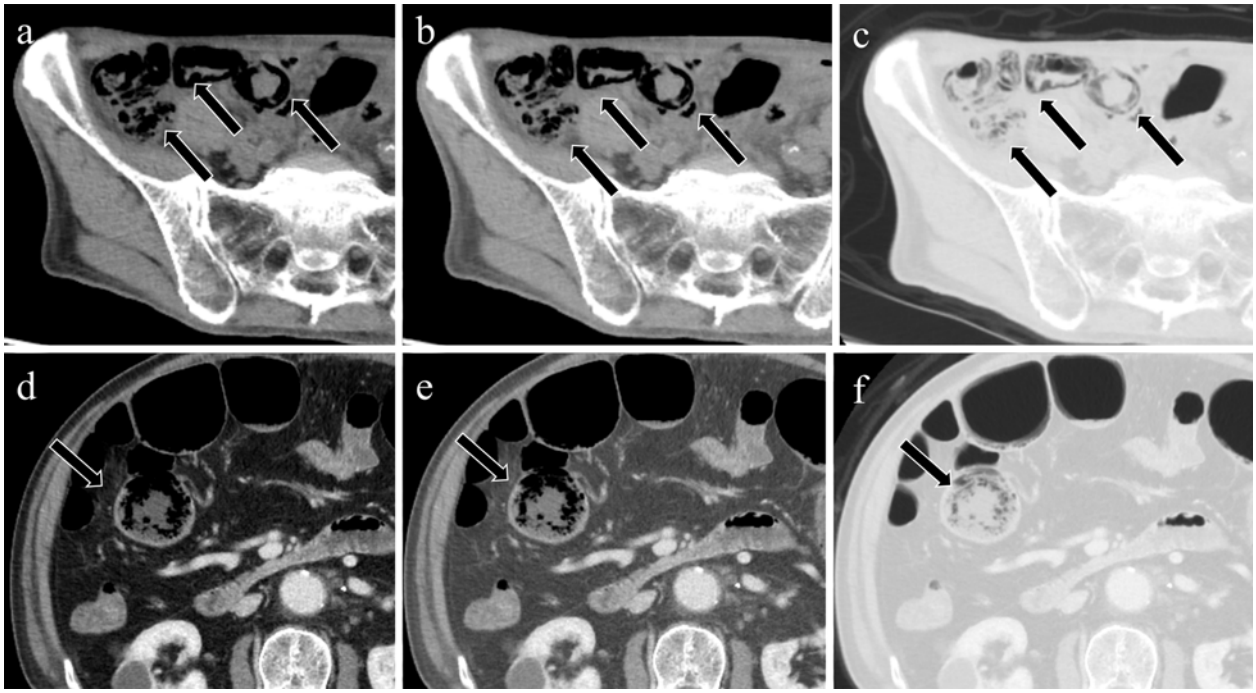

Figure 7. Optimal window setting to identify intestinal pneumatosis.
CT images of idiopathic pneumatosis intestinalis in a 70-year-old male and pneumatosis intestinalis associated with nonocclusive mesenteric ischemia in a 78-year-old male are shown different window setting (default window setting [WL 40 WW 350] (a, d), adjusted window setting [WL 0 WW 450] (b, e), and lung window setting [WL −500 WW 1,500] (c, f)). Adjusted window (b, e) and lung window settings (c, f) are better to describe pneumatosis intestinalis and contribute to analyzing internal texture than the default window settings.

navirus disease 2019 (COVID-19) infection have been reported as causes of embolism in SMAO [46]. In between 20% and 70% of patients with embolic SMAO, there is synchronous embolism in other organs SMA thrombosis pa-

tients affected, such as the lower limbs (27%), kidney (18%), and brain (10%) (**Fig. 2**) [47]. Sudden onset of acute abdominal pain is a characteristic clinical manifestation of embolic SMAO. Emboli may obstruct the proximal trunk at a distance of 3 to 10 cm from the SMA orifice, which may involve the middle colic artery. Smaller emboli may occlude the distal main trunk of or its peripheral branches [48].

