BMJ Best Practice

Ischaemic bowel disease

Straight to the point of care

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Summary

Ischaemic bowel disease can be classified into three types: acute mesenteric ischaemia, chronic mesenteric ischaemia, and colonic ischaemia. Acute mesenteric ischaemia may also be further subdivided into embolic, thrombotic, and venous mesenteric ischaemia. Colonic ischaemia is the most common type and has the most favourable prognosis.

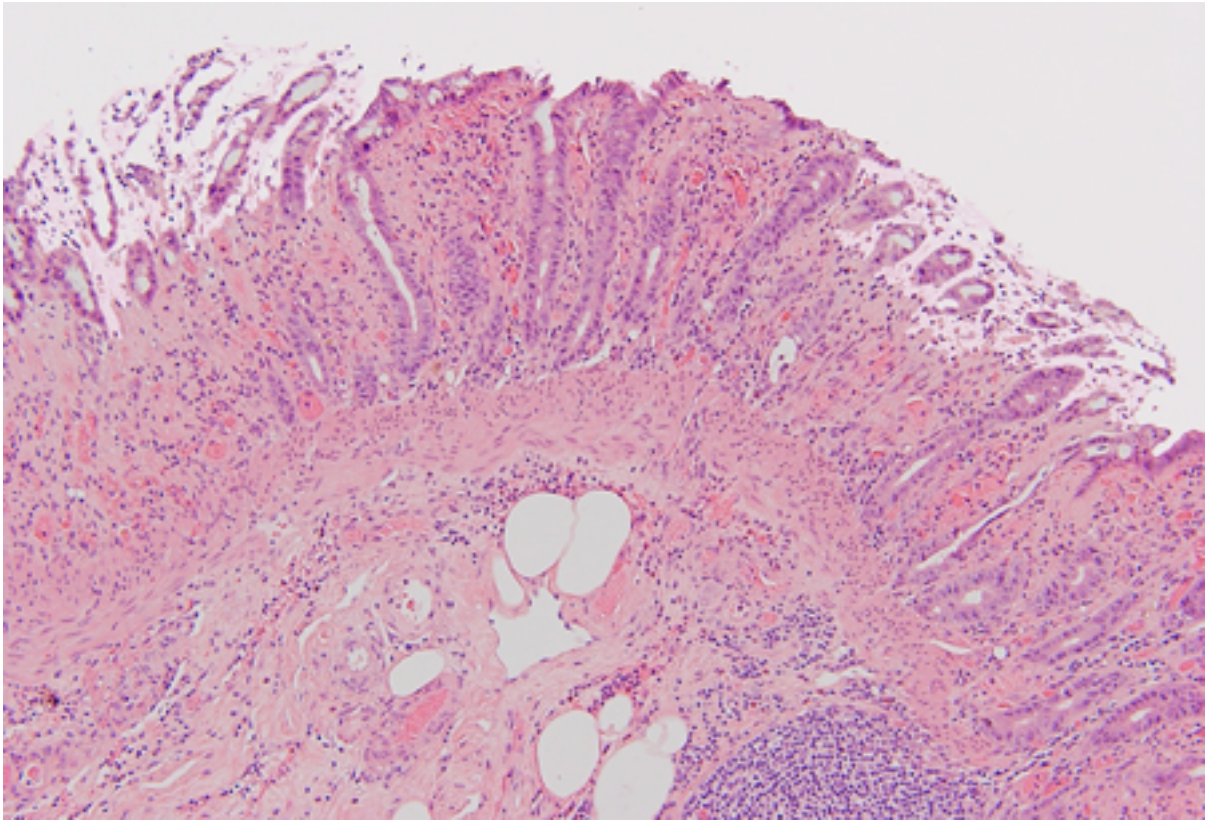
Ischaemic bowel disease may present clinically in a number of ways, including transient reversible ischaemia, chronic irreversible ischaemia, or acute fulminant ischaemia.

Mesenteric venous thrombosis may lead to acute or subacute intestinal ischaemia and may also present across a spectrum of severity.

Long-term complications of ischaemic bowel disease depend on the location and nature of the underlying pathology. Possible complications include stricture formation, short bowel syndrome, and food fear leading to malnutrition.

Definition

Ischaemic bowel disease encompasses a heterogeneous group of disorders caused by acute or chronic processes, arising from occlusive or non-occlusive aetiologies, which result in decreased blood flow to the gastrointestinal tract. The clinical course may range from transient and reversible to fulminant.



Histopathology of intestinal ischaemia

From the collection of Dr Jennifer Holder-Murray; used with permission

Epidemiology

Colonic ischaemia frequently occurs in older people with co-existing morbidities.[4] A systematic review identified 4 studies reporting incidence rates in general populations; three of the studies reported rates between 4.5 and 9 cases per 100,000 person-years and the fourth study reported a rate of 44 cases per 100,000 person-years.[5] These rates are likely to underestimate the true incidence as many patients with mild symptoms do not seek medical care.[5] Irritable bowel syndrome, recent cardiovascular surgery, constipation, chronic obstructive pulmonary disease, and other factors increase the risk of developing colonic ischaemia between two- and fourfold.[5] [6] [7]

Acute mesenteric ischaemia accounts for less than 0.2% of hospital admissions.[8] It occurs more commonly in those with comorbidities, most notably atrial fibrillation, myocardial infarction, and atherosclerosis. It has been estimated that acute arterial mesenteric ischaemia is responsible for up to 1% of patients presenting with an acute abdomen.[9] One study in Sweden found that between 1970 and 1982, the overall incidence of acute thromboembolic occlusion of the superior mesenteric artery was 8.6 cases per 100,000 person-years, increasing to 216.5 cases per 100,000 person-years in those ages >85 years.[10]

Non-occlusive mesenteric ischaemia (NOMI) accounts for 20% to 30% of acute mesenteric ischaemia.[8] [11] The overall incidence of NOMI is 2 cases per 100,000 person-years, increasing to 40 per 100,000 in patients aged >80 years.[12] NOMI may also occur in patients receiving enteral nutrition in intensive care following surgery or trauma; in this context it is associated with a very poor prognosis.[13]

Risk factors

Strong

old age

Older people frequently have medical comorbidities such as atrial fibrillation, myocardial infarction, advanced atherosclerosis, and heart failure, which are significant contributory factors to the development of bowel ischaemia.[17] [23]

history of smoking

A history of smoking, often in combination with peripheral vascular disease and hypertension, is frequently present.[19]

hypercoagulable states

A strong risk factor for mesenteric venous thrombosis (MVT). Diagnosis of MVT should be particularly suspected in patients who have sudden onset of severe abdominal pain and high risk of thromboembolism; approximately 50% of patients presenting with MVT have had a deep vein thrombosis or pulmonary embolus in the past.[19] Common causes of hypercoagulability include cirrhosis or portal hypertension; inheritable hypercoagulable states such as factor V Leiden, protein C deficiency, or prothrombin G20210A mutation; oral contraceptive use; malignancy; pancreatitis; and a history of recent surgery.[19]

atrial fibrillation

Untreated, atrial fibrillation can result in the formation of thrombi within the atria, which can then embolise to the mesenteric vasculature.[17]

myocardial infarction

Impaired wall motion secondary to myocardial infarction can act as a nidus for thrombus formation on the ventricular wall, which can then embolise to the mesenteric vessels.

structural heart defects

Defects such as right-to-left shunts can increase the risk of emboli to mesenteric vessels.

history of vasculitis

Rheumatoid arthritis, polyarteritis nodosa, systemic lupus erythematosus, dermatomyositis, Takayasu arteritis, and thrombo-angiitis obliterans can all result in ischaemia of the bowel. The exact clinical picture varies depending upon factors such as the size of the mesenteric vessel involved.

Weak

recent cardiovascular surgery

Between 0.6% and 6.7% of patients who have recently undergone cardiac or major vascular procedures develop colonic ischaemia; mortality may be as high as 80% in this population.[2] [3] Surgical management of thoracic and abdominal aortic aneurysms (AAAs) is strongly associated with bowel ischaemia; prevalence following repair of ruptured AAA is up to 10% and after elective endovascular repair incidence is up to 2.8%.[3] [24] Factors that may underlie these figures include emboli arising from cross-clamping of the aorta, a risk of intestinal hypoperfusion in the postoperative period, and a relatively high incidence of heart failure in these patients. Significant risk factors for mesenteric ischaemia post-cardiac surgery include advanced age (>70 years), prolonged bypass time, emergency surgery, higher volume of blood loss, and other evidence of post-operative organ dysfunction, such as a rise in lactate, transaminases, and creatinine.[25] [26]

shock

Hypoperfusion due to shock may exacerbate to a critical level any underlying intestinal low-flow states that may be present due to atherosclerosis. Even in the absence of an existing low-flow state, severe shock can result in ischaemia of the bowel.

congestive heart failure

Heart failure may exacerbate underlying intestinal low-flow states that may be present due to atherosclerosis. Even in the absence of an existing low-flow state, severe heart failure can lead directly to ischaemia of the bowel.

atherosclerosis

Atherosclerosis may lead directly to intestinal hypoperfusion and ischaemia due to partial or complete occlusion of vessels supplying the gut. It can also act as a source of emboli.

Severe atherosclerosis in vessels supplying the gut also makes individuals more vulnerable to bowel ischaemia, which are then worsened by congestive heart failure or shock (see above).

irritable bowel syndrome

The diagnosis of irritable bowel syndrome is associated with a 2-fold increased risk of developing colonic ischaemia.[6] [7] The underlying basis of this association is not known.

colonic carcinoma

Approximately 20% of older patients with colonic ischaemia have a distal obstruction from carcinoma, stricture, faecal impaction, or diverticular disease. The proximal colonic distension leads to intraluminal dilation and increased pressure that may result in decreased mucosal perfusion and ischaemia, typically in the caecum.

constipation

Constipation and prolonged straining during defecation result in transient decreased colonic blood flow, which in patients with low-flow states can trigger ischaemia.

long-term laxative use

The incidence of colonic ischaemia is over 4 times more common in patients who use laxatives on a long-term basis.[6] Cases associated with short-term use of laxatives or bowel preparation protocols for endoscopy have also been documented.[27] [28]

use of vasopressors, digoxin, cocaine

Especially in the setting of severe atherosclerosis, use of vasopressors, digoxin, and cocaine have been shown to exacerbate non-occlusive mesenteric ischaemia.[19]

Aetiology

Arterial compromise

- Embolism
  - Embolic arterial obstruction accounts for 40% to 50% of acute mesenteric ischaemia; most commonly affecting the superior mesenteric artery.[14] The embolus usually originates from a left-sided heart thrombus, or from spontaneous or iatrogenic rupture and embolisation from an aortic atherosclerotic plaque or aneurysm.[15][16] [17] Interventional radiological procedures are the most common cause of iatrogenic plaque rupture.
- Thrombosis
  - About 15% to 20% of acute mesenteric ischaemia results from thrombus occurring as a progression of atherosclerosis at the origin of the superior mesenteric artery.[15] [18] Mesenteric atherosclerotic plaques may rupture with associated acute thrombosis of the vessel. Subacute or chronic ischaemia may result from partial occlusion of the vessel.
- Vasculitis
  - Rheumatoid arthritis, polyarteritis nodosa, systemic lupus erythematosus, dermatomyositis, Takayasu arteritis, and thrombo-angiitis obliterans can all result in ischaemia of the bowel. The exact clinical picture varies depending upon factors such as the size of the mesenteric vessel involved.
- External compression

- Rarely, extrinsic compression of the coeliac axis can lead to mesenteric ischaemia, usually due to the median arcuate ligament of the diaphragm and surrounding nerve plexus impinging onto the coeliac axis. It occurs more often in women than in men.[19]
- Tumours and other masses within the abdomen can also surround and ultimately compress blood vessels supplying the bowel, causing ischaemic damage.

Venous compromise

- Venous thrombosis
  - Accounts for 5% to 15% of cases of acute mesenteric ischaemia.[1] [14] Frequently involves the superior mesenteric vein.
  - Usually associated with cirrhosis or portal hypertension; other potential associations include inheritable hypercoagulable states (e.g., factor V Leiden, protein C deficiency, prothrombin G20210A mutation), pancreatitis, malignancy, oral contraceptive use, and recent surgery. Approximately half of patients presenting with venous thrombosis have had a prior history of deep vein thrombosis or pulmonary embolus.[14] [19]

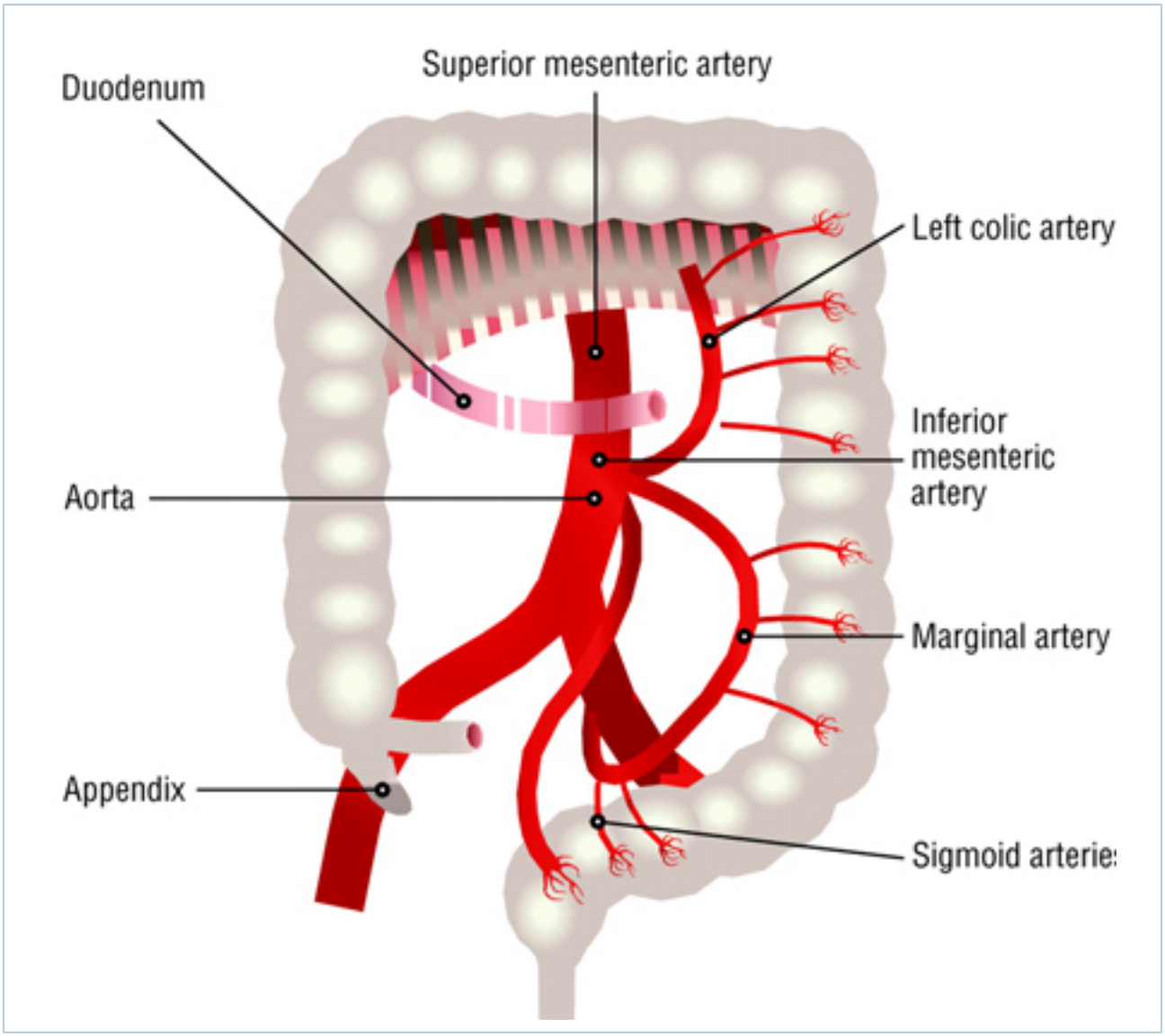
Hypoperfusion (i.e., non-occlusive ischaemia)

- Accounts for as much as 20% of cases of acute mesenteric ischaemia.[20]
- Shock, or hypotension, or relative mesenteric hypotension (from any aetiology). Prominent causes include:
  - Heart failure
  - Dialysis
  - Drug-related
    - Such as digoxin, oestrogen, contraceptives, vasopressin, vasopressors, danazol, flutamide, glycerin enema, alosetron, immunosuppressives, psychotropics, imipramine, adrenaline (epinephrine), sumatriptan, non-steroidal anti-inflammatory drugs, ergot, diconal, laxatives, pegylated interferon, methamphetamines, and cocaine.[21] [22]
  - Recent surgery or trauma
    - Such as aortic aneurysm repair, aorto-iliac bypass, colectomy, colonoscopy.
    - Increased risk with enteral nutrition in post-surgical or trauma patients in intensive care (incidence reported between 0.3% and 8.5%).[20]
  - Infection
    - Such as cytomegalovirus, hepatitis B, *Escherichia coli* O157:H7.
  - Other

- Such as pancreatitis, polycythaemia vera, phaeochromocytoma, carcinoid syndrome.

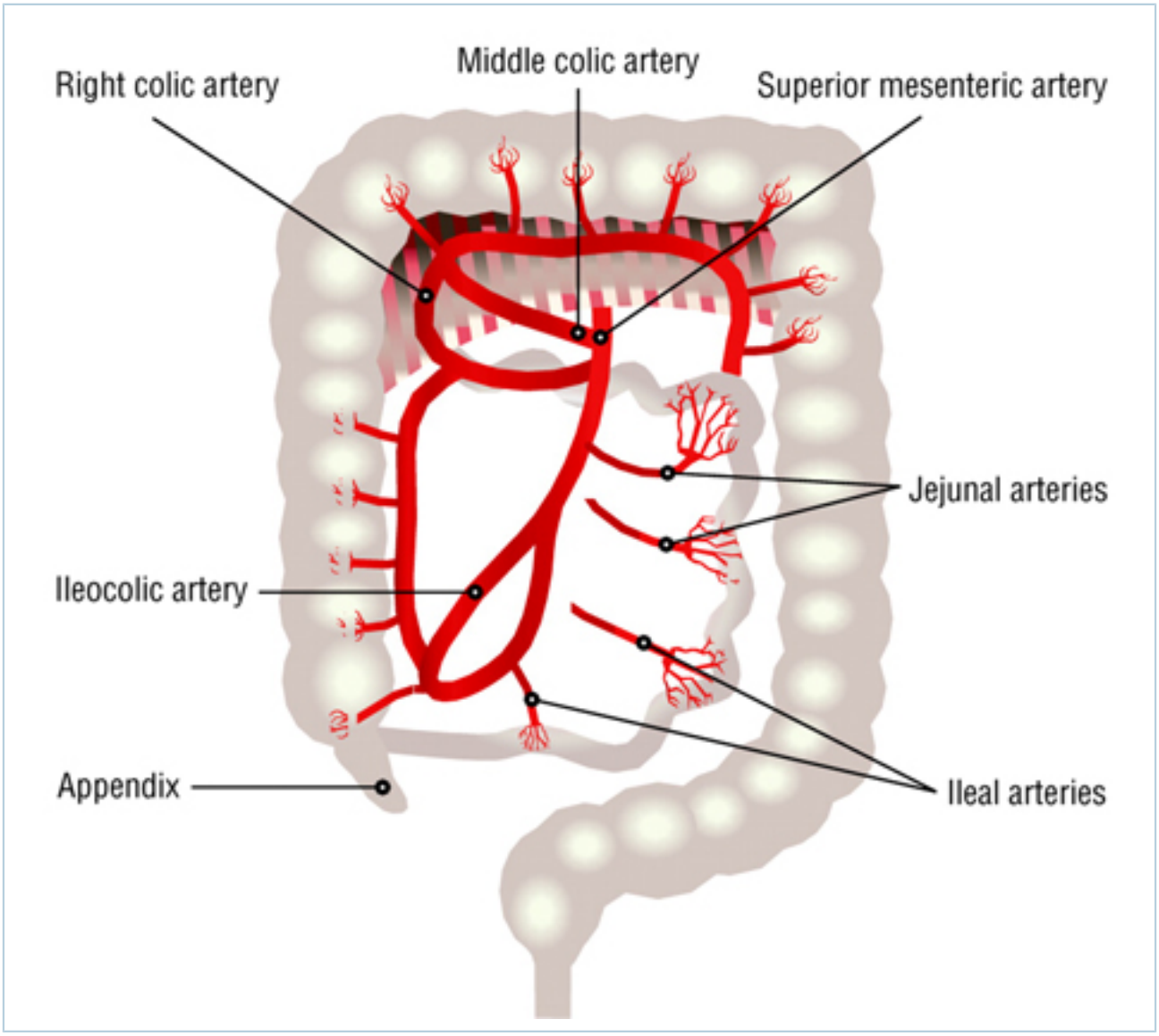
Pathophysiology

The small intestine receives blood via the coeliac artery and the superior mesenteric artery (SMA). The colon receives blood via the SMA and the inferior mesenteric artery (IMA). The rectum also receives blood via branches of the internal iliac artery. Several collateral arteries exist between the SMA and the IMA, including the marginal artery of Drummond and the arc of Riolan. The splenic flexure and the recto-sigmoid junction are two watershed areas where collateralisation of blood flow may be limited.



