Mesenteric ischaemia: an overview of diagnostic and therapeutic best practice

ABSTRACT

Mesenteric ischaemia is relatively uncommon, but is an important diagnosis to remember as it can lead to significant morbidity and mortality. Improvements in clinical imaging technology have made rapid diagnosis and treatment possible, but this relies on awareness of the condition. This article summarizes current best practice in diagnosis and management of mesenteric ischaemia to guide the clinician.

Mesenteric ischaemia is a condition caused by insufficient blood flow through the mesenteric vessels to meet the metabolic requirements of the bowel. It often presents with non-specific symptoms but, if suspected, can be readily identified on computed tomography by a number of recognized radiological signs. Early diagnosis may enable treatment to allow restoration of blood flow, but the diagnosis nevertheless carries significant risks of morbidity and mortality. Survival for patients with acute mesenteric ischaemia has improved over the last 50 years, but mortality rates remain high at 50–69% (Tilsed et al, 2016).

Pathophysiology and aetiology

Mesenteric ischaemia can be divided into occlusive and non-occlusive causes, with occlusive causes further subdivided into arterial and venous, depending on the location of vascular compromise. Mesenteric arterial occlusion can be acute or chronic and the result of embolism or thrombosis, whereas mesenteric venous occlusion is generally the result of thrombosis. The different subtypes of mesenteric ischaemia are shown in *Figure 1*.

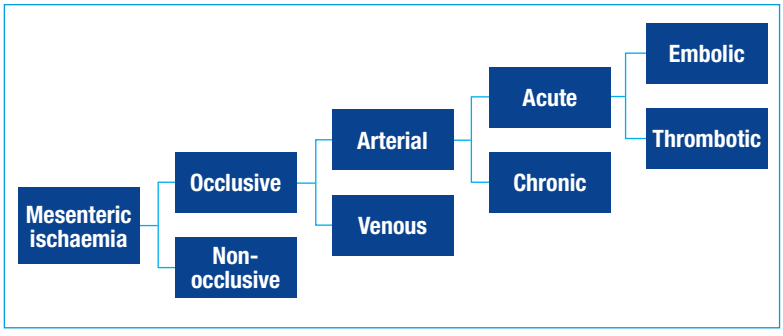

Figure 1. Categories of mesenteric ischaemia.

Mesenteric arterial embolism is usually cardiogenic, with possible aetiologies including valvular heart disease, arrhythmia, cardiac structural defects and recent myocardial infarction. Less commonly, emboli may originate from atherosclerotic ulcers or thrombosed aneurysms of the aorta. The superior mesenteric artery is the most commonly affected vessel as it has a shallow take-off angle from the aorta compared to other vessels. The central portion of the small bowel is most susceptible to embolic ischaemia as it receives its supply from the superior mesenteric artery but is furthest from collateral supply arising from the coeliac and inferior mesenteric artery. Vascular occlusion in this case is usually sudden, so there is insufficient time for an adequate collateral supply to develop if not already present. The situation may be aggravated by reactive vasospasm (Cappell, 1998).

Mesenteric arterial thrombosis is generally a chronic process caused by atherosclerosis, with the same risk factors as atherosclerosis elsewhere in the body, but (rarely) can also occur as a result of vasculitis. Occasionally a more acute presentation is seen with complete thrombosis of a previously stenosed vessel (often because of plaque rupture). This is generally only acutely symptomatic if multiple mesenteric vessels are involved because a rich collateral supply has had time to develop in chronic cases, and may give a more gradual onset of symptoms (Cappell, 1998). Whereas mesenteric arterial embolism is often in a fairly distal arterial vascular branch, thrombosis usually develops close to the origin of the vessel, leading to extensive bowel involvement (Cappell, 1998).

In mesenteric venous thrombosis, venous occlusion leads to localized venous hypertension and oedema in the bowel wall. Symptomatic mesenteric venous thrombosis is seen 20 times more frequently in the superior mesenteric vein than the inferior mesenteric vein as a result of higher calibre and flow (Cappell, 1998). Once the pressure in the bowel wall exceeds the pressure in the capillaries perfusing the bowel, arterial inflow is restricted and the bowel begins to become ischaemic.