Superior mesenteric arterial thrombosis attributed to atherosclerotic change is other etiology for SMAO [12]. Ruptured unstable atherosclerotic plaque is a trigger of SMAO onset. Prior to this stable atherosclerotic plaque may produce abdominal pain that gradually worsens depending on the collateral blood supply. The development of compensatory collateral vessels may cause major or total SMA occlusion and produce chronic mild symptoms such as postprandial abdominal angina during increased intestinal blood flow demand [5]. Compared with embolism, thrombosis typically involves the more proximal portion of SMA (i.e., 2 to 3 cm from the orifice). A preexisting atherosclerotic plaque may appear as arterial wall thickening accompanied with calcification, whereas for a ruptured unstable plaque, complete occlusion with high dense fresh thrombus is observed on noncontrast-enhanced CT images [48]. Mogi et al. [49] categorized SMA thrombosis into three types and proposed the so-called golden times in which it is possible for ischemic bowels to be rescued by thrombolysis. The three types are as follows: Type A where occlusion is present in the segment from the SMA orifice to the bifurcation of the middle colic artery (5 h), Type B where occlusion is present in the segment from the bifurcation of the middle colic artery to the bifurcation of the ileocolic artery (24 to 48 h), and Type C where occlusion is present in the more peripheral segment (several days) [49].

Isolated dissection of SMA, predominantly seen in middle-aged males, is a rare cause of SMAO [12]. Regardless of whether bowel ischemia is present, because the visceral nerve plexus is stimulated, sudden onset of severe abdominal pain is the main clinical manifestation [50]. Different to embolic and thrombotic SMAO, conservative management is successful even in most symptomatic cases of dissection SMAO. Persistent pain (4%) and bowel necrosis (1%) are infrequent [51]. Contrast-enhanced CT that allows evaluation of blood flow in the true and pseudo-lumen and bowel ischemia is an essential decision-making tool prior to endovascular or surgical intervention, and endovascular treatment using a stent is generally applied in cases where there is bowel malperfusion. Some classifications based on the condition of the pseudo-lumen on CT are advocated [52, 53].

Nonocclusive Mesenteric Ischemia (NOMI)

NOMI is characterized by mesenteric ischemia without mechanical causes such as embolism, thrombosis, dissection for the mesenteric artery, or venous occlusion and was first reported as segmental small bowel necrosis without mechanical obstruction in the main trunk of the SMA in a heart failure patient by Ende in 1958 [54].

The pathophysiology of NOMI is splanchnic hypoperfusion due to vasoconstriction by various conditions, namely, ischemic heart disease, arrhythmia, burn, diabetes, dehydration, pancreatitis, postsurgical status, hemodialysis, catecholamine, digitalis, and diuretics [55]. In systemic hypovolemia or hypoperfusion, intrinsic vasoconstrictors such as catecholamine, vasopressin, and angiotensin are secreted and catecholamine is administrated extrinsically to preserve perfusion of the vital organs, such as the brain, heart, and lung [6]. However, there is simultaneous sacrifice of blood flow to the splanchnic arteries, including the SMA. Despite the similar imaging findings of bowel involvement that have been reported following COVID-19 infection [56], given that thrombi are observed in the systemic arterial and venous systems and portal system, this condition is likely different from NOMI.

Due to the risk factors described above, NOMI generally affects especially hospitalized elderly patients. However, walk-in patients without any remarkable past history may present with NOMI due to dehydration or undiagnosed comorbidities. Potential symptoms may include acute abdominal pain, abdominal distension, nausea, and diarrhea, and physical examination may reveal muscular defense, hypotension, fever, and decreased bowel sounds, but a specific diagnostic clue has not been identified [57].

Angiography was classically recognized as a gold standard for diagnosis of NOMI, and the following specific angiographic findings were reported by Siegelman et al.: (ⅰ) narrowing at the origins of multiple branches of the SMA, (ⅱ) irregularities in intestinal branches, (ⅲ) spasm of arcades, and (ⅳ) impaired filling of intramural vessels [4]. Subsequently, to standardize interpretation of the angiogram and predict outcome, Minko et al. proposed a scoring system based on angiographic findings, including (ⅰ) SMA morphology, (ⅱ) contrast media reflux into the aorta, (ⅲ) contrast enhancement of the intestine, (ⅳ) distension of the intestine, and (ⅴ) time to portal vein filling [58]. As part of the angiography procedure therapy may be given in the form of intraarterial infusion of a vasodilator such as papaverine hydrochloride and prostaglandin that may improve patient outcome compared with conservative therapy [59, 60].