Distribution of blood flow to the colon originating from the inferior mesenteric artery, branches of which include the left colic, marginal, and sigmoid arteries and supply the left colon and superior portion of the rectum

BMJ 2003; 326 doi: 10.1136/bmj.326.7403.1372



Distribution of blood supply to the small intestine and colon from the superior mesenteric artery, branches of which include the middle, right, and ileocolic arteries as well as jejunal and ileal arteries and arterioles

BMJ 2003; 326 doi: 10.1136/bmj.326.7403.1372

Ischaemia occurs secondary to hypoperfusion of an intestinal segment. When hypoperfusion is insidious in onset, collateral blood flow may develop, precluding or minimising ischaemia; however, the regions of the intestine with a solitary arterial supply, and the watershed areas, are both at increased risk of developing ischaemia. The degree of intestinal injury is dependent on the duration and severity of ischaemia. Acute or subacute mucosal sloughing and ulcerations occur as a result of ischaemia. The loss of the mucosal barrier allows for bacterial translocation and toxin or cytokine absorption. Reperfusion injury can also occur if blood supply is re-established after a prolonged interruption. Segments of ischaemic bowel that do not suffer acute necrosis or perforation can heal with stenosis or stricture as the long-term sequelae of bowel ischaemia.

Thromboembolic events that lead to mesenteric ischaemia usually involve the SMA instead of the other mesenteric arteries (IMA and coeliac artery). This is because of the anatomical position of the SMA; the SMA is positioned vertically in relation to the aorta while the other vessels form more oblique angles with the aorta.

THEORY

Classification

Intestinal ischaemia can be classified into three broadly defined types:[1]

- Acute mesenteric ischaemia
    - Superior mesenteric artery embolus
    - Superior mesenteric artery thrombosis
    - Non-occlusive mesenteric ischaemia
    - Superior mesenteric vein thrombosis
    - Focal segmental ischaemia.
- Chronic mesenteric ischaemia
- Colonic ischaemia
    - Reversible ischaemic colonopathy
    - Transient ulcerating ischaemic colitis
    - Chronic ulcerating ischaemic colitis
    - Colonic stricture
    - Colonic gangrene
    - Fulminant universal ischaemic colitis.

Case history

Case history #1

A 48-year-old man presents with diffuse abdominal pain, worse after eating meals. The pain has been present for the previous 6 months, but has worsened recently. He has had significant weight loss since the onset of symptoms. His past medical history includes systemic lupus erythematosus, which has been difficult to manage medically.

Case history #2

A 78-year woman with a recent history of myocardial infarction and atrial fibrillation presents with sudden onset of severe, continuous, and diffuse abdominal pain, nausea, and bloody bowel movements. She is diaphoretic, tachycardic, and hypotensive, and appears very unwell.

Other presentations

Up to 6.7% of patients who have undergone open or endovascular cardiac or major vascular procedures develop colonic ischaemia; mortality may be as high as 80% in this population.[2] [3] These patients typically present with crampy abdominal pain and watery diarrhoea within a few days of surgery. Factors

that may underlie these figures include emboli arising from cross-clamping of the aorta, a risk of intestinal hypoperfusion in the postoperative period, and a relatively high incidence of heart failure in these patients.

Recommendations

Key Recommendations

Acute mesenteric ischaemia is a **life-threatening emergency**.

Maintain a **high index of suspicion** for bowel ischaemia, because the signs and symptoms are relatively non-specific yet the condition has significant morbidity and mortality, particularly if diagnosis is delayed.[29] Time is of the essence to improve outcomes; early recognition, appropriate diagnostic studies, and aggressive treatment should be instigated emergently and expeditiously.

The classic presentation of acute mesenteric ischaemia is **abdominal pain out of proportion to examination**.

- Use imaging to make a diagnosis in the absence of highly specific or definitive signs and symptoms. An urgent computed tomography scan is the first-line investigation of choice.
- A history and physical examination alone are generally not sufficient to make the diagnosis.
- Where clinically indicated, resuscitate in parallel with the diagnostic work-up in order to minimise the risk of ischaemia progressing. Administer supplemental oxygen and adequate fluid replacement, and correct acute heart failure or arrhythmias.

When fulminant ischaemic bowel disease is present, don't allow extensive diagnostic testing to delay appropriate surgical intervention.

Full Recommendations

Clinical presentation

Be aware that bowel ischaemia encompasses a wide spectrum of disorders. Presenting features and history vary considerably. Alternative diagnoses must be excluded.[30]

Acute mesenteric ischaemia is a **life-threatening emergency**.

Maintain a high index of suspicion for bowel ischaemia, because the signs and symptoms tend to be non-specific yet the condition has significant morbidity and mortality, particularly if diagnosis is delayed.[29] Time is of the essence to improve outcomes; early recognition, appropriate diagnostic studies, and aggressive treatment should be instigated emergently and expeditiously.

> **Practical tip**
>
> A hallmark of salvageable acute intestinal ischaemia is very severe abdominal pain with a lack of physiological disturbance or physical signs.

Aim to differentiate mesenteric ischaemia from ischaemic colitis. Suggestive findings for each of the possible types of ischaemic bowel disease are as follows.

Acute mesenteric ischaemia

Most patients present with sudden abdominal pain, often peri-umbilical. The classic presentation is pain disproportionate to the physical examination findings.[8] [20] It will generally persist and worsen. As ischaemia progresses to infarction, the pain may become more diffuse.[20] Nausea and vomiting may also be present.

- The patient may be severely ill in a delayed presentation, where the bowel ischaemia is extensive or has progressed to infarction.
- Patients with arterial embolus may describe sudden, severe abdominal pain with rapid, forceful bowel evacuation, possibly containing blood. Arterial embolus is the most common cause of acute mesenteric ischaemia.[31] Acute mesenteric ischaemia due to arterial embolus should be suspected in patients who describe sudden, severe abdominal pain with:[31]
    - Rapid, forceful bowel evacuation, possibly containing blood, and/or
    - High risk of thromboembolism.
- Patients with mesenteric venous thrombosis have more variable presentations than patients with arterial aetiology.
    - Pain is often tolerated initially.
    - Typically, these patients describe colicky abdominal pain for a mean of 5 to 14 days before presentation; 25% of patients have had episodes of pain for >30 days before presentation.
    - About 60% to 70% of these patients have associated nausea and vomiting, and 30% have diarrhoea or constipation.[32]
    - A longer duration of symptoms before presentation in venous ischaemia may be associated with improved outcomes.[20]
- Older patients with long-standing congestive heart failure, cardiac arrhythmias, recent myocardial infarction, hypotension, or peripheral vascular disease are at higher risk for acute mesenteric ischaemia than younger patients.
- Younger patients may have a history of collagen vascular disease, vasculitis, hypercoagulable state, or vasoactive medication or cocaine use.

Consider intestinal ischaemia if the clinical picture does not suggest another abdominal pathology.[32]

Chronic mesenteric ischaemia

Insidious onset with repeated mild transient episodes over many months, becoming progressively more severe over time. Pain is poorly localised. It may worsen after exercise and often occurs after meals, gradually resolving over a few hours.[30]

- Patients may avoid food or become fearful of eating (sitophobia).[30]
- There may be significant weight loss, giving the patient a cachectic appearance.[30]
- Patients may have associated nausea, and diarrhoea with or without blood.[30]

The 'classic triad' of chronic mesenteric ischaemia is postprandial pain, weight loss, and an abdominal bruit; however, this is found in only a minority (around 20%) of patients.[30]

Infarction of bowel is uncommon, as insidious onset allows some collateral circulation to develop.

Chronic mesenteric ischaemia usually occurs in older people. Women are affected more than men (ratio 3:1).

- Patients frequently give a history of heavy smoking and other symptoms associated with atherosclerosis.
- Coeliac compression syndrome is a type of chronic mesenteric ischaemia that occurs due to the median arcuate ligament compressing the coeliac axis.[31]
    - Consider this diagnosis particularly in younger patients (especially women) with unexplained abdominal pain and normal upper endoscopy as well as normal liver, pancreatic, and gastric

laboratory studies, particularly in those patients who have an abdominal bruit (from partially obstructed flow in the coeliac axis).

Colonic ischaemia

Soon after the onset of ischaemia, there is usually pain with frequent bloody, loose stools, reflecting mucosal or submucosal damage. However, blood transfusion is rarely needed. Passage of maroon or red blood from the rectum is particularly characteristic of colonic ischaemia.

Patients typically describe mild to moderate pain that is usually felt peripherally, in contrast to the pain of acute mesenteric ischaemia, which is often described as peri-umbilical. The ischaemia is commonly in the 'watershed' areas of the colon (the areas of the colon between two major supplying arteries; see Pathophysiology), hence the pain is typically left-sided. Tenderness to palpation over the affected bowel occurs from early in the course of ischaemia, in contrast to acute mesenteric ischaemia, where tenderness is a relatively late sign.

- Patients do not generally appear severely ill, unless fulminant ischaemia is present.
- If colonic ischaemia progresses, pain becomes more continuous and diffuse. The abdomen becomes more distended and tender and there are no bowel sounds.
- If ischaemia progresses further still and necrosis approaches, there is a significant leakage of fluid, electrolytes, and protein through the damaged mucosa, with shock and metabolic acidosis.

Important risk factors for colonic ischaemia are:[23]

- Age >60 years
    - Around 90% of people with colonic ischaemia are over 60 years old
- Haemodialysis
- Hypertension
- Hypoalbuminaemia
- Diabetes mellitus
- Constipation-inducing medications.

Colonic ischaemia is increasingly identified in younger people, associated with strenuous and prolonged physical exertion (e.g., long-distance running), various medications (e.g., oral contraceptives), cocaine use, and coagulopathies (e.g., protein C and S deficiencies, antithrombin III deficiency, activated protein C resistance).[4]

Colonic ischaemia may also occur:

- Following aortic or cardiac bypass surgery
- In association with vasculitides such as systemic lupus erythematosus or polyarteritis nodosa, infections (e.g., cytomegalovirus, *Escherichia coli* O157:H7), coagulopathies
- After any major cardiovascular episode accompanied by hypotension
- With obstructing or potentially obstructing lesions of the colon (e.g., carcinoma, diverticulitis).

Diagnosis is by colonoscopy or contrast-enhanced computed tomography (CT) imaging or flexible sigmoidoscopy/colonoscopy.[4] More than 80% resolve spontaneously or with conservative measures, but surgery may be required in acute, subacute, or chronic cases.

Predictors of poor outcomes include a lack of rectal bleeding and right-sided ischaemia.[33]

Non-occlusive ischaemia (mesenteric or colonic)

Typically seen in patients with underlying hypotension and volume deficits, usually inadequate cardiac output causing splanchnic hypoperfusion. This may be related to congestive heart failure, hypovolaemia, sepsis, and cardiac arrhythmias, haemodialysis, or even related to medications.[31] [32]

Risk factors for non-occlusive ischaemia include older age, cardiac comorbidities, and medications such as digoxin and diuretics.[34] [35] Diagnosis is by CT and CT angiography.

	Acute mesenteric ischaemia	Chronic mesenteric ischaemia	Colonic ischaemia
Site	• Periumbilical pain • Upper abdominal or generalised	Epigastric or mid abdominal	Left lower quadrant or lower abdominal
Onset	Sudden	Insidious	Sudden
Character	• Sharp or colicky • Pain is out of proportion to the exam	Dull	Dull
Radiation	No radiation	No radiation	Radiates to back
Associations	• Nausea, vomiting, diarrhoea • May have sudden forceful bloody bowel evacuation	Nausea, vomiting	Bloody bowel movements
Timing, duration, frequency	Acute	Chronic	Acute
Exacerbating and relieving factors	No association with meals, pain not relieved	Worse after meals, resolving over hours	None
Severity	Severe	Mild	Mild-to-moderate
Abdominal examination	• Soft, non-tender with hyperactive bowel sound - early phase • Rebound tenderness, rigidity with absent bowel sound - late phase	Abdominal bruit	LLQ or lower abdominal tenderness
Cardiovascular exam	May have a fibrillation or other arrhythmia, evidence of vascular disease	Atherosclerosis, peripheral vascular disease	May have a fibrillation or other arrhythmia, atherosclerosis, evidence of peripheral vascular disease
Laboratory test results	• Leukocytosis • Elevated serum amylase • Lactic acidosis		
Imaging	• Thumbprinting on plain x-rays • Mesenteric occlusion on angiography • Subdiaphragmatic air if perforated • Pneumatosis intestinalis, or air in portal vessels when bowel necrosis present • Use contrast-enhanced CT to diagnose mesenteric venous thrombosis	Angiography demonstrates severe occlusion of at least 2 of the 3 splanchnic vessels	Angiography has no role
Typical patient characteristics	• Older patients with cardiovascular disease • Younger patients with collagen vascular disease, vasculitis, hypercoagulable state, vasoactive medication use, or cocaine use	Older women Smoker Cardiovascular disease Sitophobia	Older patients with cardiovascular disease or atrial fibrillation

Comparison of symptoms/signs and investigations for the three types of ischaemic bowel disease
Designed by BMJ Knowledge Centre, with input from Dr Amir Bastawrous

History

Take a thorough history to confidently exclude other potential diagnoses. Use a tool such as the SOCRATES mnemonic to explore the key characteristics of the abdominal pain:

- Site
- Onset
- Character
- Radiation
- Associations – nausea, vomiting, diarrhoea
- Timing, duration, frequency
- Exacerbating and relieving factors

- Severity.

Other important elements to elicit include smoking history, cardiovascular risk factors, comorbidities, and past medical history.

Physical examination

Assess vital signs as a priority to determine whether immediate resuscitation measures are required. Then perform a thorough examination of all systems, particularly focusing on the abdomen and cardiovascular system. When bowel ischaemia is associated with vasculitis or specific disease entities, characteristic dermatological, musculoskeletal, or further findings specific to the disease may be present.

Abdominal examination

Early in the course of acute mesenteric ischaemia the abdomen may initially be soft and non- or minimally tender to palpation. Typically, patients with acute mesenteric ischaemia initially report levels of **abdominal pain greater than would be expected** by the physical findings.

Patients with colonic ischaemia may have mild to moderate tenderness at an earlier stage in the course of ischaemia. This is felt more laterally over the affected parts of the colon compared with the pain and tenderness of acute mesenteric ischaemia, which is generally more peri-umbilical.

As ischaemia progresses towards infarction, patients develop signs of peritonitis, with a rigid, distended abdomen; guarding and rebound; percussion tenderness; and loss of bowel sounds.

It is imperative to consider and either diagnose or exclude acute mesenteric ischaemia in patients who present with severe abdominal pain and a paucity of significant abdominal findings. The dangers of a delay in diagnosis outweigh the risk of early invasive studies.[32]

Auscultation of the abdomen reveals an epigastric bruit (indicative of turbulent flow through an area of vascular narrowing) in 48% to 63% of patients.[19]

Rectal examination may demonstrate gross blood per rectum or microscopic blood upon testing for occult haemorrhage.

Peritonitis indicates a need for urgent surgical intervention.

Cardiovascular examination

Perform a cardiovascular examination, including an ECG. ECG may demonstrate arrhythmias that predispose to cardioembolic complications, such as atrial fibrillation or atrial flutter, or acute infarction that may be the aetiology of intestinal ischaemia.

Examination may reveal bruits on carotid auscultation, along with skin changes, absent hair, and absent distal pulses on the limbs, consistent with advanced atherosclerotic disease.

Investigations

Laboratory tests

Initial blood work should include tests to direct initial resuscitation, help assess the severity of any ischaemia, and provide clues to alternative diagnoses.[36]

- Full blood count

- More than 90% of patients with acute mesenteric ischaemia will have an abnormally elevated leukocyte count.[8]
- May reveal anaemia (often as a result of repeated episodes of melaena) that exacerbates ischaemia.

- Urea and electrolytes[36]
  - Helps assess renal dysfunction and dehydration, frequently present in patients with ischaemic bowel disease.
- Liver function tests[36]
  - May be elevated, as a consequence of septic shock or concomitant with bowel ischaemia.
- Arterial blood gases and serum lactate
  - Metabolic acidosis is a common finding in patients with acute mesenteric ischaemia.[8]
  - Elevated serum lactate does not determine the presence or absence of ischaemic or necrotic bowel; however, it can be used to assist with making the diagnosis and determining severity.[8]
- Coagulation studies, group and save, and crossmatch[36]
  - Aids in the diagnosis of any underlying coagulopathy as a risk factor for further thrombosis. Allows correction of any clotting dyscrasia as part of treatment.
  - Group and save in preparation for the possibility of transfusion.
- Serum amylase
  - Elevated serum amylase is found in approximately half of patients with acute mesenteric ischaemia.[8]
- D-dimer
  - May be elevated in intestinal ischaemia, but its use is limited because D-dimer is a very non-specific test.[8]

In suspected ischaemic colitis, also include:[36] [37]

- C-reactive protein
- Faecal culture
- *Clostridium difficile* toxin assay
- Studies for ova, cysts, and parasites.

Imaging

Prioritise an urgent computed tomography (CT) scan of the abdomen to diagnose ischaemia, and to exclude other potential diagnoses.[8] [20] [30]

If no alternative diagnosis is made following the CT scan, consider selective angiography in discussion with radiology. Based on the angiographic findings, treat the patient according to the specific cause of the ischaemia.