Non-occlusive mesenteric ischaemia is a very different entity to the other types of mesenteric ischaemia. As the name suggests, there is no intraluminal obstruction to mesenteric blood flow, but instead flow is limited by ineffective cardiac contraction, systemic hypotension or

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Table 1. Aetiologies of mesenteric ischaemia

Pathological subtype	Aetiologies or risk factors
Mesenteric arterial embolism	■ Arrhythmia ■ Valvular heart disease ■ Structural heart defects ■ Post-myocardial infarction ■ Thoracic aortic atheromatous plaques or aneurysms
Mesenteric arterial thrombosis	■ Atherosclerosis ■ Vasculitis
Mesenteric venous thrombosis	■ Hypercoagulable states ■ Pancreatitis ■ Malignancy ■ Trauma ■ Liver disease – cirrhosis, portal hypertension
Non-occlusive mesenteric ischaemia	■ Hypotensive states, e.g. cardiogenic, septic, hypovolaemic shock ■ Arrhythmia ■ Mesenteric vasoconstrictive drugs

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vasoconstrictive drugs. Non-occlusive mesenteric ischaemia often affects watershed areas between vascular territories, as these areas are served by smaller vessels from two territories so are the first to suffer from ischaemia when multiple vascular territories are involved. The two main watershed areas are at the splenic flexure (Griffith's point – crossover between superior mesenteric artery and inferior mesenteric artery territories) and at the rectosigmoid junction (Sudeck's point – crossover between inferior mesenteric artery and internal iliac territories), although the clinical significance of the latter is questionable (Meyers, 1976).

Whatever the underlying cause, the resulting damage to the affected segment of bowel can range from reversible ischaemia to complete infarction, with subsequent necrosis and potentially perforation. The ischaemic bowel segment may undergo spastic contraction, giving the patient symptoms of vomiting and/or diarrhoea (Cappell, 1998). Although the gut can survive up to 12 hours with only 25% of its normal blood supply, infarction can occur within 6 hours if there is complete occlusion (Tilsed et al, 2016).

If bowel infarction does occur, the bowel releases toxins and the normal mucosal barrier starts to break down, allowing bacterial translocation into the systemic circulation potentially leading to septic shock (Sise, 2014). By the same mechanism of increased mucosal permeability, gas can pass from the bowel lumen into the bowel wall and portomesenteric venous system (Ho et al, 2007). If transmural necrosis occurs, the mucosal surface may be shed intraluminally, with associated haemorrhage, or the bowel wall may perforate leading to pneumoperitoneum and peritonitis (Sise, 2014).

Aetiologies for the different subtypes of mesenteric ischaemia are illustrated in *Table 1*.

Clinical history and examination

The clinical presentation for patients with mesenteric ischaemia varies depending on the underlying aetiology. In acute cases, patients often have severe generalized abdominal pain refractory to analgesia, and frequently nausea. If the bowel has infarcted and become gangrenous, the patient may present systemically unwell with signs of sepsis, peritonitis and per rectal bleeding. Chronic mesenteric ischaemia typically presents with abdominal pain shortly after mealtimes (abdominal angina), but can present with non-specific symptoms such as nausea or weight loss (Sise, 2014). Non-occlusive mesenteric ischaemia can be clinically silent as it often occurs in patients who are already critically unwell, e.g. ventilated on the intensive care unit (Tilsed et al, 2016).

Examination findings are classically non-specific and may be less impressive than the severity of pain the patient reports, with some patients having a soft and non-tender abdomen on examination (Kozuch and Brandt, 2005). However, if a patient presents late with established bowel infarction he/she may be peritonitic with signs of sepsis. Bowel sounds are variable and are not a reliable indicator. In elderly patients, non-specific features such as mental status change and tachypnoea are frequently seen (Kozuch and Brandt, 2005). Examination findings are usually insufficient to determine the aetiology of ischaemia, but there may be helpful clues from the history.

Investigation

Laboratory tests

Serological markers may be supportive of the diagnosis but are not diagnostic. Findings may include raised levels of serum lactate and/or inflammatory markers, but no single blood test is sensitive or specific enough to confirm or exclude the diagnosis (Tilsed et al, 2016). Imaging is required to confirm the diagnosis.