There have been major developments in CT imaging technology during the past two decades, and abdominopelvic CT is the modality of choice for diagnosing the cause of acute abdominal pain [61]. CT imaging has demonstrated similar vascular findings and has largely replaced angiography for diagnosing NOMI [62]. In particular, MIP and VR images are helpful for identifying focal narrowing in the main trunk and proximal segments of branches of the SMA [21], whereas CT imaging is limited to depicting vasospasms in the peripheral arteries, such as marginal arteries and vasa recta. As vasospasm of the SMA due to systemic hypovolemia or hypoperfusion generally involves other splanchnic arteries, ischemic change may occur in the territory of the celiac, inferior mesenteric, and renal arteries and this possibility should be looked for.

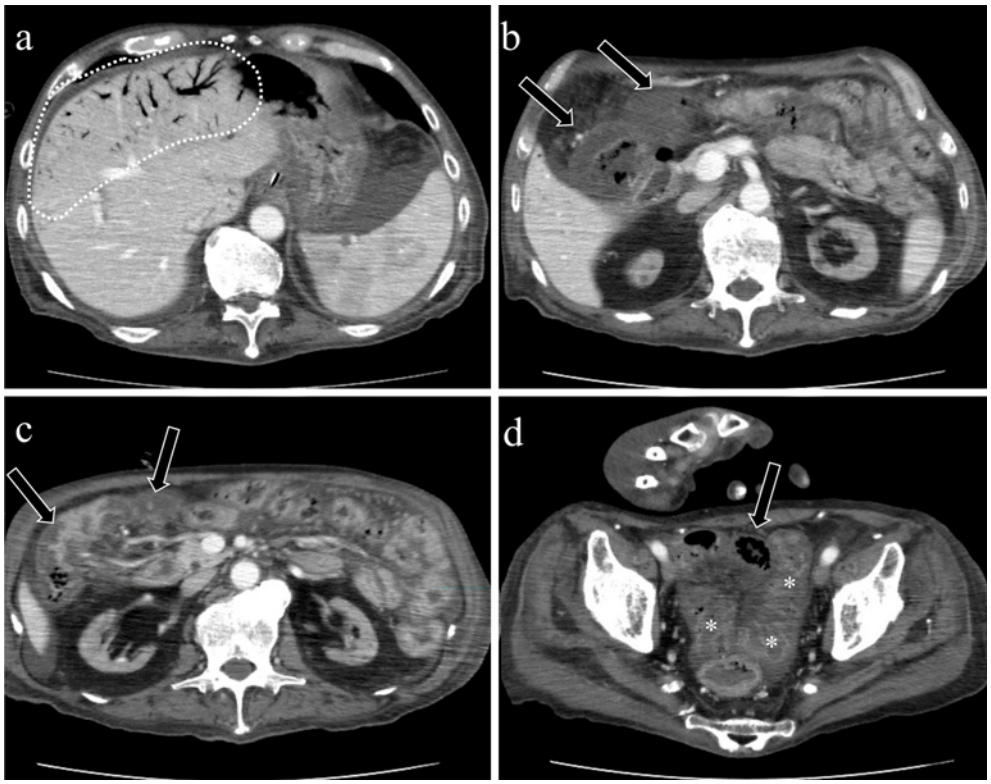

Figure 8. Various imaging findings in nonocclusive mesenteric ischemia.
A 72-year-old male who take hemodialysis for end-stage renal failure complained of hematochezia and nausea. Apparent branching gas in the liver, intrahepatic portal vein gas, is observed in the dorsal side of the liver (a: circle). Contrast-enhanced CT at the portal phase demonstrates irregular mucosal enhancement in the thickened gastric antrum and duodenal bulb (b: arrows), patchy remaining enhancement in the transverse colon (c: arrows), and decreased wall enhancement associated (d: asterisks) with pneumatosis in the small bowel (d: arrow). His symptom was naturally resolved without any treatment.