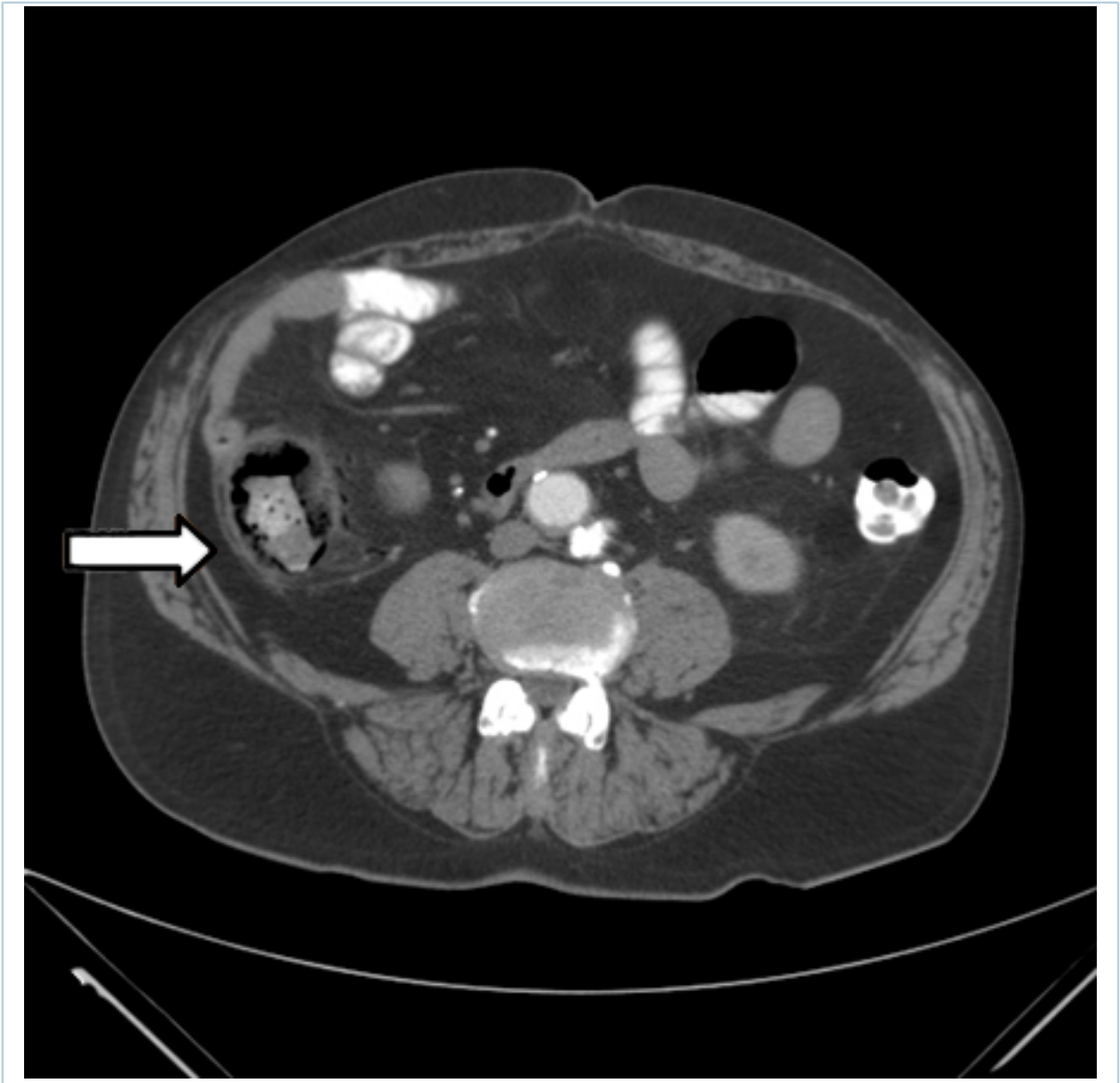
If you suspect chronic mesenteric ischaemia, discuss the imaging findings and the overall clinical picture with a multidisciplinary team (including at least a gastroenterologist, an interventional radiologist, and a vascular surgeon).[30]

If you suspect acute ischaemia and CT imaging or angiography is not immediately available, discuss the patient with a consultant surgeon, as prompt exploratory laparoscopy and possible laparotomy may be indicated in patients with suspected ischaemic bowel. Laparotomy without prior imaging may be indicated in unstable patients with peritoneal signs. Bear in mind, however, that CT imaging is widely available in developed countries such as the UK.

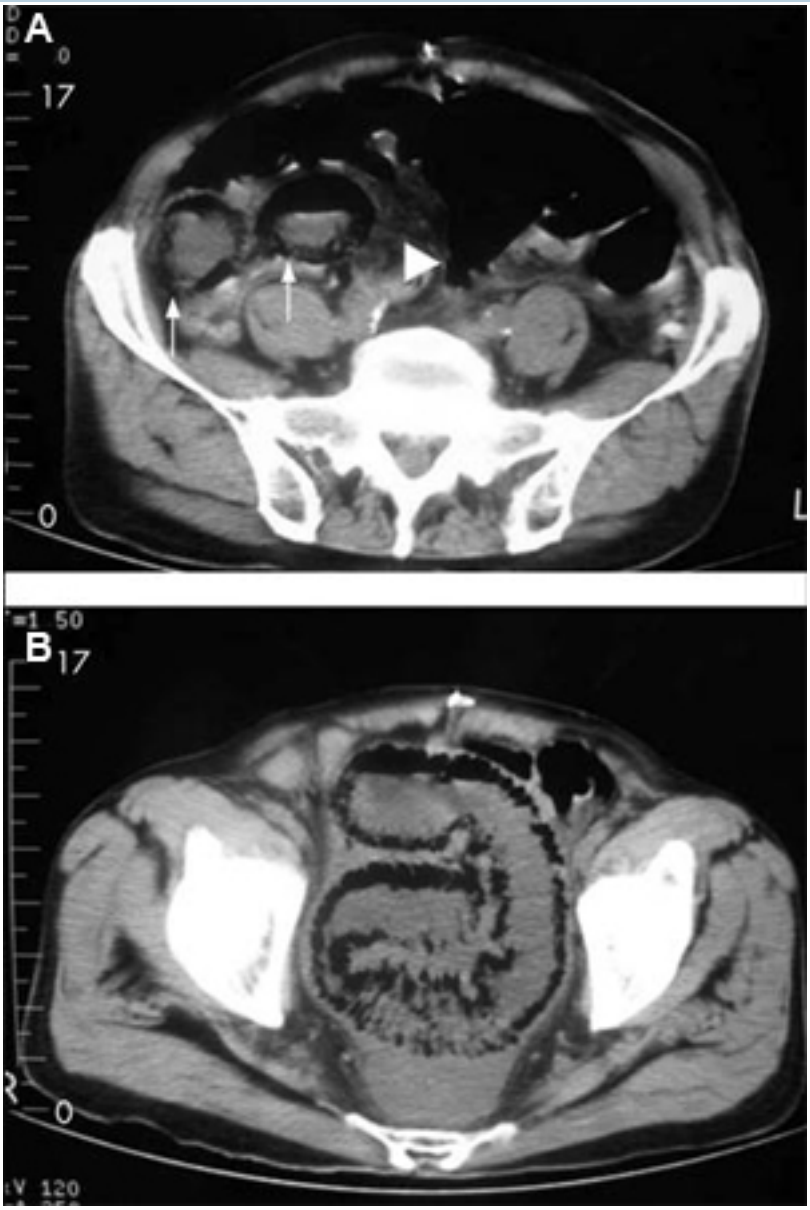
CT scanning

A CT scan is the first-line investigation of choice in the diagnosis of acute or chronic mesenteric ischaemia.[8] [20] [30][38] Obtain the scan early; prompt diagnosis (and intervention) is essential to improve the clinical outcome. Use multidetector CT scanning with intravenous contrast for suspected acute mesenteric ischaemia.[20] Consider a CT scan even in the presence of renal impairment in order to save life and prevent worsening renal injury.[8] [9][20]

CT provides evidence for the extent of bowel compromise from ischaemia; it is useful for diagnosing acute mesenteric ischaemia, but findings can be non-specific in early ischaemia.[39] [40] Early signs include bowel wall thickening and luminal dilation. Late signs include pneumatosis (gas in the bowel wall) and mesenteric or portal venous gas, which typically indicate necrotic bowel.[32] [41] Other late signs include oedematous bowel, and variable enhancement of the bowel surrounded by free fluid.[31] May show thickening of the bowel wall with thumbprinting sign suggestive of submucosal oedema or haemorrhage, which suggests a worse prognosis.

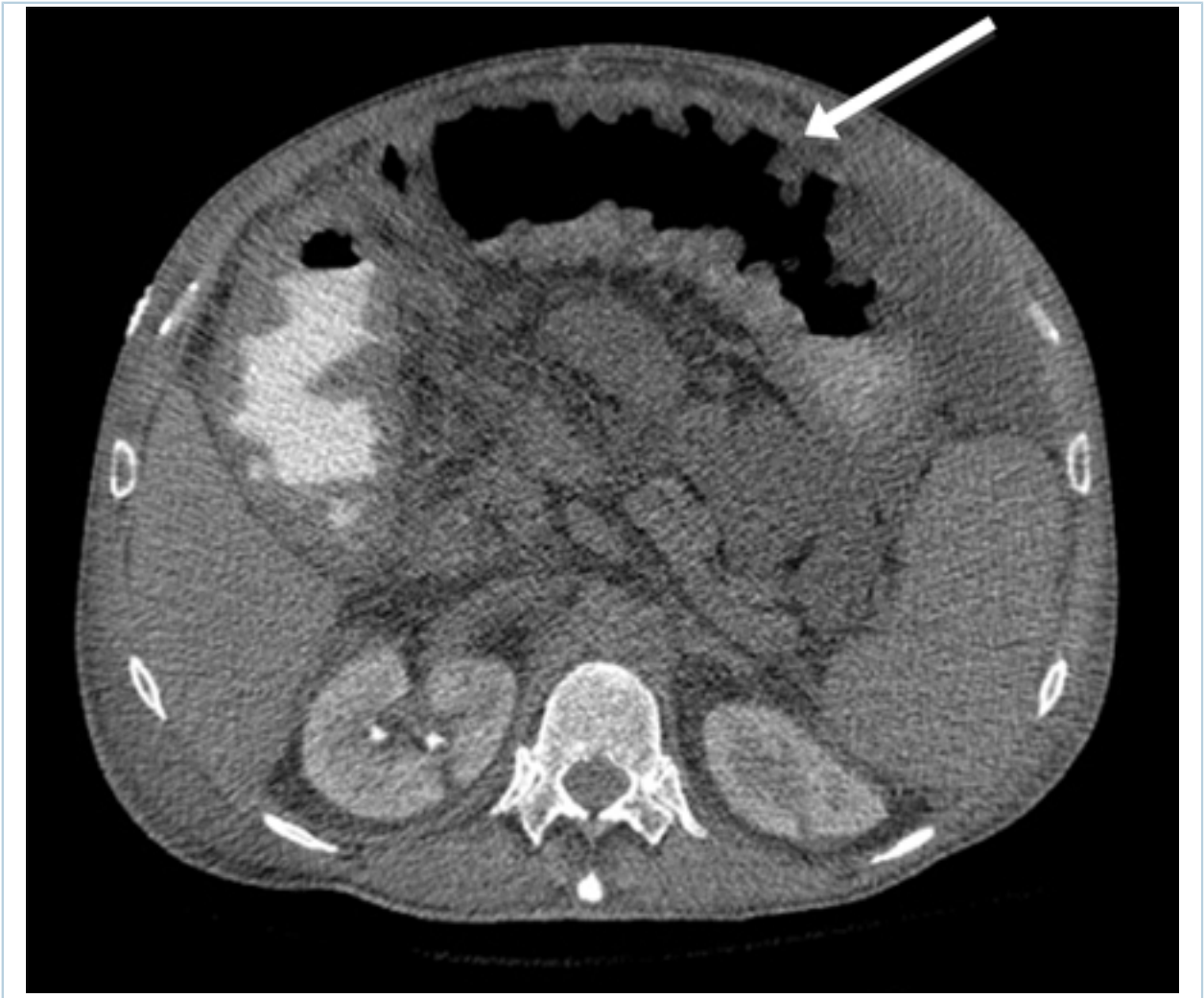


CT scan: colonic thickening with pneumatosis intestinalis
From the collection of Dr Jennifer Holder-Murray; used with permission



84-year-old man presenting with symptoms suggestive of ischaemic bowel disease: (A) Abdominal CT revealing a massive circumferential and band-like air formation as intestinal pneumatosis (arrows) and pronounced oedema of mesenteric fat (arrowhead) around necrotic bowel loops; (B) Another slice of abdominal CT showing long segmental pneumatosis of the small bowel

Lin I, Chang W, Shih S, et al. Bedside echogram in ischaemic bowel. BMJ Case Reports 2009:bcr.2007.053462

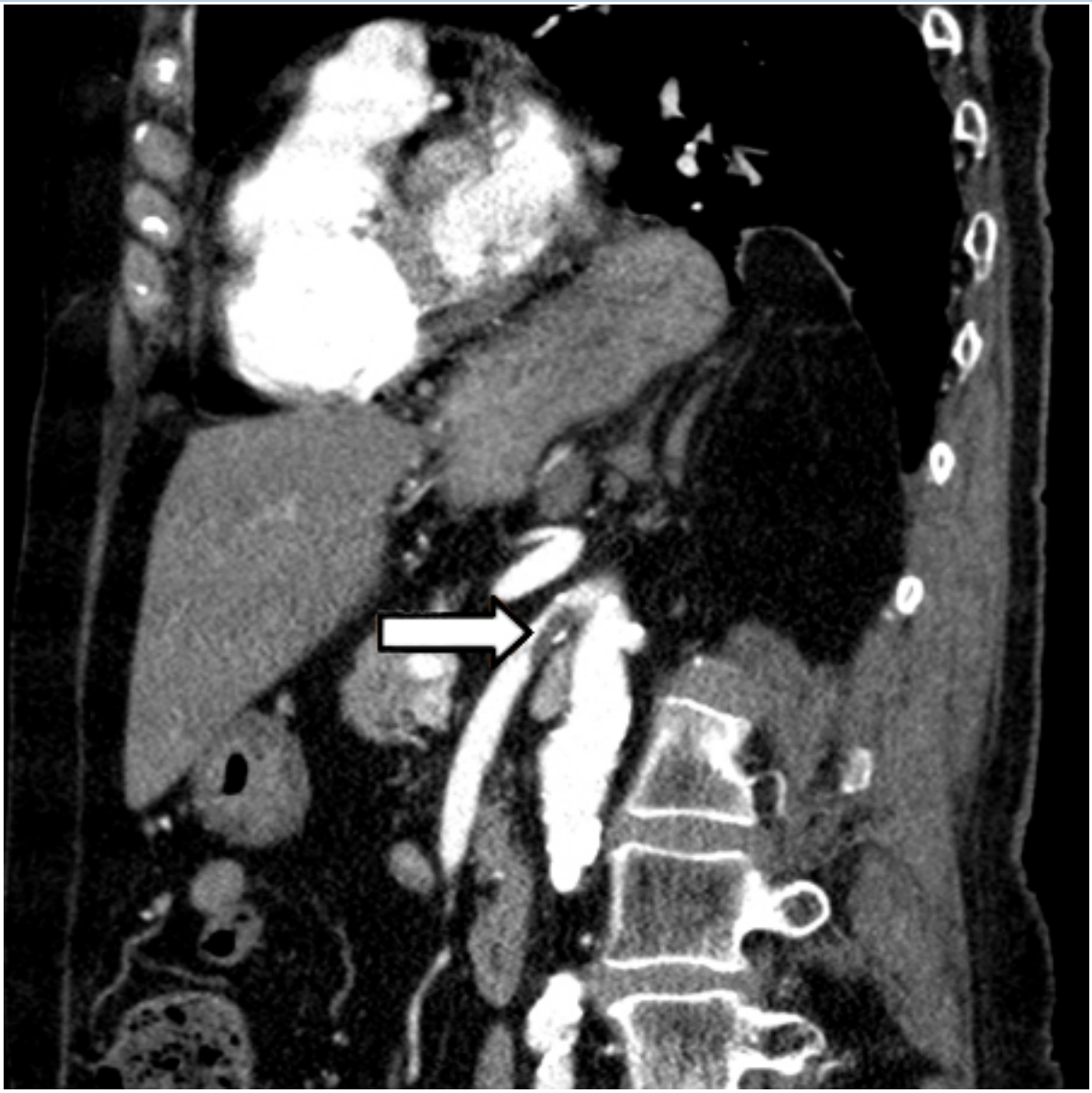


CT scan: circumferential wall thickening of the transverse colon; white arrow shows thumbprinting
From the collection of Dr Amir Bastawrous; used with permission

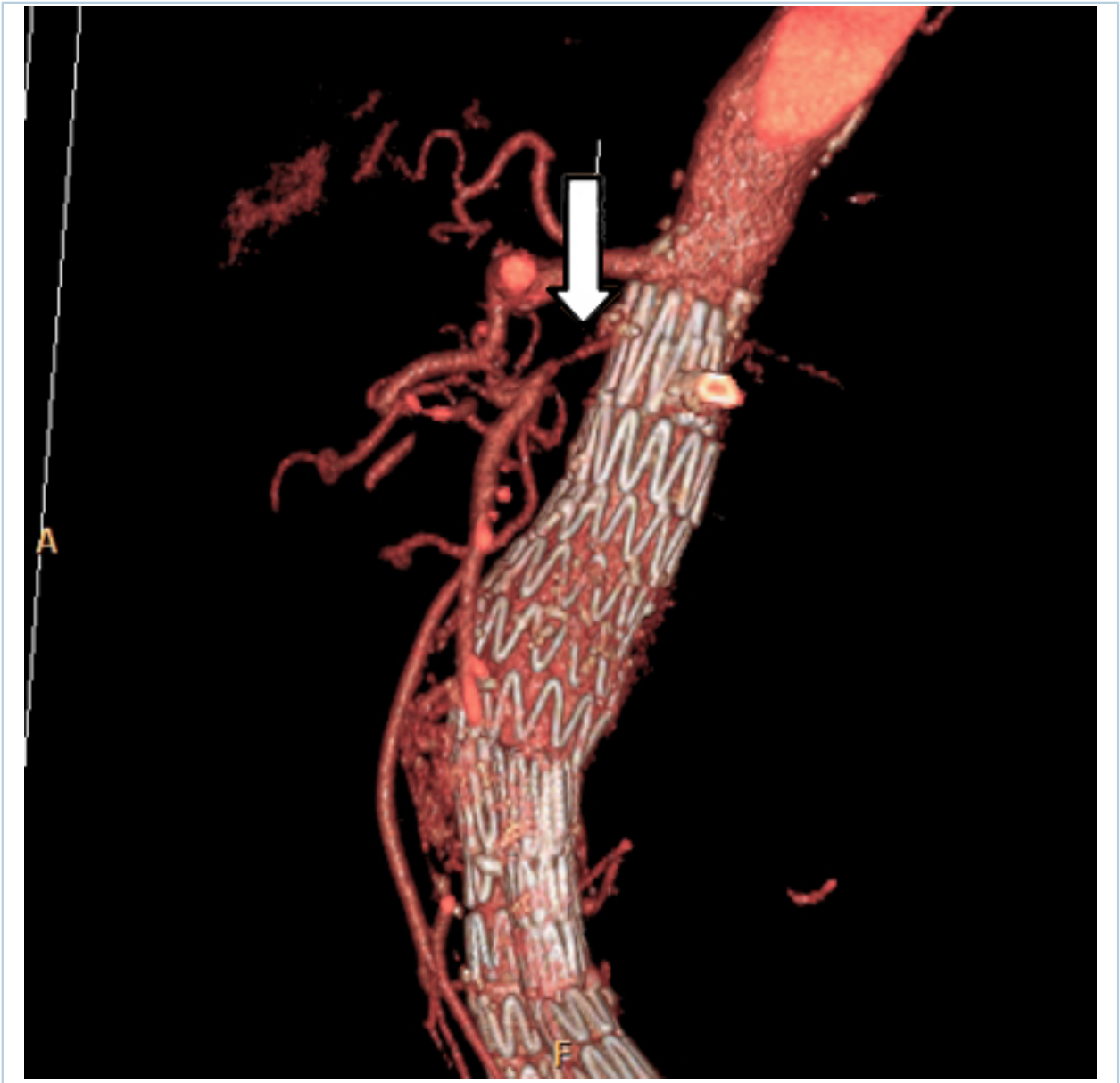
It also enables stratification of patients to differentiate those who would benefit from mesenteric angiography from those who require primary surgery; CT can also inform pathological staging; the degree of involvement of each layer of the gastrointestinal tract (mucosa, submucosa, muscularis, and serosa) can be used to estimate the probability of reversibility of the ischaemia, therefore helping to guide treatment.[42]

For diagnosing acute mesenteric vein thrombosis (MVT), contrast-enhanced CT is the procedure of choice, enabling diagnosis in >90% of patients. A central lucency in the mesenteric veins after injection of contrast is suggestive of a thrombosis. Other suggestive findings include enlargement of the superior mesenteric vein, thickening of the bowel wall, or dilated collaterals in a thickened mesentery. If MVT is diagnosed on CT scan, angiography may not be necessary, although it does provide better delineation of thrombosed veins and facility for intra-arterial vasodilators.[32]

Consider early CT angiography in a patient with abdominal pain and lactic acidosis.[8] CT angiography has replaced conventional angiography as standard practice for evaluation of the mesenteric vasculature and diagnosis of acute mesenteric ischaemia.[30] It can be used in the diagnosis of non-occlusive mesenteric ischaemia (NOMI).[43] CT angiography can also provide information to help decide on treatment modality, such as endovascular repair.