Radiology

Plain abdominal radiography has low sensitivity and poor specificity for mesenteric ischaemia, but may help to exclude alternative causes of abdominal pain such as obstruction or perforation (Smerud et al, 1990). Note, however, that the cardinal radiological features of these conditions, namely dilated bowel loops and pneumoperitoneum, can both also be seen in mesenteric ischaemia. If these features are seen in mesenteric ischaemia, it is generally once bowel has already infarcted (Tilsed et al, 2016).

Ultrasound may show non-specific signs such as bowel wall thickening and free fluid, but is not considered a useful test in the diagnosis of mesenteric ischaemia, particularly in the acute setting where prompt diagnosis is critical.

Multiphase computed tomography is the most useful diagnostic test in the acute setting, yielding a reported sensitivity from pooled primary studies of 93% and specificity of 96% (Tilsed et al, 2016). Multiplanar reformats and maximum intensity projection are useful for angiographic assessment. Acquiring computed tomography

Table 2. Salisbury computed tomography protocols

Phase	Protocol
Computed tomography portal venous phase	■ Volume: 70–80 ml Omnipaque 300 ■ Flow rate: 2.5–3 ml/sec ■ 60 s delay
Computed tomography arterial phase	■ Volume: 100 ml Omnipaque 300 ■ Flow rate: 4–5 ml/sec ■ 25–30 s delay

images in the arterial phase requires the patient to have a wide calibre intravenous cannula, because of the high flow rate of contrast administered for the study (*Table 2*).

Magnetic resonance angiography is another potential option for assessing mesenteric vasculature, but the prolonged time taken to acquire images makes it less suitable for patients presenting acutely and it is less readily available than computed tomography in most centres. Magnetic resonance angiography also has lower spatial resolution than computed tomography angiography, so is less suitable for demonstrating occlusion of small vessels (Shih and Hagspiel, 2007).

Conventional angiography is not recommended as the initial diagnostic test, but can be useful following computed tomography angiography if endovascular intervention is feasible (Tilsed et al, 2016).

Radiological features suggestive of mesenteric ischaemia

Pneumatosis intestinalis

This describes the appearance of gas within the bowel wall (*Figures 2* and *3*). The term pneumatosis coli may be used instead if the gas is solely within the wall of the colon. The appearance is best appreciated on computed tomography but may be visible on plain abdominal radiography. It is thought to occur in mesenteric ischaemia as a result of gas-forming organisms in the gangrenous bowel wall and carries a poor prognosis (Ho et al, 2007). The same appearance is seen in the neonatal period in patients with necrotizing enterocolitis, but the prognosis in these cases is usually much better. The sign may also be seen with a number of other causes including chronic obstructive pulmonary disease, recent colonoscopy, inflammatory bowel disease, and even in asymptomatic patients (Ho et al, 2007).

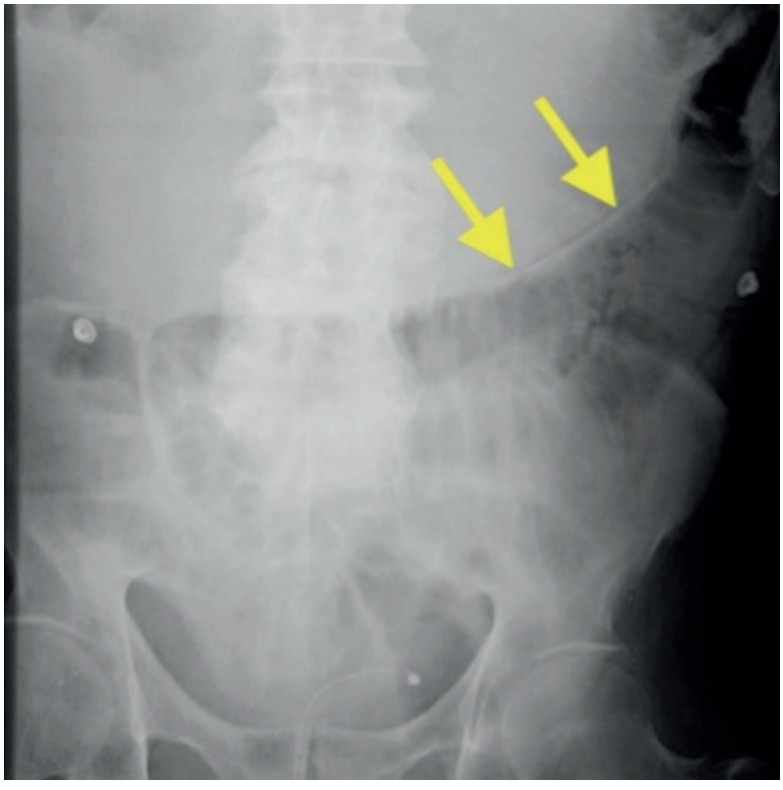

Figure 2. Abdominal X-ray demonstrating pneumatosis intestinalis.