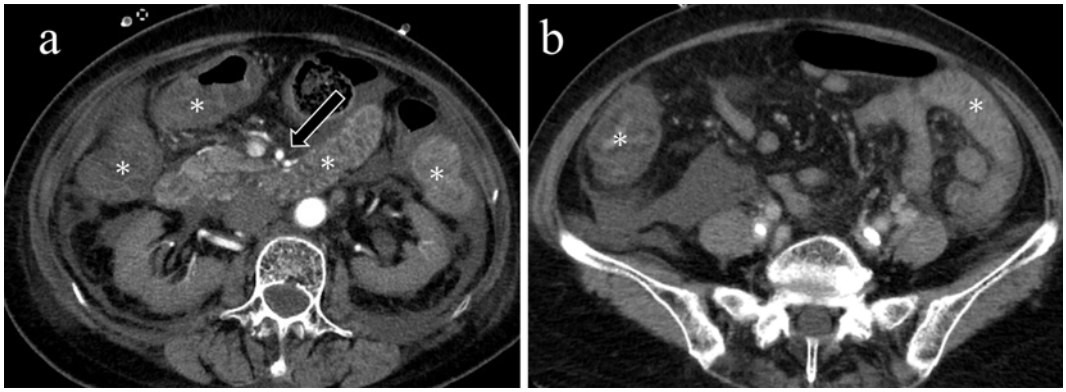

Figure 9. Reperfusion status in nonocclusive mesenteric ischemia.
A 72-year-old female underwent aortic arch replacement 3 days ago. She was managed in intensive care unit and her blood pressure decreased. The diameter is small but controversial due to the lack of baseline image (a: arrow). Massive edematous wall thickening is seen in the jejunum and ascending and transverse colon (a, b: asterisks). Laparoscopic examination shows no ischemic bowel.

To be able to depict vascular narrowing, imaging examinations should ideally be performed during the vasospasm. If angiographic and CT images are obtained after vanishing vasospasm, despite no diagnostic clue in the vascular pathology, in most symptomatic cases, bowel ischemic changes will nevertheless be observed on CT images. Imaging findings of bowel ischemia tend to be heterogenous, with ischemic bowel interposed between normal bowel (**Fig. 8**). Similar to SMAO, stratified bowel thickening in NOMI suggests reperfusion indicating a favorable prognosis (**Fig. 9**). On the contrary, unlike SMAO, bowel loops showing pneumatosis and portal vein gas in some patients with NOMI

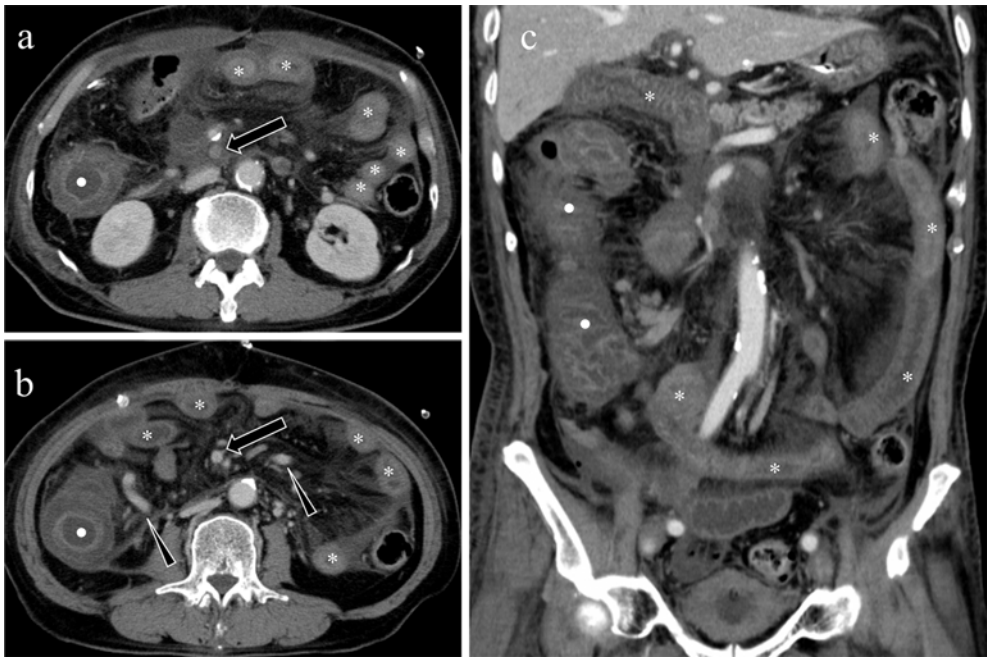

Figure 10. Superior mesenteric venous occlusion due to thrombosis.
A 76-year-old male who underwent subtotal stomach preserving pancreaticoduodenectomy for pancreatic adenocarcinoma 4 days ago. He was managed in an intensive care unit and laboratory investigation showed lactate acidosis. Contrast-enhanced CT demonstrates a filling defect in the proximal superior mesenteric vein (SMV) (a: arrow) whereas patent in the distal SMV (b: arrow) developed collateral (b: arrowheads). Massive, stratified wall thickening associated with mesenteric haziness is observed in the small bowel (a–c: asterisks) and ascending colon (a–c: dots).

may heal naturally without any surgery because mesenteric ischemia is temporary in NOMI.