CT angiogram: Acute superior mesenteric artery thrombus
From the collection of Dr Jennifer Holder-Murray; used with permission



CT angiography: 3-dimensional reconstruction with superior mesenteric artery stenosis from severe atherosclerotic plaque in a patient on follow-up imaging for endovascular aneurysm repair
From the collection of Dr Jennifer Holder-Murray; used with permission

In ischaemic colitis, features of colitis, such as bowel wall thickening and pericolic fat stranding, may be seen on CT.[36] [44] Right-sided involvement seen on CT may indicate severe disease needing surgical intervention.[36] [44]

Magnetic resonance angiography

Magnetic resonance angiography may have a role in diagnosing chronic mesenteric ischaemia.[30] However, CT angiography is a better examination than magnetic resonance angiography (MRA) for the diagnosis of chronic mesenteric ischaemia because of its capacity for higher resolution in combination with faster scans.

The time required to perform MRA examinations, and the possible need for bowel stimulation with a meal, limit the usefulness of MRA in the diagnosis of acute mesenteric ischaemia.

Mesenteric angiography

Historically, mesenteric angiography has been the definitive test for diagnosing mesenteric ischaemia. In current practice it is usually preceded by positive CT angiography in the acute setting. Sensitivity is 74% to 100%, and specificity is 100%.[32]

Mesenteric angiography can diagnose NOMI before infarction occurs; look for:

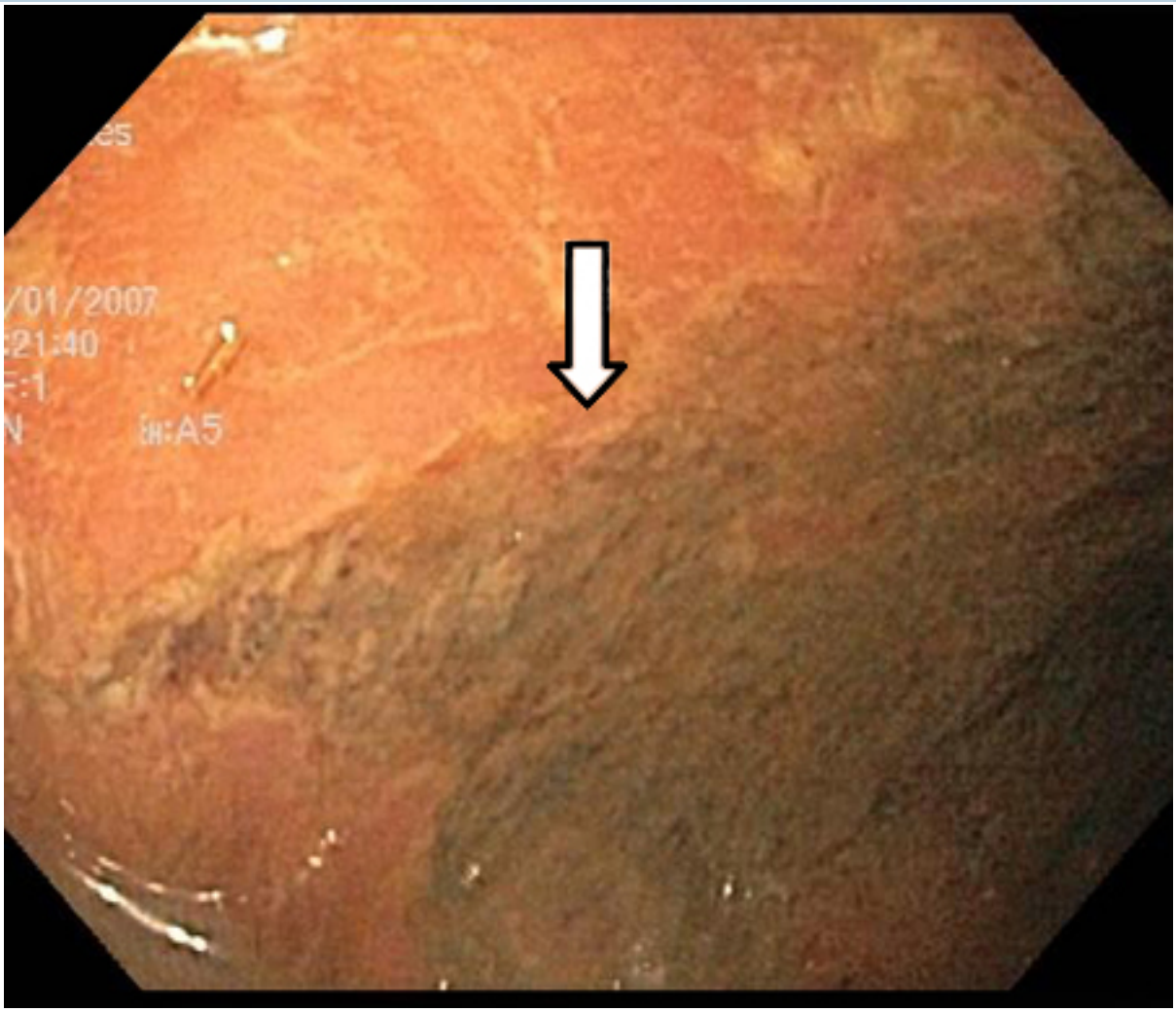
- Narrowing of the origins of the superior mesenteric artery branches
- Irregularities in these branches
- Spasm of the mesenteric arcades
- Impaired filling of the intramural vessels.

Mesenteric angiography is often performed with the intention of proceeding to intervention. It enables treatment by infusion of vasodilators or thrombolytic agents (which have been shown to improve outcomes).

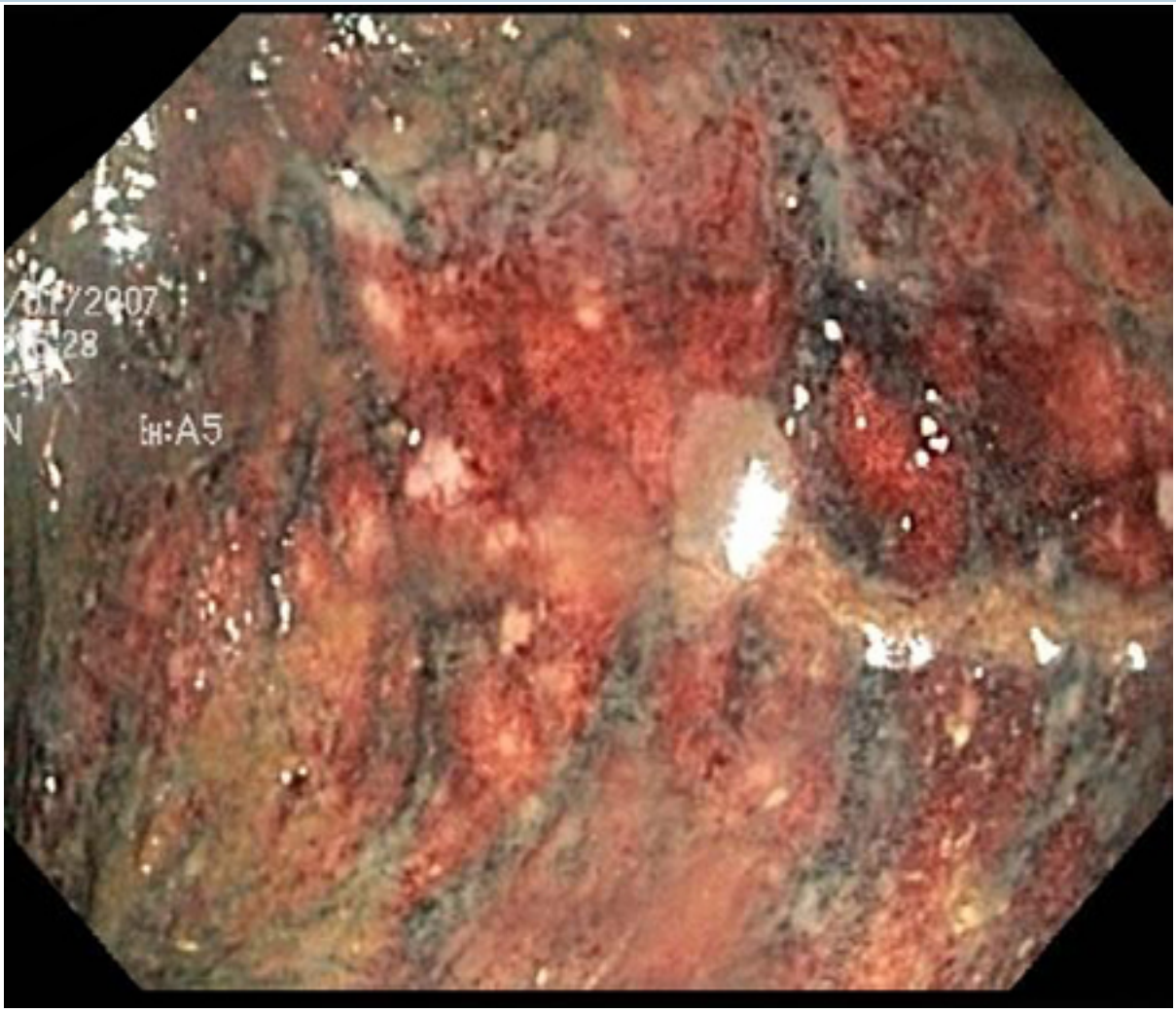
For the diagnosis of chronic mesenteric ischaemia, angiography needs to demonstrate severe occlusion of at least 2 of the 3 splanchnic vessels, although in the absence of symptoms an abnormal angiography result alone is not sufficient for diagnosis.[45]

Sigmoidoscopy or colonoscopy

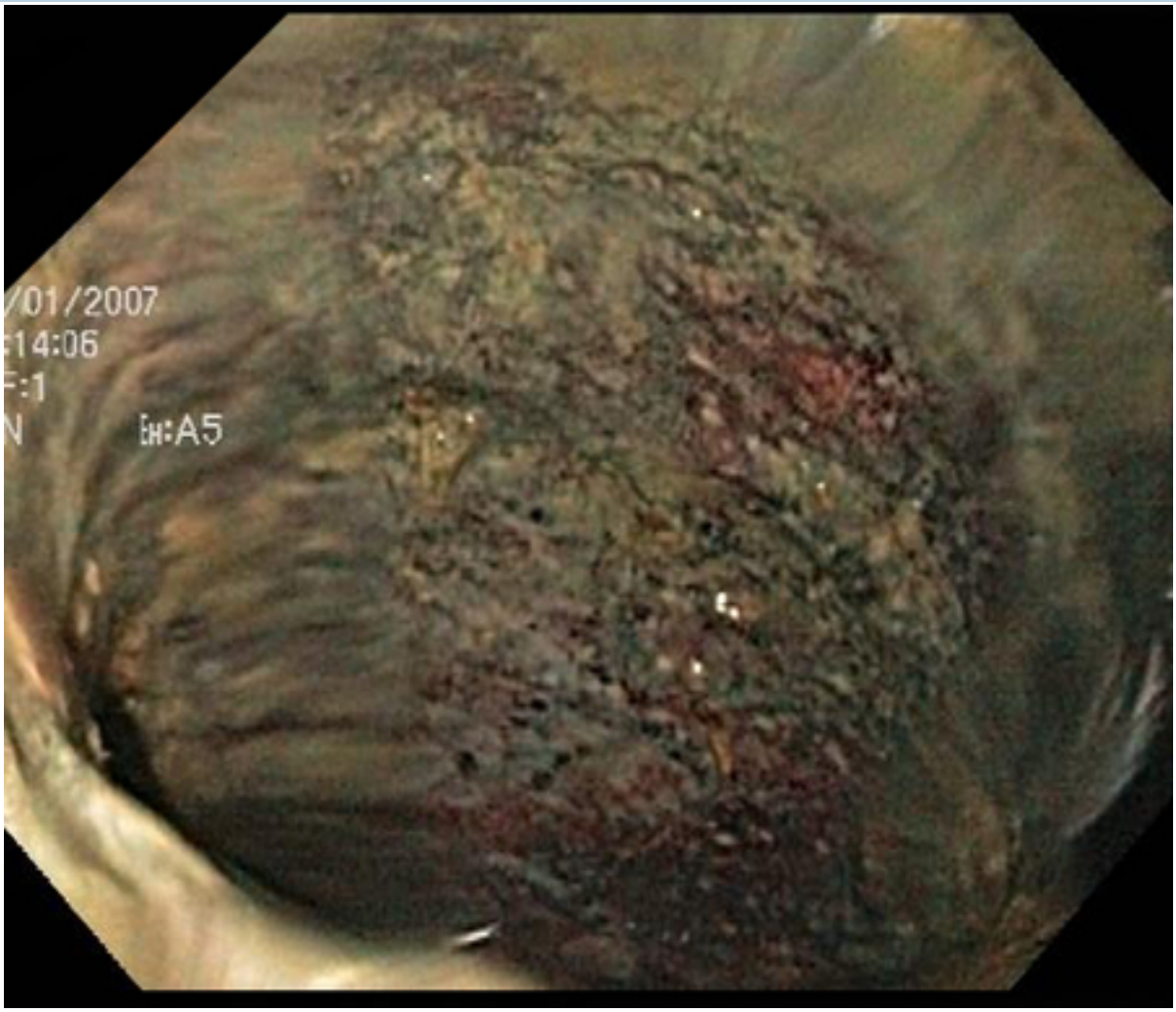
Sigmoidoscopy or colonoscopy can be used to establish the diagnosis of colonic ischaemia, establish severity, and exclude alternative causes of colonic inflammation. However, if urgent surgical intervention is required due to the condition of the patient, surgery should not be delayed to carry out sigmoidoscopy or colonoscopy.



Colonoscopy: demarcation between ischaemic and normal colon
From the collection of Dr Jennifer Holder-Murray; used with permission



Colonoscopy: denudation of colonic mucosa
From the collection of Dr Jennifer Holder-Murray; used with permission



Colonoscopy: mucosal sloughing and likely to be non-viable colon
From the collection of Dr Jennifer Holder-Murray; used with permission

Upper gastrointestinal endoscopy

Perform upper gastrointestinal endoscopy in patients with suspected chronic mesenteric ischaemia, in order to rule out alternative diagnoses involving the upper gastrointestinal tract.[30]

Mesenteric duplex ultrasound

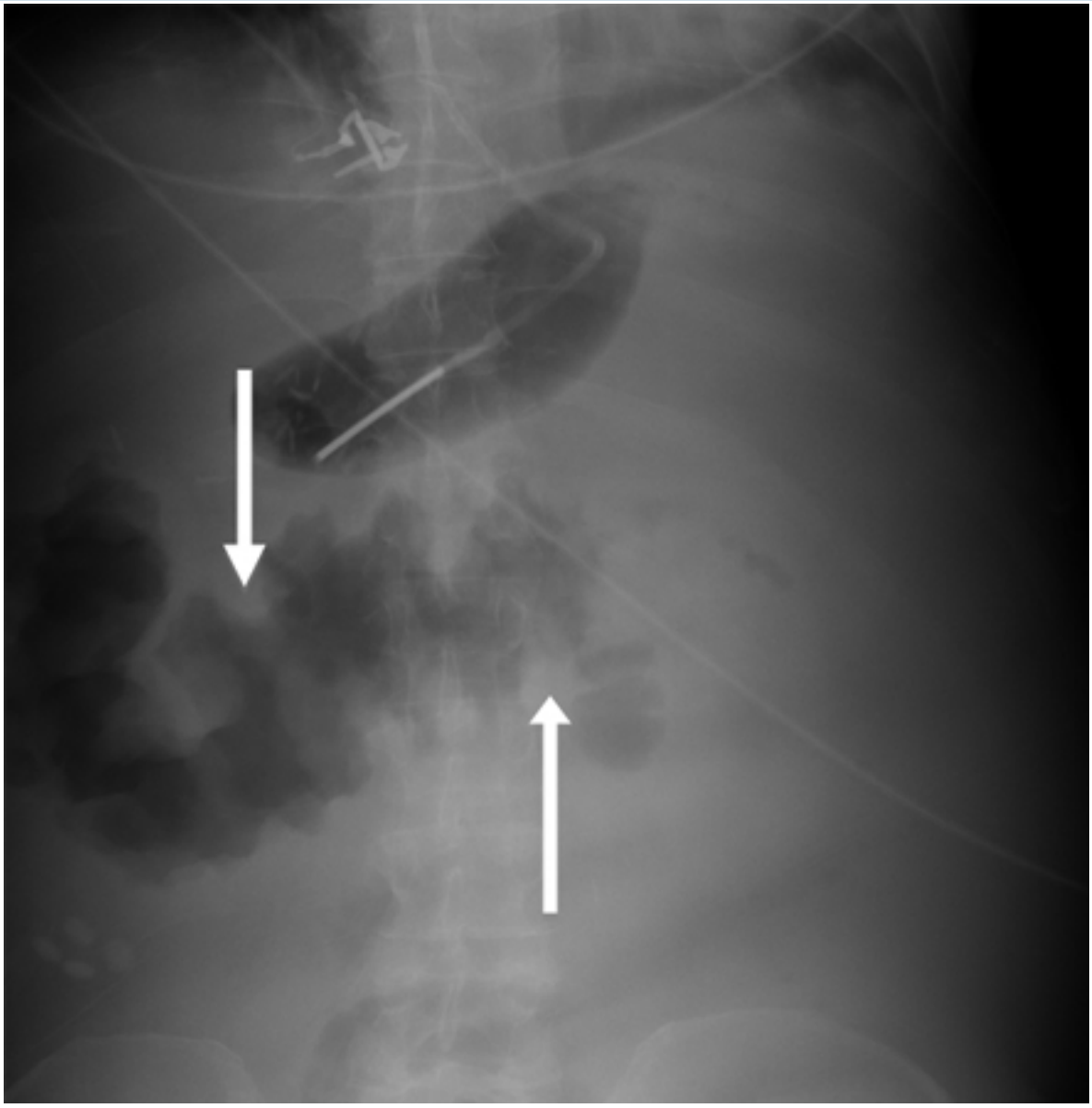
Mesenteric duplex ultrasound is particularly useful if obstruction is proximal in the mesenteric vessels but ultrasound cannot assess distal mesenteric blood vessel flow and non-occlusive aetiology of ischaemia.[9] This is mostly used in vascular units where it is the first-line investigation of choice for the assessment of chronic mesenteric ischaemia.[46]

Ultrasound of the bowel can be used to diagnose ischaemic colitis (providing the user has adequate expertise) and differentiate between left- and right-sided disease.[37] It is an alternative for patients unable to tolerate contrast media.[36] However, it is not considered a routine investigation and should only be performed by a radiologist with sufficient ultrasound expertise.