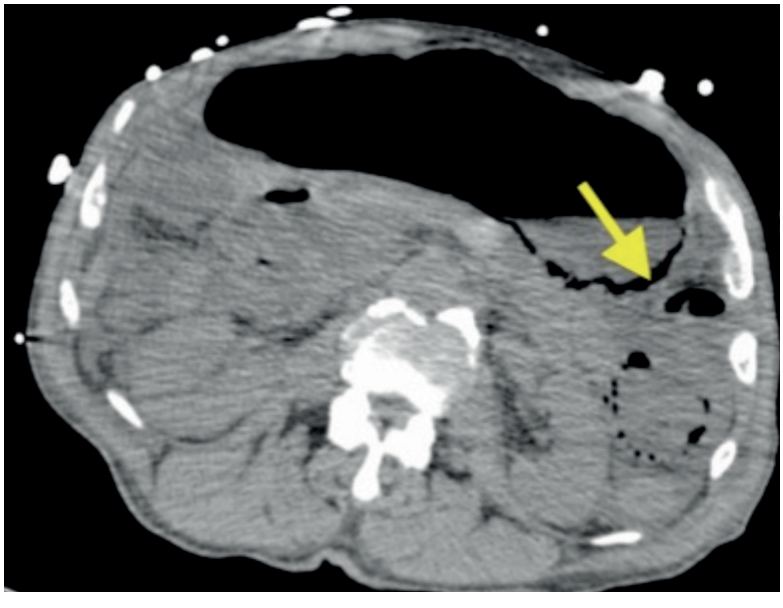

Figure 3. Unenhanced axial computed tomography image demonstrating pneumatosis intestinalis.

Portal venous gas

This is seen as a branching gas pattern within the liver, following the course of the portal venous tracts (*Figures 4* and *5*). It is important to differentiate this from pneumobilia (*Figure 6*), as the portal venous tracts and biliary tracts follow a similar course. A key way to differentiate the two is that the gas pattern is peripheral in portal venous gas, but central in pneumobilia as a result of the direction of flow – hepatopetal (from central to peripheral) in the portal tracts and hepatofugal (from peripheral to central) in the biliary tracts (Abboud et al, 2009). Comparison with previous imaging may be useful as if the patient has pneumobilia, for example following previous sphincterotomy at endoscopic retrograde cholangiopancreatography, the gas may be chronic and visible on earlier studies. Portal venous gas can be seen in a similar range of conditions to those responsible for pneumatosis intestinalis, and indeed the two signs are frequently seen together. It can also be seen incidentally

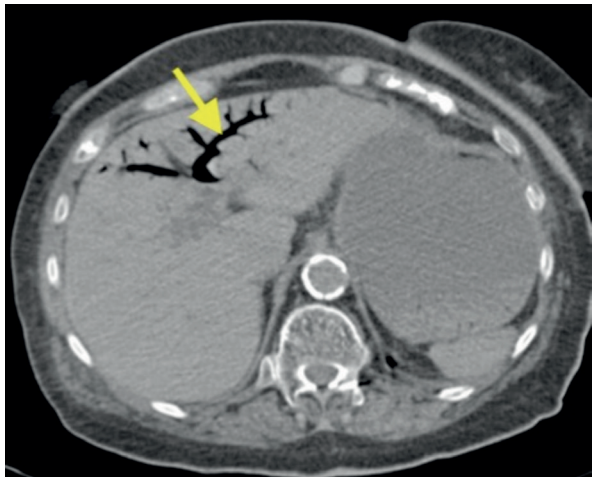

Figure 4. Unenhanced axial computed tomography image demonstrating portal venous gas.