Superior Mesenteric Venous Occlusion (SMVO)

SMVO is a less common type of AMI [63]. The SMV is more often the site of thrombus formation than the inferior mesenteric vein [64]. SMVO occurs more frequently in younger patients than in older ones with SMAO [5], and the mean age at presentation is between 45 and 60 years with slight incidence in males than in females [64]. Systemic hypercoagulability may be due to inherited disease such as Factor V Leiden mutation, prothrombin mutation, protein S deficiency, protein C deficiency, antithrombin deficiency, or antiphospholipid syndrome [5]. Recently, COVID-19 infection has also been associated with acute mesenteric thrombosis, including a documented case of SMVO related to hypercoagulability [65]. Use of oral contraceptives is also a risk factor especially in young women. Mechanical factors such as abdominal mass, pancreatitis, and postsurgical status appear to be associated with initial thrombus formation in the large veins, whereas systemic hypercoagulability leads to thrombosis that is initiated in the intramural venules, vasa recta, and venous arcades [66].

The most common symptom of SMVO is subacute disproportionate abdominal pain, which is typically seen in 90-100% of patients. Other gastrointestinal symptoms such as nausea, vomiting, abdominal distention, or occult fecal blood may also occur as early findings [67]. The bowel regions that are most involved are ileum and jejunum [68]. Even in those patients with an extensive clot burden, ischemia develops more gradually in patients with SMVO than in those with SMAO or NOMI. Furthermore, patients with SMVO typically do not suffer infarction unless there is extensive involvement of the upstream peripheral arcade or the vasa recta branches with the development of collateral venous drainage (**Fig. 10**) [69]. However, when occlusion of the mesenteric vein occurs acutely, there is not sufficient time for the formation of collateral vessels, resulting in bowel ischemia [70].

The distribution of the effects of SMVO is often initially segmental and slowly progressive, first affecting the smaller upstream veins before progressing into the SMV proper [7, 31]. Interestingly, SMVO with portal vein involvement has a lower risk of developing transmural infarction than SMVO that is isolated to the mesenteric vein, and SMVO of the more distal mesenteric veins tend to produce higher rates of bowel infarction than SMVO of the more proximal SMV and portal vein [64, 71]. It is important to recognize that in SMVO, the degree of bowel wall thickening, mesenteric congestion, and amount of ascites does not reflect the severity of ischemic bowel damage [5].

In general, the prognosis for survival of patients with SMVO who respond to conservative management has been reported to be as high as 93% at 3 years and is better in patients with SMAO and NOMI [72]. However, patient prognosis is often determined by the severity of the underlying disease, and long-term complications such as recurrence of

thrombosis and intestinal ischemic strictures can also develop, with variable incidences reported in only small cohort studies [73].

Conclusions

A comprehensive diagnostic approach involving a multidisciplinary team and employing patient background information, clinical history, physical examination, laboratory data, and, not least, the radiology report from imaging is essential for diagnosing and managing AMI. CT is considered the first-line imaging modality for diagnosis and a vital decision-making tool for developing therapeutic strategy. The CT imaging findings of AMI may be broadly classified into two types: vessel abnormalities and ischemia of the bowel wall. In SMAO and SMVO, mechanical occlusion is observed in SMA and SMV, respectively, whereas vasospasm of SMA is observed in NOMI. Moreover, in arterial ischemia in SMAO and NOMI, hemorrhagic infarction, paper-thin bowel wall, and intestinal pneumatosis highly suggest transmural bowel necrosis, and bowel wall thickening with stratification indicates reperfusion. In comparison, high-grade wall thickening with stratification is characteristic of venous ischemia.

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Shohei Chatani did a literature search for "Superior mesenteric venous occlusion (SMVO)."
Ryo Uemura wrote a draft of "CT scanning technique" and did a literature search for "CT scanning technique."
Yugo Imai did a literature search for "Superior mesenteric arterial occlusion (SMAO)."
Yuki Tomozawa wrote a draft of "Superior mesenteric venous occlusion (SMVO)."
Yoko Murakami wrote a draft of "Epidemiology, clinical manifestation, physical examination, and laboratory test" and did a literature search for "Epidemiology, clinical manifestation, physical examination, and laboratory test."
Akinaga Sonoda wrote a draft of "Superior mesenteric arterial occlusion (SMAO)."
Neil Roberts edited the manuscript as a native English speaker who is familiar with radiology.
Yoshiyuki Watanabe supervised the project.

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