Abdominal x-ray

Abdominal x-ray has a limited role in the diagnosis and evaluation of acute mesenteric ischaemia.[8] [20] A negative radiograph does not exclude the diagnosis.[8] Plain x-rays are often normal early in the course of ischaemia or when ischaemia is mild. X-rays are not a frequently used investigation for ischaemic bowel disease.

With worsening ischaemia, plain x-rays may show formless loops of bowel, ileus, or thickening of the bowel wall with thumbprinting sign suggestive of submucosal oedema or haemorrhage.



Plain abdominal x-ray: shows marked wall thickening of the transverse colon compatible with the finding of thumbprinting (white arrows)
From the collection of Dr Amir Bastawrous; used with permission

Erect chest x-ray

Chest X-ray may show sub-diaphragmatic air, indicative of perforation of the bowel requiring prompt surgical intervention. X-rays are not a frequently used investigation for ischaemic bowel disease.

History and exam

Key diagnostic factors

abdominal pain (common)

The majority of patients with ischaemic bowel experience pain, which is poorly localised and can vary depending on the type and segment of bowel involved. The classic presentation of acute mesenteric ischaemia is **abdominal pain out of proportion to examination**.

In patients with chronic mesenteric ischaemia, abdominal pain with postprandial worsening is a typical presenting symptom.[30] Abdominal pain may worsen during exercise and often occurs after meals, gradually resolving over a few hours.[30]

abdominal tenderness (common)

In colonic ischaemia, tenderness to palpation over the affected bowel may be noticed from early in the course of ischaemia, in contrast to acute mesenteric ischaemia, where tenderness is a relatively late sign.

Perceived pain may be out of proportion to tenderness appreciated on physical examination in acute mesenteric ischaemia.

presence of risk factors (common)

Older age, smoking, and a history of medical conditions such as atrial fibrillation, myocardial infarction, structural heart defects, vasculitis, and hypercoagulable states are risk factors for ischaemic bowel disease.

Other diagnostic factors

haematochezia/melaena (common)

Intestinal ischaemia leads to mucosal sloughing that can cause blood loss into the bowel lumen.

Depending on the size of the vessels involved and their position within the bowel, this blood loss may manifest as anything on the spectrum from fresh or partially altered blood (colonic or brisk proximal bleeding) to melaena (proximal small bowel).

If bleeding is severe, this can potentially cause further hypoperfusion and worsening of ischaemia.

diarrhoea (common)

Diarrhoea may occur.[30] Mucosal sloughing occurs due to intestinal ischaemia, frequently causing episodes of diarrhoea.

nausea (common)

Nausea may occur.[30]

weight loss (common)

This is a notable feature of chronic mesenteric ischaemia, which is usually related to avoidance of or a fear of food (sitophobia) in these patients.[19] [30]

abdominal bruit (common)

Physical examination may reveal an epigastric bruit in 48% to 63% of patients with bowel ischaemia, indicative of turbulent flow through an area of vascular narrowing.[19]

vasculitis (uncommon)

Clinical picture may vary depending on the size of the mesenteric vessel involved.

light headedness, pallor, dyspnoea (uncommon)

Anaemia may occur as a result of repeated episodes of melaena.

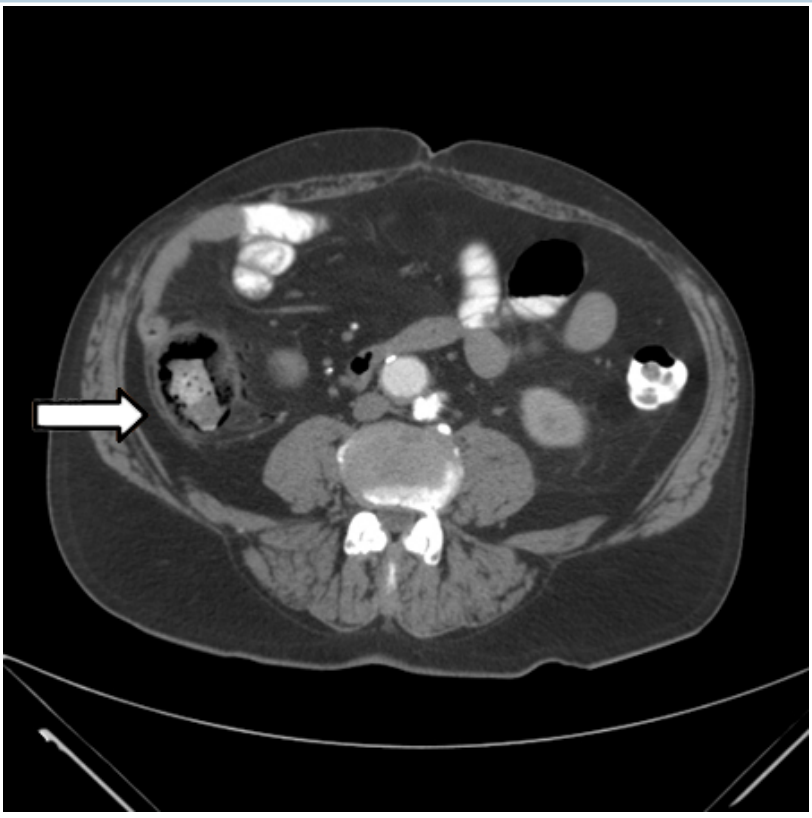
food fear (sitophobia) (uncommon)

Chronic ischaemia results in symptoms related to oral intake; food fear and food avoidance may develop.[30] This should not be confused with anorexia due to acute onset of pain or discomfort.

Investigations

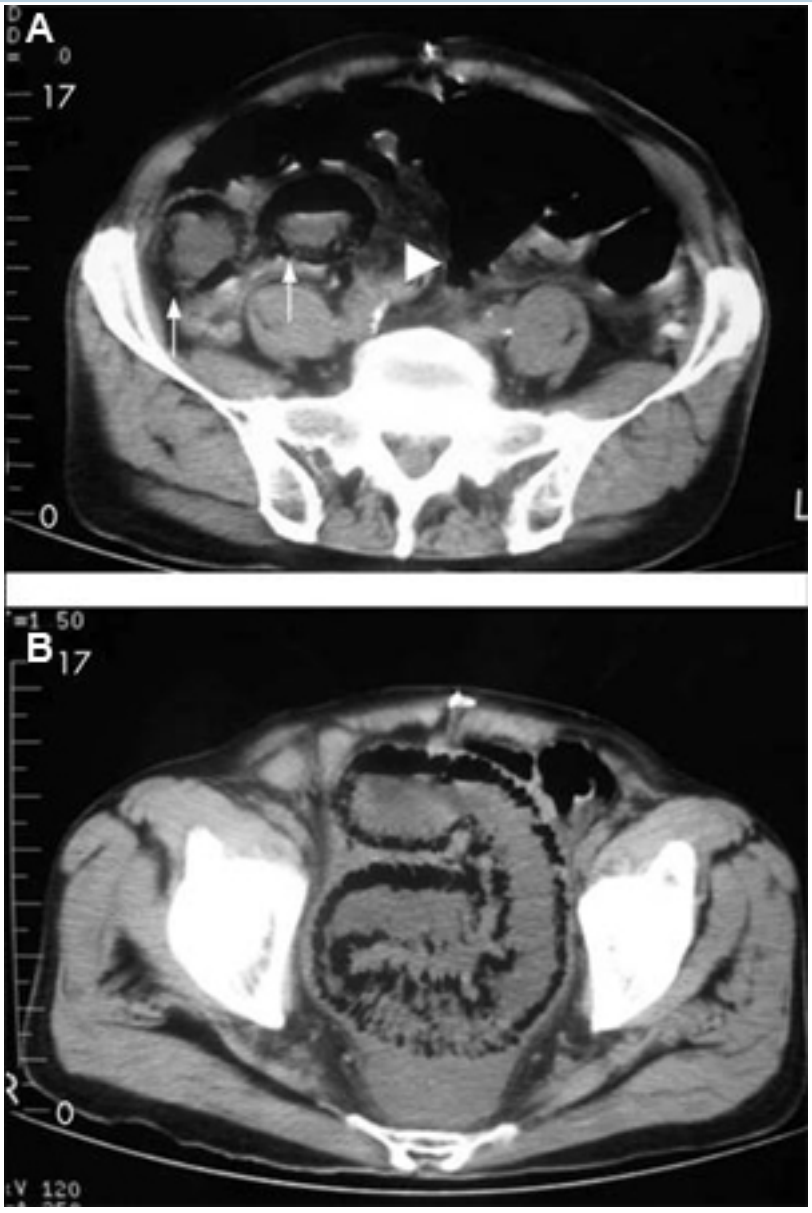
1st test to order

Test	Result
CT scan with contrast/CT angiogram **Prioritise an urgent CT scan of the abdomen** if you suspect acute or chronic mesenteric ischaemia.[8] [20] [30] Obtain the scan early; prompt diagnosis (and intervention) is essential to improve the clinical outcome. Use multidetector CT scanning with intravenous contrast for suspected acute mesenteric ischaemia.[20] Consider a CT scan even in the presence of renal impairment in order to save life and prevent worsening renal injury.[8] [9][20] CT provides evidence for the extent of bowel compromise from ischaemia.[39] [40] It also enables stratification of patients to differentiate those who would benefit from mesenteric angiography from those who require primary surgery; CT can inform pathological staging, the degree of involvement of each layer of the gastrointestinal tract (mucosa, submucosa, muscularis, and serosa) can be used to estimate the probability of reversibility of the ischaemia, therefore helping to guide treatment.[42]	bowel wall thickening, bowel dilation, pneumatosis intestinalis, portal venous gas, occlusion of the mesenteric vasculature, bowel wall thickening with thumbprinting sign suggestive of submucosal oedema or haemorrhage



CT scan: colonic thickening with pneumatosis intestinalis
From the collection of Dr Jennifer Holder-Murray; used with permission

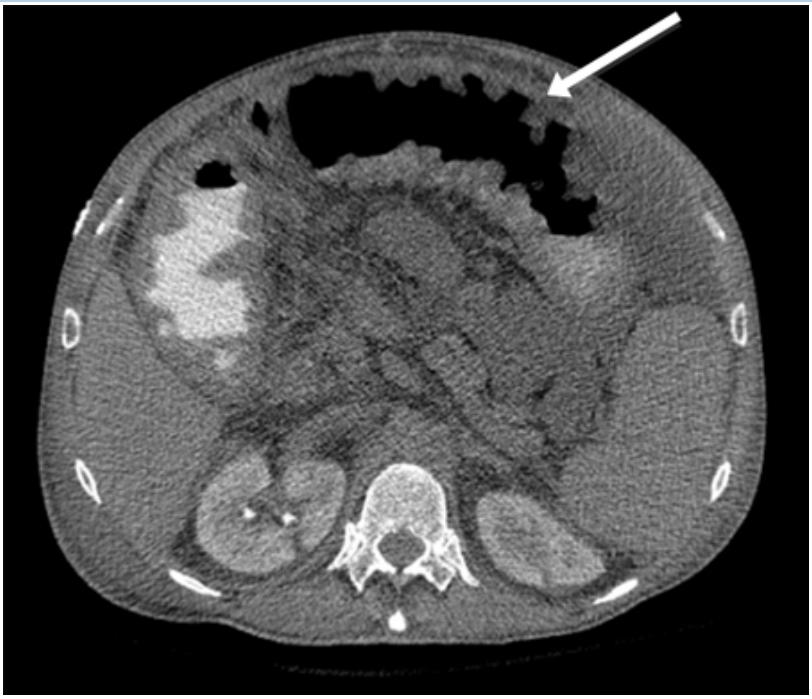
Test	Result



84-year-old man presenting with symptoms suggestive of ischaemic bowel disease: (A) Abdominal CT revealing a massive circumferential and band-like air formation as intestinal pneumatosis (arrows) and pronounced oedema of mesenteric fat (arrowhead) around necrotic bowel loops; (B) Another slice of abdominal CT showing long segmental pneumatosis of the small bowel
Lin I, Chang W, Shih S, et al. Bedside echogram in ischaemic bowel. BMJ Case Reports 2009:bcr.2007.053462

Presence of the thumbprinting sign, indicative of mucosal oedema, suggests a worse prognosis.

Test	Result



CT scan: circumferential wall thickening of the transverse colon; white arrow shows thumbprinting
From the collection of Dr Amir Bastawrous; used with permission

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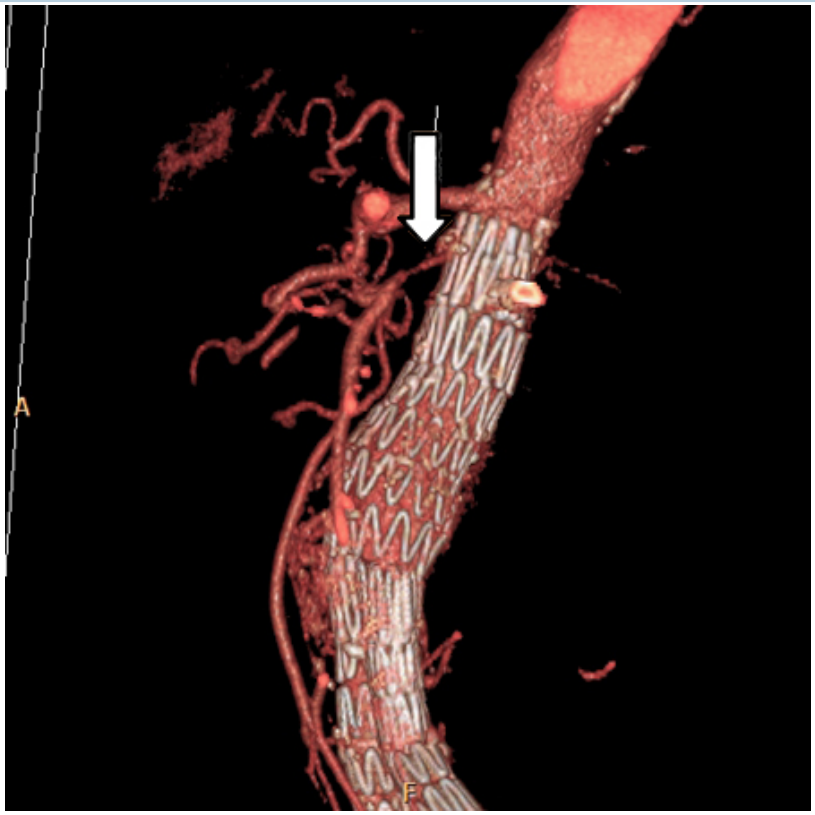
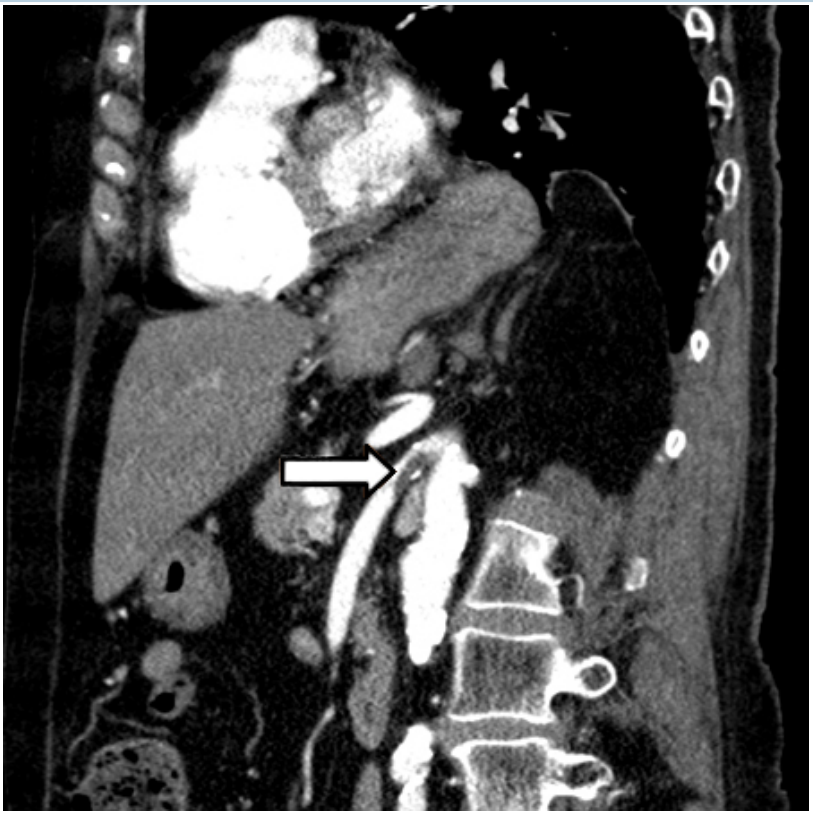
CT angiography has replaced conventional angiography as standard practice for diagnosis of acute mesenteric ischaemia. It can be used in the diagnosis of non-occlusive mesenteric ischaemia (NOMI).[43] CT angiography can also provide information to help decide on treatment modality, such as endovascular repair.