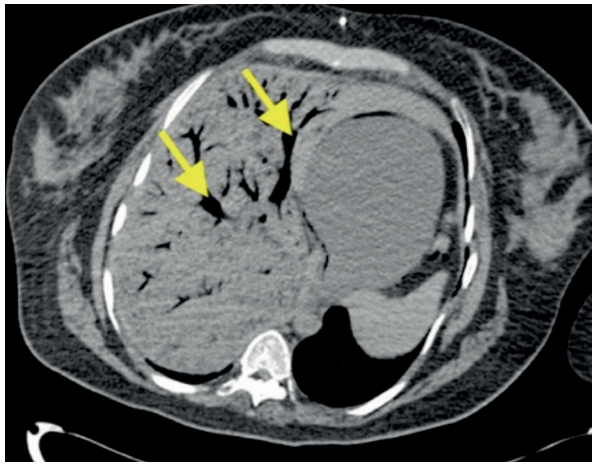

Figure 5. Unenhanced axial computed tomography image demonstrating portal venous gas and gas in the stomach wall.

in asymptomatic patients, but in cases of mesenteric ischaemia is normally a very poor prognostic sign, carrying a mortality rate of 75–90% (Abboud et al, 2009).

Bowel wall thickening

This is a fairly common sign in mesenteric ischaemia (*Figure 7*) as a result of inflammation and is particularly seen in mesenteric venous thrombosis as a result of venous hypertension and engorgement, but is very non-specific. Conversely, the bowel wall can be thinned in arterial embolic ischaemia as there is no mucosal oedema or haemorrhage in the early stages (Furukawa et al, 2009). It should be noted that bowel wall thickening does not correlate with the severity of ischaemia (Wiesner et al, 2003).

Differential bowel wall attenuation

This is only appreciable if no oral contrast or negative oral contrast (e.g. water) is given – artefact from positive (e.g. iodinated) oral contrast directly adjacent to the bowel mucosa can mask the true density of the bowel wall, so such agents should not be used in cases of suspected

Figure 6. Contrast-enhanced axial computed tomography image demonstrating pneumobilia (note the gas within the bile duct anterior to the main portal vein).

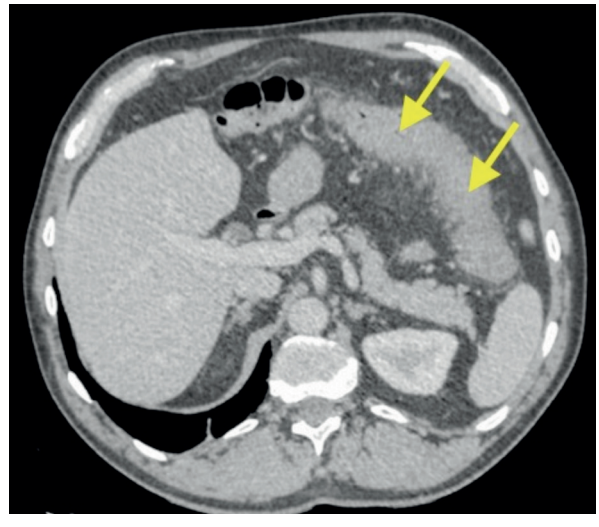

Figure 7. Contrast-enhanced axial computed tomography image demonstrating thickening of the splenic flexure at the watershed territory between superior mesenteric artery and inferior mesenteric artery supply (Griffith's point).

mesenteric ischaemia. Low bowel wall attenuation may be present, caused by oedema, either as a result of venous occlusion or reperfusion following arterial occlusion (Schieda et al, 2013). High bowel wall attenuation is either caused by submucosal haemorrhage, which is most frequently seen in mesenteric venous thrombosis as a result of venous hypertension, or as a result of hyperaemia or hyperperfusion (Schieda et al, 2013). Differentiating between haemorrhage and abnormal mural enhancement is only possible by performing an unenhanced scan before intravenous contrast is given. However, studies have shown there is no diagnostic benefit in terms of extra cases of mesenteric ischaemia being detected by performing a pre-contrast scan to look for this sign, so most centres do not perform a pre-contrast scan for acute presentations (Schieda et al, 2013).