Test	Result

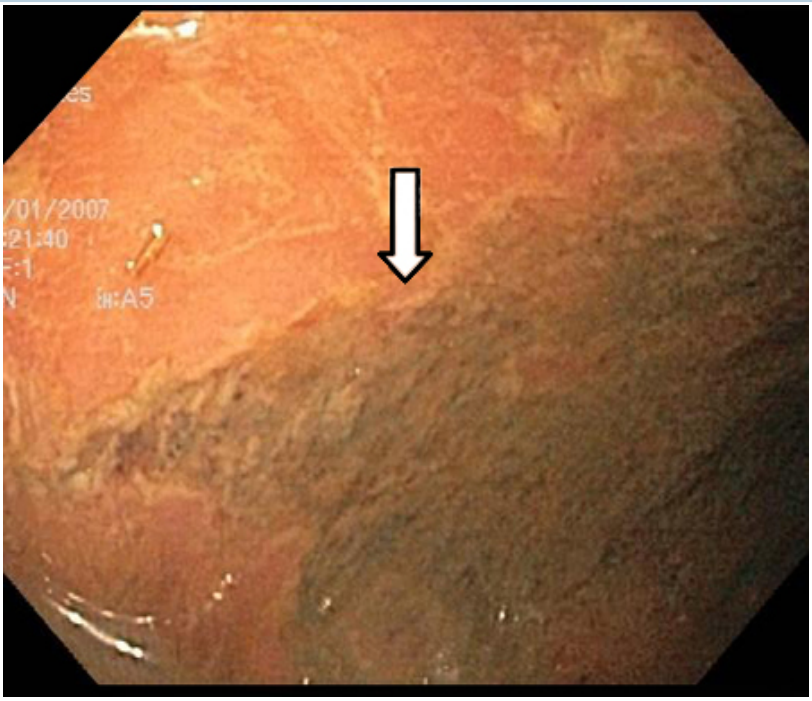


CT angiography: 3-dimensional reconstruction with superior mesenteric artery stenosis from severe atherosclerotic plaque in a patient on follow-up imaging for endovascular aneurysm repair
From the collection of Dr Jennifer Holder-Murray; used with permission

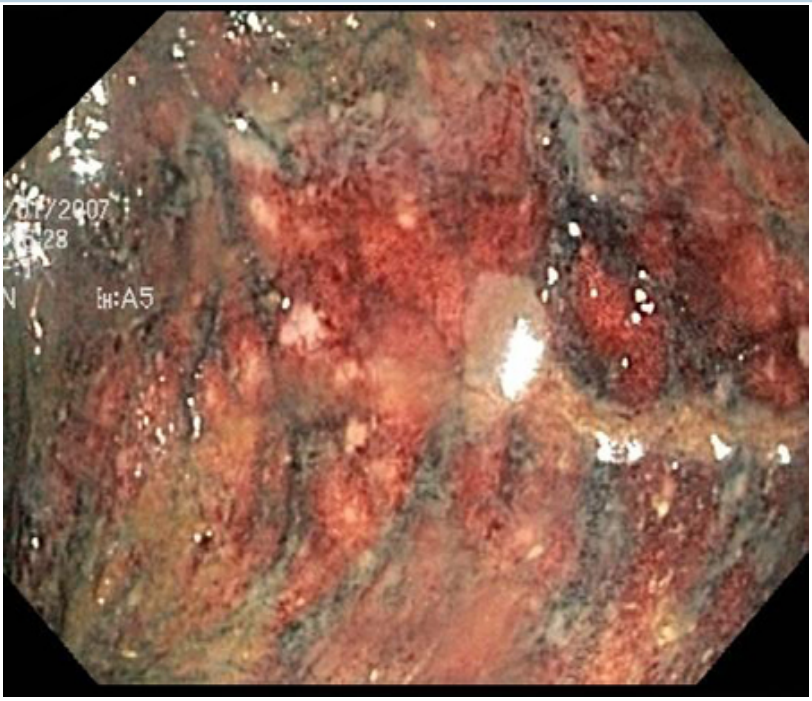
Test	Result
 *CT angiogram: Acute superior mesenteric artery thrombus* *From the collection of Dr Jennifer Holder-Murray; used with permission* In ischaemic colitis, features of colitis such as bowel wall thickening and pericolic fat stranding may be seen on CT.[36] [37] Right-sided involvement seen on CT may indicate severe disease needing surgical intervention.[36]	
FBC More than 90% of patients with acute mesenteric ischaemia will have an abnormally elevated leukocyte count.[8] May reveal anaemia (often as a result of repeated episodes of melaena) that exacerbates ischaemia.	leukocytosis, anaemia, evidence of haemoconcentration
arterial blood gases and serum lactate Metabolic acidosis is a common finding in patients with acute mesenteric ischaemia.[8] Elevated serum lactate does not determine the presence or absence of ischaemic or necrotic bowel; however, it can be used to assist with making the diagnosis and determining the severity of the illness.[8]	acidosis
urea and electrolytes Helps assess renal dysfunction and dehydration, frequently present in patients with ischaemic bowel disease.	uraemia, elevated creatinine
liver function tests Check for derangement.	may be elevated as a consequence of septic shock

Test	Result
	or concomitant with bowel ischaemia
CRP Request in suspected ischaemic colitis.[36]	elevated in the presence of inflammation; not specific but may be indicative of disease severity
coagulation studies, group and save and crossmatch Aids in the diagnosis of any underlying coagulopathy as a risk factor for further thrombosis. Allows correction of any clotting dyscrasia as part of treatment. Group and save in preparation for the possibility of transfusion.	underlying prothrombotic disorder
ECG Perform as part of the cardiovascular examination. May demonstrate arrhythmias that predispose to cardioembolic complications, such as atrial fibrillation or atrial flutter, or acute infarction that may be the aetiology of intestinal ischaemia.	atrial fibrillation, atrial flutter, acute myocardial infarction
erect CXR Chest x-ray may show sub-diaphragmatic air, indicative of perforation of the bowel requiring prompt surgical intervention. X-rays are not a frequently used investigation for ischaemic bowel disease.	free air if perforation present
sigmoidoscopy or colonoscopy Sigmoidoscopy or colonoscopy can be used to establish the diagnosis of colonic ischaemia, establish severity, and exclude alternative causes of colonic inflammation. However, if urgent surgical intervention is required due to the condition of the patient, surgery should not be delayed to carry out sigmoidoscopy or colonoscopy.	mucosal sloughing or friability; mucosal petechiae; submucosal haemorrhagic nodules, erosions, or ulcerations; submucosal oedema; luminal narrowing; necrosis, gangrene

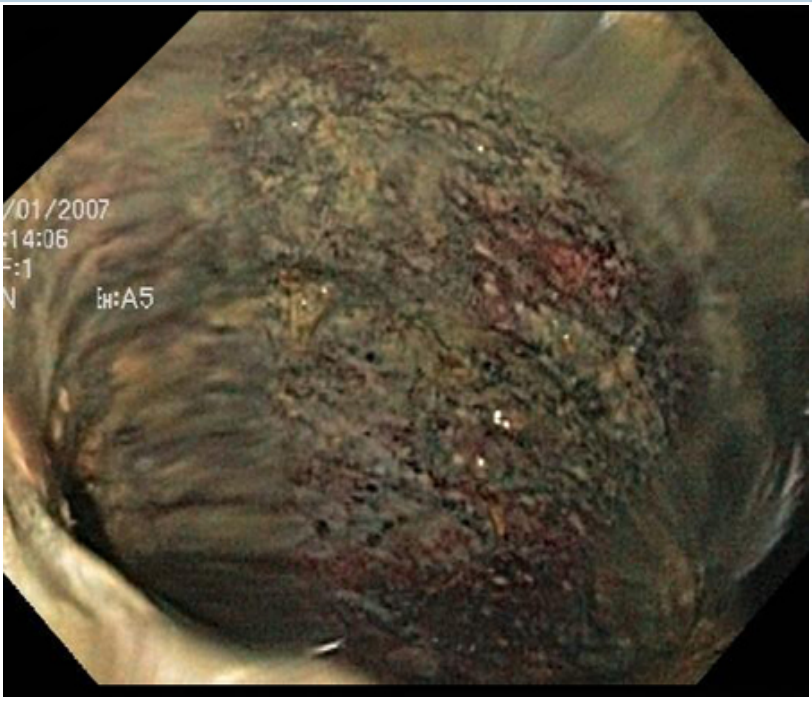
Test	Result



Colonoscopy: demarcation between ischaemic and normal colon
From the collection of Dr Jennifer Holder-Murray; used with permission



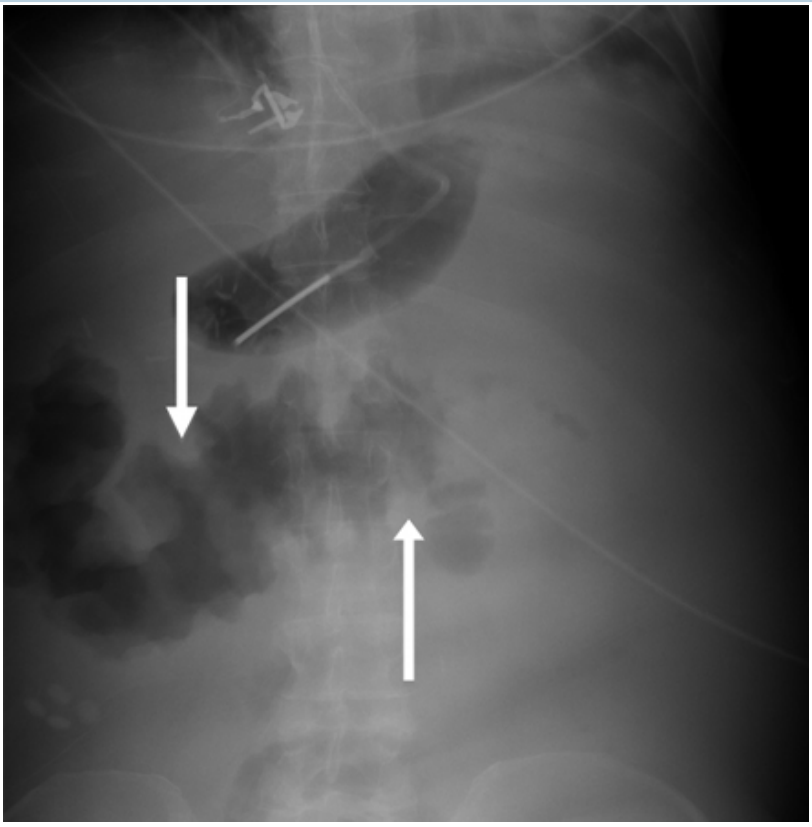
Colonoscopy: denudation of colonic mucosa
From the collection of Dr Jennifer Holder-Murray; used with permission

Test	Result
 *Colonoscopy: mucosal sloughing and likely to be non-viable colon* *From the collection of Dr Jennifer Holder-Murray; used with permission*	
upper gastrointestinal endoscopy Perform upper gastrointestinal endoscopy in patients with suspected chronic mesenteric ischaemia, in order to rule out alternative diagnoses involving the upper gastrointestinal tract.[30]	normal in patients with chronic mesenteric ischaemia
D-dimer May be elevated in intestinal ischaemia, but its use is limited because D-dimer is a very non-specific test.[8]	may be elevated

Other tests to consider

Test	Result
mesenteric angiography Historically, mesenteric angiography has been the definitive test for diagnosing mesenteric ischaemia. In current practice it is usually preceded by positive CT angiography in the acute setting. Sensitivity is 74% to 100%, and specificity is 100%. Mesenteric angiography can diagnose non-occlusive mesenteric ischaemia before infarction occurs. Look for: • Narrowing of the origins of the superior mesenteric artery branches • Irregularities in these branches • Spasm of the mesenteric arcades • Impaired filling of the intramural vessels. Mesenteric angiography is often performed with the intention of proceeding to intervention. It enables treatment by infusion of vasodilators or thrombolytic agents (which have been shown to improve outcomes). For the diagnosis of chronic mesenteric ischaemia, angiography needs to demonstrate severe occlusion of at least 2 of the 3 splanchnic vessels, although in the absence of symptoms an abnormal angiography result alone is not sufficient for diagnosis.[45]	proximal defect of a mesenteric vessel or vasoconstriction of all mesenteric arcades
mesenteric duplex ultrasound Mesenteric duplex ultrasound is particularly useful if obstruction is proximal in the mesenteric vessels, but ultrasound cannot assess distal mesenteric blood vessel flow and non-occlusive aetiology of ischaemia.[9] This is mostly used in vascular units where it is the first-line investigation of choice for the assessment of chronic mesenteric ischaemia.[46] Ultrasound of the bowel can be used to diagnose ischaemic colitis (providing the user has adequate expertise) and differentiate between left- and right-sided disease.[37] It is an alternative for patients unable to tolerate contrast media.[36] However, it is not considered a routine investigation and should only be performed by a radiologist with sufficient ultrasound expertise.	reduced or lack of blood flow through proximal mesenteric vessels
magnetic resonance angiography Magnetic resonance angiography may have a role in diagnosing chronic mesenteric ischaemia.[30] However, CT angiography is a better examination than MRA for the diagnosis of chronic mesenteric ischaemia because of its capacity for higher resolution in combination with faster scans. The time required to perform MRA examinations, and the possible need for bowel stimulation with a meal, limit the usefulness of MRA in the diagnosis of acute mesenteric ischaemia.	narrowing or obstruction of mesenteric vasculature; decreased bowel wall enhancement

Test	Result
amylase Elevated serum amylase is found in approximately half of patients with acute mesenteric ischaemia.[8]	possibly elevated
studies for ova, cysts, and parasites Request in suspected ischaemic colitis.[36] [37]	determines presence of infection
faecal culture Request in suspected ischaemic colitis.[36] [37]	determines presence of infection
Clostridium difficile toxin assay Request in suspected ischaemic colitis.[36] [37]	determines presence of infection
abdominal x-rays Abdominal x-ray has a limited role in the diagnosis and evaluation of acute mesenteric ischaemia.[8] [20] A negative radiograph does not exclude the diagnosis.[8] Plain x-rays are often normal early in the course of ischaemia or when ischaemia is mild. X-rays are not a frequently used investigation for ischaemic bowel disease. With worsening ischaemia, plain x-rays may show formless loops of bowel, ileus, or thickening of the bowel wall with thumbprinting sign suggestive of submucosal oedema or haemorrhage.	air-fluid levels, bowel dilation, bowel wall thickening, pneumatosis

Test	Result
 *Plain abdominal x-ray: shows marked wall thickening of the transverse colon compatible with the finding of thumbprinting (white arrows)* *From the collection of Dr Amir Bastawrous; used with permission*	

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Infectious colitis	• May have similar clinical features.	• Colonoscopy will demonstrate if ischaemia or pseudomembranes are present. • Stool cultures may reveal causative organism. • CT may show marked thickening of the colon with bacterial infection.
Ulcerative colitis	• Form of inflammatory bowel disease that affects the rectum and extends proximally. Characterised by diffuse inflammation of the colonic mucosa and a relapsing, remitting course. • Patients commonly experience bloody diarrhoea, chronic diarrhoea (or both), lower abdominal pain, faecal urgency, and extraintestinal manifestations, particularly those related to activity of the colitis.	• Diagnosis requires endoscopy with biopsy and negative stool culture.
Crohn's disease	• Inflammatory bowel disease that may involve the entire gastrointestinal tract. • Common presenting symptoms include chronic diarrhoea, weight loss, and right lower quadrant abdominal pain mimicking acute appendicitis.	• Diagnosis confirmed by colonoscopy with ileoscopy and tissue biopsy.
Diverticular disease	• Symptomatic disease may have similar clinical features to bowel ischaemia. Fever common in diverticulitis; diarrhoea common, usually no haematochezia.	• CT may demonstrate focal colonic thickening and evidence of diverticulosis and diverticulitis.
Large bowel obstruction	• Obstipation may be a symptom. • Closed loop obstruction, in particular, can lead to ischaemia.	• In most instances, CT will demonstrate cause of obstruction, such as tumour, internal hernia, or volvulus.
Peptic ulcer disease	• Pain is generally epigastric and less severe, but may be generalised abdominal	• Oesophagogastroduodenoscopy will demonstrate gastritis and ulcers.

Condition	Differentiating signs / symptoms	Differentiating tests
	discomfort. Nausea and vomiting are common. Usually symptoms are less acute.	• Erect chest x-ray will demonstrate pneumoperitoneum in patients with perforated peptic ulcer disease.
Small bowel obstruction	• Often have a history of previous abdominal surgery. Nausea, vomiting and abdominal distension are the predominant features.	• CT will show dilated proximal small bowel with distal decompression, with a possible transition point.
Acute pancreatitis	• May provide a history of gallstones or recent alcohol use. Pain usually focal at epigastrium and radiates to back. Usually no diarrhoea or haematochezia.	• Elevated serum amylase and lipase (usually much higher than in bowel ischaemia). Abdominal ultrasound and CT demonstrate pancreatic inflammation and may show related gall bladder pathology.
Gastroenteritis	• May have similar clinical features. Possible history of ill contacts. May have a significant component of nausea and vomiting. Pain is often less profound than in patients with ischaemia.	• CT may demonstrate thickened loops of small bowel or mesenteric lymphadenopathy without evidence of ischaemia or infarction.

Recommendations

Key Recommendations

Acute mesenteric ischaemia is a medical **emergency**. **Urgent surgery** is required if there is evidence on computed tomography scan of peritonitis, infarction, or perforation.

Liaise early with surgery and the intensive care unit.

Treatment involves a combination of resuscitation and supportive care, antibiotics, endovascular therapy (if there is no evidence of peritonitis, infarction, or perforation), or surgery.

The optimal approach will depend on the anatomical location and severity of ischaemia, its underlying pathophysiology, and time course.

Full Recommendations

Resuscitation and supportive measures

Administer adequate fluid resuscitation and supplemental oxygen to optimise tissue perfusion and oxygenation.[20] [38]

Check your local protocols for specific recommendations on fluid choice. There is debate, based on conflicting evidence, on whether there is a benefit in using normal saline or balanced crystalloid in critically ill patients.

Monitor controlled oxygen therapy. An upper SpO_2 limit of 96% is reasonable when administering supplemental oxygen to most patients with acute illness who are not at risk of hypercapnia.

- Evidence suggests that liberal use of supplemental oxygen (target SpO_2 >96%) in acutely ill adults is associated with higher mortality than more conservative oxygen therapy.[48]
- A lower target SpO_2 of 88% to 92% is appropriate if the patient is at risk of hypercapnic respiratory failure.[49]

Initial resuscitation should also aim to address any acute heart failure and correct any cardiac arrhythmias.

Involve the critical care team for further management as necessary.

Consider invasive monitoring as appropriate. Nil by mouth status should be enforced, with nasogastric tube decompression for symptomatic relief.[8]

More info: Sepsis

Think ' ***Could this be sepsis?*** ' based on acute deterioration in an adult patient in whom there is clinical evidence or strong suspicion of infection.[50] [51] [52] See Sepsis in adults .

- The patient may present with non-specific or non-localised symptoms (e.g., acutely unwell with a normal temperature) or there may be severe signs with evidence of multi-organ dysfunction and shock.[50] [51] [52]
- Remember that sepsis represents the severe, life-threatening end of infection.[53]
- Ischaemic bowel disease and bowel perforation can cause rapid deterioration into septic shock.[54] For this reason, the assessment of severity status and subsequent action should be upgraded according to patient need, and at least to the next NEWS score band.[54] There should be no avoidable delay in giving antimicrobials and achieving source control.[54] In practice, if computed tomography (CT) shows extensive ischaemia in a patient who is very frail or has significant comorbidities, palliative care may be the treatment of choice (rather than antibiotics and source control); this decision should always be made in discussion with a consultant.

Use a systematic approach (e.g., National Early Warning Score 2 [NEWS2]), alongside your clinical judgement, to assess the risk of deterioration due to sepsis.[50] [51] [55] [56] [52] Consult local guidelines for the recommended approach at your institution.

Arrange urgent review by a senior clinical decision-maker (e.g., ST3 level doctor in the UK) if you suspect sepsis:[54]

- **Within 30 minutes** for a patient who is critically ill (e.g., NEWS2 score of 7 or more, evidence of septic shock, or other significant clinical concerns).
    - A patient is also at high risk of severe illness or death from sepsis if they have a NEWS2 score below 7 and a single parameter contributes 3 points to their NEWS2 score and a medical review has confirmed that they are at high risk.
- **Within 1 hour** for a patient who is severely ill (e.g., NEWS2 score of 5 or 6) or **within 1 hour** of any intervention for suspected sepsis (antibiotics/fluid resuscitation/oxygen) if there is no improvement in the patient's condition.

Follow your local protocol for investigation and treatment of all patients with suspected sepsis, or those at risk. Start treatment promptly. Determine urgency of treatment according to likelihood of infection and severity of illness, or according to your local protocol.[54] [56]

In the community and in custodial settings: refer for emergency medical care in hospital (usually by blue-light ambulance in the UK) any patient who is acutely ill with a suspected infection and is:[52]

- Deemed to be at high risk of deterioration due to organ dysfunction (as measured by risk stratification)
- At risk of neutropenic sepsis.

Acute mesenteric ischaemia

Acute mesenteric ischaemia is a **medical emergency**. Liaise early with surgery and intensive care unit colleagues.

Patients with delayed presentation and abdominal signs of peritonitis or organ failure generally have a worse prognosis than patients without these clinical factors.[20] It may be appropriate to discuss palliative care options with the multidisciplinary team if the patient is unlikely to benefit from invasive procedures.[20]

Antibiotics

Consult your local antimicrobial guidelines to administer empirical antibiotics suitable for enteric coverage (e.g., a third-generation cephalosporin plus metronidazole in critically ill patients, or a fluoroquinolone-based regimen in patients with beta-lactam allergy such as ciprofloxacin plus metronidazole) to all patients who are being treated with curative intent. Ischaemia can lead to significant bacterial translocation due to damage to the normal intestinal mucosal barrier.[8] [57]

Antibiotics may not be indicated if a palliative treatment pathway is being followed.

Practical tip

Drug safety alert

Systemic fluoroquinolone antibiotics may cause **serious, disabling**, and potentially **long-lasting or irreversible adverse events**. This includes, but is not limited to: tendinopathy/tendon rupture; peripheral neuropathy; arthropathy/arthralgia; aortic aneurysm and dissection; heart valve regurgitation; dysglycaemia; and central nervous system effects including seizures, depression, psychosis, and suicidal thoughts and behaviour.[58]

- **Prescribing restrictions apply** to the use of fluoroquinolones, and these restrictions may vary between countries. In general, fluoroquinolones should be **restricted** for use in serious, life-threatening bacterial infections only. Some regulatory agencies may also recommend that they must only be used in situations where other antibiotics, that are commonly recommended for the infection, are inappropriate (e.g., resistance, contraindications, treatment failure, unavailability).
- Consult your local guidelines and drug formulary for more information on suitability, contraindications, and precautions.

Endovascular treatment

With the emergence of interventional radiology, consider endovascular treatment for haemodynamically stable patients where available. Options include thrombolysis, transjugular intrahepatic portosystemic shunt, and thrombectomy.[8] [9][20] Endovascular therapy for acute mesenteric ischaemia has been shown to be associated with a reduced mortality and reduced risk of small bowel resection, though duration of patency may be shorter.[59] [60] [61] [62] [63] However, endovascular treatment is unlikely to be appropriate in a patient determined to have evidence of peritonitis.

Peritonitis, infarction, or perforation

If there are clinical signs of peritonitis, or radiographic or laboratory evidence suggesting infarction or perforation, proceed urgently with exploratory laparotomy and include resection of non-viable intestine.[8]

[38] Ideally, revascularisation procedures should be completed prior to any bowel resection, as borderline ischaemic bowel may recover satisfactorily after revascularisation. Second-look operations may be necessary to evaluate progression of ischaemia or reperfusion injury resulting in non-viable intestine requiring resection. Anastomosis can at times be delayed until the second-look laparotomy, especially if the patient is clinically unstable.

Depending on the underlying pathology and findings at surgery, several interventions may be appropriate.

- Consider revascularisation with embolectomy or thrombectomy at the level of arterial occlusion for proximal embolisation or thrombosis.
- Consider systemic-mesenteric bypass if arterial occlusion is due to severe and widespread atherosclerotic disease and the patient is stable enough to tolerate increased operative duration.
- If mesenteric venous thrombosis is identified at the time of exploratory laparotomy, anticoagulation with intravenous heparin should be started (and continued until bowel function has normalised).[64] Venous thrombectomy may also be appropriate.[64]
- If vasculitis is identified as a contributory cause (e.g., by thickened blood vessels on computed tomography scan, or the presence of other vasculitic symptoms, or a previous diagnosis) postoperative corticosteroid therapy may be considered.
- If a source of sepsis is identified, use appropriate swabs and cultures to identify causative organisms and allow subsequent targeting of antibiotic therapy. See the *More information* box in *Resuscitation and supportive measures*; see also Sepsis in adults .

Post-operative anticoagulation is generally recognised as being beneficial, although timing of treatment is controversial. Some authorities recommend a delay of 48 hours following surgery because of the risk of intraluminal bleeding from damaged bowel, while others advocate immediate anticoagulation. Another suggested approach has been immediate anticoagulation if no infarction is present at surgery, and delayed anticoagulation if intestinal infarction is found. Good data on these approaches are lacking. [32]

Fulminant ischaemic colitis

Patients with fulminant ischaemic colitis usually appear acutely unwell and are unresponsive to medical therapy.

Ischaemia and necrosis of the right-side colon may be treated by right hemicolectomy with primary anastomosis, providing the remaining ileal and colonic ends are well perfused. If there is perforation and peritonitis, resection with terminal ileostomy and a colonic mucocutaneous fistula is indicated. Damage control surgery is another option; this involves resecting the ischaemic bowel and planning a second look with possible anastomosis 24 to 48 hours later.

Left-sided colonic involvement may require a proximal end stoma and distal mucous fistula or Hartmann's procedure. If most of the colon is ischaemic, subtotal colectomy with terminal ileostomy is indicated.

Depending on the findings of the initial surgery, a second-look operation within around 24 hours to reassess bowel viability may be indicated.

Superior mesenteric artery embolus and thrombosis

Superior mesenteric artery (SMA) embolus causes a critical reduction in flow of both the obstructed and unobstructed branches of the SMA. If not corrected promptly this will progress to infarction.

- If available and the clinical condition of the patient permits, consider endovascular treatment as a first-line option.
- Refer to the vascular team and a vascular radiologist.
- Consider local thrombolytic therapy if there is no evidence of infarction, perforation, or peritonitis requiring urgent surgical intervention (and there are no contraindications). If lysis of the embolus cannot be demonstrated within 4 hours, or there is evidence of ischaemia progression, patients should undergo exploratory laparotomy with a view to conventional surgical embolectomy.
- The open alternative is SMA embolectomy or arterial bypass. If not amenable to embolectomy, arterial bypass may be required. Any infarcted bowel should be resected.
- Patients will generally be maintained on an intravenous heparin infusion once a diagnosis of SMA embolus is established. A heparin infusion titrated to therapeutic dosing following partial thromboplastin time (PTT) prolongation to 1.5 to 2.5 times normal PTT levels is recommended.

Acute SMA thrombosis is suggested by the absence of collaterals on angiography; this necessitates immediate intervention.

- Surgical procedures that may be used in these circumstances include antegrade and retrograde bypass grafting, aortic re-implantation of the SMA, and transarterial and transaortic mesenteric endarterectomy.[31] [65]
- Endovascular treatment is unlikely to be appropriate in a patient determined to have evidence of peritonitis.
- Patients with SMA thrombosis and no evidence of infarction, perforation, or peritonitis will generally be maintained on an intravenous heparin infusion once a diagnosis of SMA thrombosis is established. A heparin infusion titrated to therapeutic dosing following partial thromboplastin time (PTT) prolongation to 1.5 to 2.5 times normal PTT levels is recommended.

The presence of collaterals on angiography suggests a chronic SMA thrombosis. In this case endovascular treatment is the first-line therapy.

- Options include angioplasty ± stenting, aspiration thrombectomy, or local drug instillation. If patients are unsuitable for endovascular intervention, surgery may be an option if the patient is fit enough. Refer to the vascular team and a vascular radiologist.
- Surgical procedures that may be used in these circumstances include antegrade and retrograde bypass grafting, aortic reimplantation of the SMA, and transarterial and transaortic mesenteric endarterectomy.[31]
- Timing of postoperative anticoagulation is controversial, although it is generally recognised as being beneficial.

For patients with SMA embolus or thrombosis and peritonitis, infarction, or perforation, see *Peritonitis, infarction, or perforation* above in *Acute mesenteric ischaemia*.

Non-occlusive mesenteric ischaemia

Non-occlusive mesenteric ischaemia (NOMI) is caused by splanchnic hypoperfusion, which may be precipitated by congestive heart failure, cardiac arrhythmia, or shock, or by large volume shifts, which can sometimes occur during haemodialysis.

Urgent correction of any underlying medical cause of hypoperfusion (e.g., heart failure) is of paramount importance to restore perfusion. After correction of the underlying medical condition, endovascular

therapy is the first-line option, with selective mesenteric angiography and local intra-arterial infusion of vasodilators.[66] The infusion should be continued until there is no angiographic or clinical evidence of persistent vasoconstriction.

Laparotomy is indicated if there is evidence of peritonitis, perforation, or if the patient clinically declines.[20] Surgery should preserve bowel of questionable viability, unless necrosis is clear. With use of temporary abdominal closure and re-exploration at 24 to 48 hours after the first operation, intestinal resection can be kept to a minimum.

Mesenteric vein thrombosis

Anticoagulation is the first-line treatment option for venous mesenteric ischaemia when the clinical condition permits. This may be successful in up to 95% of cases. These patients need close clinical observation, and surgery is indicated if signs of peritonitis develop.

Therapeutic anticoagulation with intravenous heparin should continue until bowel function normalises.[64] In patients who receive heparin, the recurrence rate is lowered from 25% to 13% and mortality is reduced from 50% to 13%.[32] Once patients are stable, symptom-free, and able to tolerate oral medication, they can be converted to warfarin, which should then be administered for 3 to 6 months.

Surgery is indicated in patients with venous ischaemia when there are signs of infarction or peritonitis. Infarcted bowel should be resected.[64] A second-look procedure is a valuable tool to assess the recovery of any questionable segments of bowel that can be left in situ at the index surgery if potentially viable.[64]

If a mesenteric vein thrombus is discovered incidentally in an asymptomatic patient who undergoes a computed tomography scan for another reason besides abdominal pain, a 3- to 6-month course of warfarin is recommended, especially if a predisposing hypercoagulable state or concomitant deep vein thrombosis can be identified.[67]

Chronic mesenteric ischaemia

The treatment of chronic mesenteric ischaemia will depend on several factors, most notably whether or not the patient is a surgical candidate.

Consider endovascular treatment, particularly in patients with severe malnutrition, as it is associated with less morbidity and mortality than open therapy.[68] [69]

Practical tip

Malnutrition

The National Institute for Health and Care Excellence in the UK defines malnourishment as:[70]

- A body mass index (BMI) of less than 18.5 kg/m^2
- Unintentional weight loss greater than 10% within the last 3 to 6 months
- A BMI of less than 20 kg/m^2 and unintentional weight loss greater than 5% within the last 3 to 6 months.

If the patient is assessed as suitable for open surgery after medical optimisation of any cardiovascular, respiratory, or other comorbidities, then surgical systemic-mesenteric bypass forms the mainstay of treatment, although other procedures may be used. Antegrade and retrograde bypass grafting,

aortic re-implantation of the superior mesenteric artery, and transarterial and transaortic mesenteric endarterectomy all have a role.[31] Open surgery is better for long-term patency when compared with endovascular approaches for chronic mesenteric ischaemia.[68] [71]

In patients who are not fit to undergo an open procedure, percutaneous transluminal mesenteric angioplasty alone or with stent insertion may be an option.[72]

Colonic ischaemia

This is the most common form of intestinal ischaemia and comprises a spectrum of disorders including:[73]

- Reversible colonopathy
- Transient colonic ischaemia
- Chronic colonic ischaemia
- Stricture
- Gangrene
- Fulminant pancolitis.

Most patients with colonic ischaemia do not have any identifiable, specific, and precipitating cause, and treatment varies with severity of presentation.[4] Most cases resolve spontaneously (reversible ischaemic colonopathy or transient colonic ischaemic changes). Patients with severe or continuing symptoms might need admission to hospital, supportive measures, bowel rest, and investigation and treatment of any underlying cause.

There are several indications for surgery in colonic ischaemia.[4]

Acute indications:

- Peritoneal signs, suggestive of necrosis or perforation
- Massive bleeding (rare): may require subtotal colectomy
- Universal fulminant colitis with or without toxic megacolon.

Subacute indications:

- Failure of an acute segmental ischaemic colitis to respond within 2 to 3 weeks, with continued symptoms or a protein-losing colonopathy
- Apparent healing but recurrent bouts of sepsis.

Chronic indications:

- Symptomatic colon stricture: may be treated with endoscopic balloon dilation or segmental resection
- Symptomatic segmental ischaemic colitis.

Ischaemic colitis

Patients with colonic ischaemia can develop chronic ulcerating ischaemic colitis.[37] Seek gastroenterological and surgical input for ischaemic colitis.[36]

In mild cases, trial conservative management, including intravenous antibiotics, fluids (and blood glucose control in patients with diabetes), and bowel rest.

Consider nutrition support in patients at risk of malnutrition who:[70]

- Have eaten little or nothing for more than 5 days and/or are likely to eat little or nothing for the next 5 days or longer
- Have a poor absorptive capacity, and/or have high nutrient losses and/or have increased nutritional needs from causes such as catabolism.

Frequently review the patient, examining the abdomen and monitoring vital signs. Further investigations and imaging are required if symptoms do not resolve, symptoms worsen, or new symptoms appear.[36]

Prophylactic low molecular weight heparin is generally used. Secondary prevention with anticoagulation should be considered at the point of discharge.[36]

Surgical intervention for ischaemic colitis usually involves segmental resection and stoma formation.[36]

Non-acute colonic ischaemia

Patients who have an acute episode of colonic ischaemia that evolves into a segmental colitis pattern with symptoms persisting for >2 weeks, or who develop a protein-losing colonopathy, are usually best treated by segmental colectomy.[4]

Episodes of recurrent sepsis in a patient who has symptomatically recovered from an acute episode of colonic ischaemia may be an indication for surgery. These patients usually have a short segment of unhealed bowel that is the source of sepsis, and resection of the segment is usually curative.[4]

See Sepsis in adults .

Interventions such as endoscopic dilation of stricture or segmental resection should only be used if strictures are symptomatic. Transendoscopic dilation may be successful in less severe cases. Alternatively, segmental resection can be used.[4]

Transient or mild ischaemia with no evidence of infarction, perforation, or peritonitis

Patients with acute transient or mild ischaemia have physical findings with no evidence of peritonitis, intestinal perfusion, or full-thickness necrosis on a computed tomography scan or mesenteric angiography.

Use conservative measures if imaging suggests mucosal or submucosal involvement only. These include:

- Nil by mouth status
- Fluid resuscitation
- Nasogastric tube decompression for symptomatic relief.

The underlying cause should be treated promptly. This may include:

- Anticoagulation for mesenteric venous thrombosis[64]
- Tailored antibiotic therapy when an infectious cause is identified
- Corticosteroids for vasculitis

- Fluid resuscitation and cardiac optimisation for shock (see Shock).

Frequently reassess the patient to detect patients in whom conservative management fails and who then require operative intervention due to evidence of peritonitis or infarction.

Procedural videos

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute (summary)

evidence of infarction, perforation, or peritonitis on diagnostic computed tomography scan		
	1st	resuscitation and supportive measures
	plus	empirical antibiotics
■ superior mesenteric artery (SMA) embolus	plus	open embolectomy or arterial bypass ± bowel resection
	plus	postoperative anticoagulation
■ acute superior mesenteric artery (SMA) thrombosis	plus	arterial reconstruction or bypass ± bowel resection
	plus	postoperative anticoagulation
■ non-occlusive mesenteric ischaemia	plus	correct underlying medical cause ± bowel resection
	plus	postoperative anticoagulation
■ mesenteric vein thrombosis	plus	anticoagulation
	plus	open surgery ± thrombectomy ± bowel resection
■ fulminant ischaemic colitis	plus	subtotal or total colectomy
no evidence of infarction, perforation, or peritonitis on diagnostic computed tomography scan		
	1st	supportive measures and treatment of the underlying cause
■ superior mesenteric artery (SMA) embolus	plus	endovascular therapy ± open embolectomy or arterial bypass ± bowel resection
	plus	anticoagulation
■ superior mesenteric artery (SMA) thrombosis	plus	anticoagulation
	plus	endovascular therapy ± arterial reconstruction or bypass ± bowel resection
■ non-occlusive mesenteric ischaemia	plus	endovascular therapy + observation

Acute (summary)		
mesenteric vein thrombosis	plus	anticoagulation + observation
vasculitis-associated mesenteric ischaemia	plus	corticosteroid therapy

Ongoing (summary)		
chronic mesenteric ischaemia		
surgical candidate	1st	medical optimisation + surgical systemic-mesenteric bypass
non-surgical candidate	1st	medical optimisation + percutaneous angioplasty and stenting
ischaemic colitis		
	1st	conservative management
	consider	anticoagulation
	consider	segmental resection and stoma
non-acute colonic ischaemia		
segmental colitis symptomatic for >2 weeks, or protein-losing colonopathy	1st	segmental colectomy
recurrent sepsis in a patient who has symptomatically recovered from an acute episode	1st	segmental colectomy
chronic symptomatic ischaemic stricture as a result of healing after ischaemic event	1st	endoscopic dilation of stricture or segmental resection

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute

evidence of infarction, perforation, or peritonitis on diagnostic computed tomography scan

1st **resuscitation and supportive measures**

» Acute mesenteric ischaemia is a **medical emergency**. Liaise early with surgery and intensive care unit colleagues.

» Administer adequate fluid resuscitation and supplemental oxygen to optimise tissue perfusion and oxygenation.[20]

» Check your local protocols for specific recommendations on fluid choice. There is debate, based on conflicting evidence, on whether there is a benefit in using normal saline or balanced crystalloid in critically ill patients.

» Monitor controlled oxygen therapy. An upper SpO_2 limit of 96% is reasonable when administering supplemental oxygen to most patients with acute illness who are not at risk of hypercapnia.

- Evidence suggests that liberal use of supplemental oxygen (target SpO_2 >96%) in acutely ill adults is associated with higher mortality than more conservative oxygen therapy.[48]
- A lower target SpO_2 of 88% to 92% is appropriate if the patient is at risk of hypercapnic respiratory failure.[49]

» Initial resuscitation should also aim to address any acute heart failure and correct any cardiac arrhythmias.

» Involve the critical care team for further management as necessary.

» Consider invasive monitoring as appropriate. Nil by mouth status should be enforced, with nasogastric tube decompression for symptomatic relief.[8]

More info: Sepsis

Think '***Could this be sepsis?***' based on acute deterioration in an adult patient in

Acute

whom there is clinical evidence or strong suspicion of infection.[50] [51] [52] See Sepsis in adults .