Continued on p. 91

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Vascular filling defect

This is highly specific in cases of occlusive arterial mesenteric ischaemia but is poorly sensitive, particularly in small vessels (Kirkpatrick et al, 2003). The best chance of identifying a vascular 'filling defect' is achieved by performing bolus tracked mesenteric angiography (in addition to portal venous phase images) with a power injector using a high contrast volume and flow rate. An embolus will manifest as a dark clot (filling defect) surrounded by bright contrast in a fairly distal arterial segment usually without significant mural calcification (Acosta and Björck, 2014). Arterial thrombus is often seen as a filling defect in a proximal segment with heavy atherosclerosis (*Figure 8*)(Acosta and Björck, 2014). If mesenteric venous thrombus is present (*Figure 9*), the thrombus may extend into the portal vein, but alternative causes of portal venous thrombosis should also be considered in these cases (Kirkpatrick et al, 2003).

Free fluid

Abdominal and/or pelvic free fluid is a frequent finding in patients with mesenteric ischaemia (Wiesner et al, 2003). The fluid can be either exudative (high protein content) because of the resultant inflammatory process, or transudative (low protein count) in cases of mesenteric venous thrombosis with established portal vein thrombosis (Wiesner et al, 2003). There is often a haemorrhagic component to the fluid particularly in advanced cases where the bowel has infarcted and become gangrenous. The presence of free fluid can be supportive of a diagnosis of mesenteric ischaemia, but is not specific as it may be seen in a wide range of other conditions (Wiesner et al, 2003).

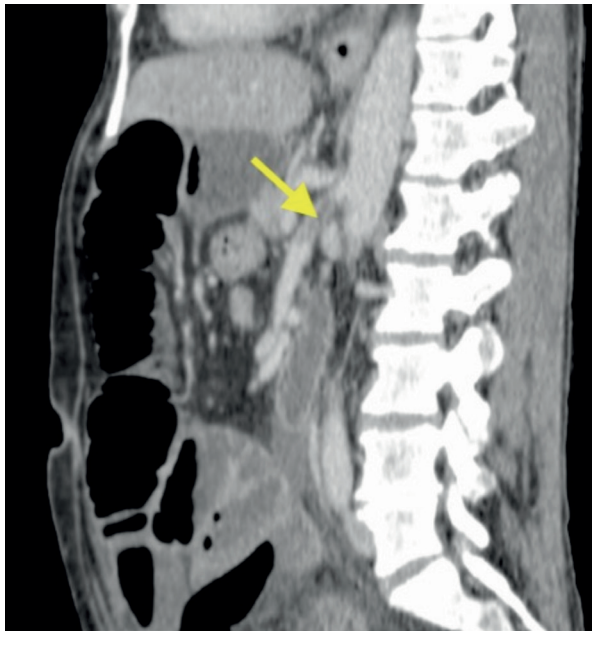

Figure 8. Sagittal reconstruction of a contrast-enhanced computed tomography scan showing a filling defect (thrombus) in the superior mesenteric artery.

Atherosclerosis

This is associated with thrombotic arterial mesenteric ischaemia, either in its chronic or acute form (Cappell, 1998). Typical signs include mural calcification and non-occlusive wall-adherent thrombus resulting in a vascular stenosis. It is important to understand that these findings are fairly common in the general population, usually in asymptomatic patients – particularly in the elderly and those with vascular risk factors. A relative paucity of mesenteric atherosclerosis favours embolism or non-occlusive mesenteric ischaemia rather than a thrombotic cause (Cappell, 1998).

Pneumoperitoneum

Breakdown of the bowel wall in advanced cases with bowel necrosis can readily lead to perforation, giving free gas within the peritoneal cavity (Cappell, 1998). Other causes of pneumoperitoneum such as trauma or recent surgery should be excluded using clinical history and examination.

Treatment

There is a lack of high quality evidence for optimal treatment of acute mesenteric ischaemia (Tilsed et al, 2016).

The preferred treatment depends largely on the aetiology and clinical condition of the patient – prompt resuscitation and diagnosis is essential in patients presenting acutely to ensure the best outcome possible (Tilsed et al, 2016). Improvements in the availability and quality of imaging have enabled faster recognition of mesenteric ischaemia and the likely cause of the ischaemia.

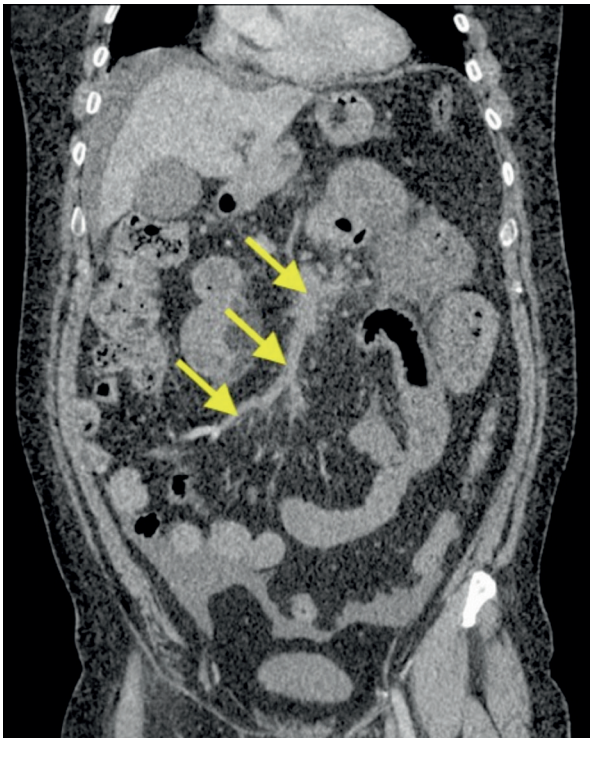

Figure 9. Coronal reconstruction of a contrast-enhanced computed tomography scan, showing a filling defect (thrombus) within an engorged superior mesenteric vein, with associated small bowel wall thickening.

KEY POINTS

- Mesenteric ischaemia is a condition that often presents with non-specific signs and symptoms but carries significant risk of morbidity and mortality.
- Early diagnosis is critical, with multiphase computed tomography offering very good sensitivity and specificity.
- Treatment varies depending on the cause, but the focus has historically been surgical. However, in recent years endovascular treatment has gained traction as an alternative.

Medical

Early treatment with broad spectrum antibiotics is generally recommended to cover the risk of sepsis from mucosal breakdown and bacterial translocation (Tilsed et al, 2016).

Anticoagulation is the mainstay of treatment for patients with mesenteric venous thrombosis (Acosta and Björck, 2014). It is also important in cases of arterial mesenteric ischaemia after surgery or endovascular treatment or when definitive treatment is not appropriate or possible (Acosta and Björck, 2014).

Vasopressors should be avoided where possible as they can lead to a reduction in mesenteric perfusion and worsening of ischaemia (Tilsed et al, 2016).

Surgical

In cases of established acute mesenteric ischaemia the definitive treatment has traditionally been surgical to allow open embolectomy where possible, revascularization, direct assessment of bowel viability and resection of gangrenous segments (Acosta and Björck, 2014). Vascular bypass surgery is another option (Tilsed et al, 2016). The surgeon may opt to perform a 'second look' operation 24–48 hours after the initial procedure to further assess bowel viability and progress (Tilsed et al, 2016).

Interventional radiology

There is increasing evidence that endovascular treatment can result in decreased morbidity and complications compared to surgery, although a significant proportion of patients still require subsequent laparotomy to enable resection of necrotic bowel (Arthurs et al, 2011).

Direct infusion of thrombolytic agents can also be lifesaving, but with a risk of bleeding as a complication. Clot retrieval or thrombolysis may be followed up with angioplasty and/or stenting (Tilsed et al, 2016). Endovascular treatment is suggested as first line therapy in patients with mesenteric arterial thrombosis, both in its acute and chronic guises (Schaefer et al, 2007; Tilsed et al, 2016).

Infusion of papaverine (an opioid derivative that acts as a smooth muscle relaxant) into the affected vessel at catheter angiography can be beneficial, particularly in cases of non-occlusive mesenteric ischaemia or critical acute mesenteric arterial ischaemia (Tilsed et al, 2016).

There is little evidence for endovascular treatment of mesenteric venous thrombus, but case reports have been published discussing local thrombolysis or thrombectomy following transjugular intrahepatic portosystemic shunt (Ferro et al, 2007).

Conclusions

Mesenteric ischaemia is subdivided based on the underlying mechanism, with common but also distinguishing features helping to differentiate between them. This article discusses the key radiological features, with a summary of current treatment options. **BJHM**

Table 1 *is adapted from https://cdemcurriculum.com/mesenteric-ischemia/*
Conflict of interest: none.

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