- The patient may present with non-specific or non-localised symptoms (e.g., acutely unwell with a normal temperature) or there may be severe signs with evidence of multi-organ dysfunction and shock.[50] [51] [52]
- Remember that sepsis represents the severe, life-threatening end of infection.[53]
- Ischaemic bowel disease and bowel perforation can cause rapid deterioration into septic shock. For this reason, the assessment of severity status and subsequent action should be upgraded according to patient need, and at least to the next NEWS band. There should be no avoidable delay in giving antimicrobials and achieving source control.[54] In practice, if computed tomography (CT) shows extensive ischaemia in a patient who is a very frail or has significant comorbidities, palliative care may be the treatment of choice (rather than antibiotics and source control) this decision should always be made in discussion with a consultant.

Use a systematic approach (e.g., National Early Warning Score 2 [NEWS2]), alongside your clinical judgement, to assess the risk of deterioration due to sepsis.[50] [51] [55] [56] [52] Consult local guidelines for the recommended approach at your institution.

Arrange urgent review by a senior clinical decision-maker (e.g., ST3 level doctor in the UK) if you suspect sepsis:[54]

- **Within 30 minutes** for a patient who is critically ill (e.g., NEWS2 score of 7 or more, evidence of septic shock, or other significant clinical concerns).
    - A patient is also at high risk of severe illness or death from sepsis if they have a NEWS2 score below 7 and a single parameter contributes 3 points to their NEWS2 score and a

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medical review has confirmed that they are at high risk.

- **Within 1 hour** for a patient who is severely ill (e.g., NEWS2 score of 5 or 6) or **within 1 hour** of any intervention for suspected sepsis (antibiotics/fluid resuscitation/oxygen) if there is no improvement in the patient's condition.

Follow your local protocol for investigation and treatment of all patients with suspected sepsis, or those at risk. Start treatment promptly. Determine urgency of treatment according to likelihood of infection and severity of illness, or according to your local protocol.[54] [56]

In the community and custodial settings: refer for emergency medical care in hospital (usually by blue-light ambulance in the UK) any patient who is acutely ill with a suspected infection and is:[52]

- Deemed to be at high risk of deterioration due to organ dysfunction (as measured by risk stratification)
- At risk of neutropenic sepsis.

» If there are clinical signs of peritonitis, or radiographic or laboratory evidence suggesting infarction or perforation, proceed urgently with exploratory laparotomy and include resection of non-viable intestine. Ideally, revascularisation procedures should be completed prior to any bowel resection, as borderline ischaemic bowel may recover satisfactorily after revascularisation.

» Patients with delayed presentation and abdominal signs of peritonitis or organ failure generally have a worse prognosis than patients without these clinical factors.[20] It may be appropriate to discuss palliative care options with the multidisciplinary team if the patient is unlikely to benefit from invasive procedures.[20]

plus **empirical antibiotics**

Treatment recommended for ALL patients in selected patient group

Primary options

» ceftriaxone: 1-2 g intravenously every 24 hours
-and-

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» metronidazole: 500 mg intravenously every 8 hours

Secondary options

» ciprofloxacin: 400 mg intravenously every 8-12 hours
-and-
» metronidazole: 500 mg intravenously every 8 hours

» Consult your local antimicrobial guidelines to administer empirical antibiotics suitable for enteric coverage (e.g., a third-generation cephalosporin plus metronidazole in critically ill patients, or a fluoroquinolone-based regimen in patients with beta-lactam allergy such as ciprofloxacin plus metronidazole) to all patients who are being treated with curative intent. Ischaemia can lead to significant bacterial translocation due to damage to the normal intestinal mucosal barrier.[8] [57] Antibiotics may not be indicated if a palliative treatment pathway is being followed.

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Practical tip

Drug safety alert
Systemic fluoroquinolone antibiotics may cause **serious, disabling** and potentially **long-lasting or irreversible adverse events**. This includes, but is not limited to: tendinopathy/tendon rupture; peripheral neuropathy; arthropathy/arthralgia; aortic aneurysm and dissection; heart valve regurgitation; dysglycaemia; and central nervous system effects including seizures, depression, psychosis, and suicidal thoughts and behaviour.[58]

- **Prescribing restrictions apply** to the use of fluoroquinolones, and these restrictions may vary between countries. In general, fluoroquinolones should be **restricted** for use in serious, life-threatening bacterial infections only. Some regulatory agencies may also recommend that they must only be used in situations where other antibiotics, that are commonly recommended for the infection, are inappropriate (e.g., resistance, contraindications, treatment failure, unavailability).
- Consult your local guidelines and drug formulary for more information on suitability, contraindications, and precautions.

superior mesenteric artery (SMA) embolus

plus **open embolectomy or arterial bypass ± bowel resection**

Treatment recommended for ALL patients in selected patient group

» If there is evidence of ischaemia progression, patients should undergo urgent exploratory laparotomy with a view to conventional surgical embolectomy.

» If not amenable to embolectomy, arterial bypass may be required.

plus **postoperative anticoagulation**

Treatment recommended for ALL patients in selected patient group

Primary options

» heparin: consult specialist for guidance on dose

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» Timing of postoperative anticoagulation is controversial, although it is generally recognised as being beneficial.

» Some authorities recommend a delay of 48 hours because of the risk of intraluminal bleeding from damaged bowel, while others advocate immediate anticoagulation. Another suggested approach has been immediate anticoagulation if no infarction is present, and delayed anticoagulation if intestinal infarction is present. Good data on these approaches are lacking.[32]

- **acute superior mesenteric artery (SMA) thrombosis**

plus **arterial reconstruction or bypass ± bowel resection**

Treatment recommended for ALL patients in selected patient group

» The absence of collaterals on angiography suggests an acute SMA thrombosis has occurred and necessitates immediate intervention.

» Surgical procedures that may be used in these circumstances include antegrade and retrograde bypass grafting, aortic re-implantation of the SMA, and transarterial and transaortic mesenteric endarterectomy.[31]

» Endovascular treatment is unlikely to be appropriate in a patient determined to have evidence of peritonitis.

plus **postoperative anticoagulation**

Treatment recommended for ALL patients in selected patient group

Primary options

» heparin: consult specialist for guidance on dose

» Timing of postoperative anticoagulation is controversial, although it is generally recognised as being beneficial.

» Some authorities recommend a delay of 48 hours because of the risk of intraluminal bleeding from damaged bowel, while others advocate immediate anticoagulation. Another suggested approach has been immediate anticoagulation if no infarction is present, and delayed anticoagulation if intestinal infarction is present. Good data on these approaches are lacking.[32]

- **non-occlusive mesenteric ischaemia**

plus **correct underlying medical cause ± bowel resection**

Acute

Treatment recommended for ALL patients in selected patient group

» Splanchnic hypoperfusion may be precipitated by congestive heart failure, cardiac arrhythmia, or shock, or by large volume shifts, which can sometimes occur during haemodialysis.

» Urgent correction of any underlying medical cause of hypoperfusion (e.g., heart failure) is of paramount importance to restore perfusion.

» Laparotomy is indicated if there is evidence of peritonitis, perforation, or if the patient clinically declines.[20] Surgery should preserve bowel of questionable viability, unless necrosis is clear. With use of temporary abdominal closure and re-exploration at 24 to 48 hours after the first operation, intestinal resection can be kept to a minimum.

plus **postoperative anticoagulation**

Treatment recommended for ALL patients in selected patient group

Primary options

» heparin: consult specialist for guidance on dose

» Timing of postoperative anticoagulation is controversial, although it is generally recognised as being beneficial. Some authorities recommend a delay of 48 hours because of the risk of intraluminal bleeding from damaged bowel, while others advocate immediate anticoagulation. Another suggested approach has been immediate anticoagulation if no infarction is present, and delayed anticoagulation if intestinal infarction is present. Good data on these approaches are lacking.[32]

mesenteric vein thrombosis

plus **anticoagulation**

Treatment recommended for ALL patients in selected patient group

Primary options

» heparin: consult specialist for guidance on dose

OR

» warfarin: consult specialist for guidance on dose

» Anticoagulation is the first-line treatment option for venous mesenteric ischaemia when the clinical condition permits. This may be

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successful in up to 95% of cases. These patients need close clinical observation, and surgery is indicated if signs of peritonitis develop. Therapeutic anticoagulation with intravenous heparin should continue until bowel function normalises.[64] In patients who receive heparin, the recurrence rate is lowered from 25% to 13% and mortality is reduced from 50% to 13%.[32] Once patients are stable, and able to tolerate oral medication, they can be converted to warfarin, which should then be administered for 3 to 6 months.

plus **open surgery ± thrombectomy ± bowel resection**

Treatment recommended for ALL patients in selected patient group

» Surgery is indicated in patients with venous ischaemia when there are signs of infarction or peritonitis. Infarcted bowel should be resected.[64] A second-look procedure is a valuable tool to assess the recovery of any questionable segments of bowel that can be left in situ at the index surgery if potentially viable.[64]

fulminant ischaemic colitis

plus **subtotal or total colectomy**

Treatment recommended for ALL patients in selected patient group

» These patients usually appear acutely unwell and are unresponsive to medical therapy.

» Ischaemia and necrosis of the right-side colon may be treated by right hemicolectomy with primary anastomosis, providing the remaining ileal and colonic ends are well perfused. If there is perforation and peritonitis, resection with terminal ileostomy and a colonic mucocutaneous fistula is indicated. Damage control surgery is another option; this involves resecting the ischaemic bowel and planning a second look with possible anastomosis 24 to 48 hours later.

» Left-sided colonic involvement may require a proximal end stoma and distal mucous fistula or Hartmann's procedure.

» If most of the colon is ischaemic, subtotal colectomy with terminal ileostomy is indicated.

» Depending on the findings of the initial surgery, a second-look operation within around 24 hours to reassess bowel viability may be indicated.

no evidence of infarction, perforation, or peritonitis on

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diagnostic computed tomography scan

no evidence of infarction, perforation, or peritonitis on diagnostic computed tomography scan

1st **supportive measures and treatment of the underlying cause**

» Patients with acute transient or mild ischaemia have physical findings with no evidence of peritonitis, intestinal perfusion, or full-thickness necrosis on a computed tomography scan or mesenteric angiography.

» Use conservative measures if imaging suggests mucosal or submucosal involvement only. These include nil by mouth status; resuscitation with intravenous fluids; and nasogastric tube decompression for symptomatic relief. Also consider supplemental oxygen as needed; and correction of hypotension, heart failure, and arrhythmias.

» The underlying cause should be treated promptly. This may include anticoagulation for mesenteric venous thrombosis, tailored antibiotic therapy when an infectious cause is identified, corticosteroids for vasculitis, and fluid resuscitation and cardiac optimisation for shock (see Shock).[64]

» Frequently reassess vital signs, and repeat the physical examination and laboratory values to detect failure of non-surgical management that may then require operative intervention. These patients require close observation, and surgery is indicated should signs of peritonitis develop (e.g., rigid, distended abdomen; guarding and rebound; loss of bowel sounds).

superior mesenteric artery (SMA) embolus

plus **endovascular therapy ± open embolectomy or arterial bypass ± bowel resection**

Treatment recommended for ALL patients in selected patient group

» An embolus in the superior SMA causes a critical reduction in flow of both the obstructed and unobstructed branches of the SMA. If not corrected promptly this will progress to infarction.

» If available and the clinical condition of the patient permits, consider endovascular treatment as a first-line option. Refer to the vascular team and a vascular radiologist.

» Consider local thrombolytic therapy if there is no evidence of infarction, perforation, or peritonitis requiring urgent surgical intervention (and there are no contraindications). If lysis of the embolus cannot be demonstrated within

Acute

4 hours, or there is evidence of ischaemia progression, patients should undergo exploratory laparotomy with a view to conventional surgical embolectomy.

» The open alternative is SMA embolectomy or arterial bypass. If not amenable to embolectomy, arterial bypass may be required. Any infarcted bowel should be resected.

plus **anticoagulation**

Treatment recommended for ALL patients in selected patient group

Primary options

» heparin: consult specialist for guidance on dose

» Patients will generally be maintained on an intravenous heparin infusion once a diagnosis of SMA embolus is established.

» A heparin infusion titrated to therapeutic dosing following partial thromboplastin time (PTT) prolongation to 1.5 to 2.5 times normal PTT levels is recommended.

superior mesenteric artery (SMA) thrombosis

plus **anticoagulation**

Treatment recommended for ALL patients in selected patient group

Primary options

» heparin: consult specialist for guidance on dose

» Patients will generally be maintained on an intravenous heparin infusion once a diagnosis of SMA thrombosis is established.

» A heparin infusion titrated to therapeutic dosing following partial thromboplastin time (PTT) prolongation to 1.5 to 2.5 times normal PTT levels is recommended.

plus **endovascular therapy ± arterial reconstruction or bypass ± bowel resection**

Treatment recommended for ALL patients in selected patient group

» The presence of collaterals on angiography suggests a chronic thrombosis.

» Endovascular treatment is the first-line therapy for chronic thrombotic mesenteric ischaemia. Options include angioplasty ± stenting, aspiration thrombectomy, or local

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drug instillation. If patients are unsuitable for endovascular intervention, surgery may be an option if the patient is fit enough. Refer to the vascular team and a vascular radiologist.

» Surgical procedures that may be used in these circumstances include antegrade and retrograde bypass grafting, aortic reimplantation of the SMA, and transarterial and transaortic mesenteric endarterectomy.[31]

non-occlusive mesenteric ischaemia **plus** **endovascular therapy + observation**

Treatment recommended for ALL patients in selected patient group

» Splanchnic hypoperfusion may be precipitated by congestive heart failure, cardiac arrhythmia, or shock, or by large volume shifts, which can sometimes occur during haemodialysis.

» Urgent correction of any underlying medical cause of hypoperfusion (e.g., heart failure) is of paramount importance to restore perfusion.

» After correction of the underlying medical condition, endovascular therapy is the first-line option, with selective mesenteric angiography and local intra-arterial infusion of vasodilators.[66]

» The infusion should be continued until there is no angiographic or clinical evidence of persistent vasoconstriction.

mesenteric vein thrombosis **plus** **anticoagulation + observation**

Treatment recommended for ALL patients in selected patient group

Primary options

» heparin: consult specialist for guidance on dose

OR

» warfarin: consult specialist for guidance on dose

» Anticoagulation is the first-line treatment option for venous mesenteric ischaemia when the clinical condition permits. This may be successful in up to 95% of cases. These patients need close clinical observation, and surgery is indicated if signs of peritonitis develop.

» Therapeutic anticoagulation with intravenous heparin should continue until bowel function normalises.[64]

Acute

» In patients who receive heparin, the recurrence rate is lowered from 25% to 13% and mortality is reduced from 50% to 13%.[32] Once patients are stable, symptom-free, and able to tolerate oral medication, they can be converted to warfarin, which should then be administered for 3 to 6 months.

» If a mesenteric vein thrombus is discovered incidentally in an asymptomatic patient who undergoes a computed tomography scan for another reason besides abdominal pain, a 3- to 6-month course of warfarin is recommended, especially if a predisposing hypercoagulable state or concomitant deep vein thrombosis can be identified.[67]

vasculitis-associated mesenteric ischaemia **plus** **corticosteroid therapy**

Treatment recommended for ALL patients in selected patient group

Primary options

» methylprednisolone: consult specialist for guidance on dose

» If vasculitis is identified as a contributory cause of the ischaemia (e.g., by thickened blood vessels on computed tomography scan, the presence of other vasculitic symptoms and serological markers, or a previous diagnosis), postoperative corticosteroid therapy may be considered.

Ongoing

chronic mesenteric ischaemia

surgical candidate

1st **medical optimisation + surgical systemic-mesenteric bypass**

» The treatment of chronic mesenteric ischaemia will depend on several factors, most notably whether or not the patient is a surgical candidate. Consider endovascular treatment, particularly in patients with severe malnutrition, as it is associated with less morbidity and mortality than open therapy.[68] [69]

» The National Institute for Health and Care Excellence in the UK defines malnutrition as:[70]

- A body mass index (BMI) of less than 18.5 kg/m^2
- Unintentional weight loss greater than 10% within the last 3 to 6 months
- A BMI of less than 20 kg/m^2 and unintentional weight loss greater than 5% within the last 3 to 6 months.

» If the patient is assessed as suitable for open surgery after medical optimisation of any cardiovascular, respiratory, or other comorbidities, then surgical systemic-mesenteric bypass forms the mainstay of treatment.

» Antegrade and retrograde bypass grafting, aortic re-implantation of the superior mesenteric artery, and transarterial and transaortic mesenteric endarterectomy may all have a role.[31] Open surgery is better for long-term patency when compared with endovascular approaches for chronic mesenteric ischaemia.[68] [71]

non-surgical candidate

1st **medical optimisation + percutaneous angioplasty and stenting**

» If the patient is assessed as unsuitable for open surgery despite medical optimisation of any cardiovascular, respiratory, or other comorbidities, then percutaneous transluminal mesenteric angioplasty alone or with stent insertion may be an option.[72]

ischaemic colitis

1st **conservative management**

» Patients with colonic ischaemia can develop chronic ulcerating ischaemic colitis.[37] Seek

Ongoing

gastroenterological and surgical input for ischaemic colitis.[36]

» In mild cases, trial conservative management, including intravenous antibiotics, fluids (and blood glucose control in patients with diabetes), and bowel rest.

» Consider nutrition support in patients at risk of malnutrition who:[70]

- Have eaten little or nothing for more than 5 days and/or are likely to eat little or nothing for the next 5 days or longer
- Have a poor absorptive capacity, and/or have high nutrient losses and/or have increased nutritional needs from causes such as catabolism.

» Frequently review the patient, examining the abdomen and monitoring vital signs. Further investigations and imaging are required if symptoms do not resolve, symptoms worsen, or new symptoms appear.[36]

consider **anticoagulation**

Treatment recommended for SOME patients in selected patient group

Primary options

» heparin: consult specialist for guidance on dose

OR

» warfarin: consult specialist for guidance on dose

» Prophylactic low molecular weight heparin is generally used. Secondary prevention with anticoagulation should be considered at the point of discharge.[36]

consider **segmental resection and stoma**

Treatment recommended for SOME patients in selected patient group

» Surgical intervention for ischaemic colitis usually involves segmental resection and stoma formation.[36]

non-acute colonic ischaemia

segmental colitis symptomatic for >2 **1st** **segmental colectomy**

MANAGEMENT

Ongoing

weeks, or protein-losing colonopathy		» Patients who have an acute episode of colonic ischaemia that evolves into a segmental colitis pattern with symptoms persisting for >2 weeks, or who develop a protein-losing colonopathy, are usually best treated by segmental colectomy.[4]
recurrent sepsis in a patient who has symptomatically recovered from an acute episode	**1st**	**segmental colectomy** » Episodes of recurrent sepsis in a patient who has symptomatically recovered from an acute episode of colonic ischaemia may be an indication for surgery. These patients usually have a short segment of unhealed bowel that is the source of sepsis, and resection of the segment is usually curative.[4] » See Sepsis in adults .
chronic symptomatic ischaemic stricture as a result of healing after ischaemic event	**1st**	**endoscopic dilation of stricture or segmental resection** » These interventions should only be used if strictures are symptomatic. Transendoscopic dilation may be successful in less severe cases. Alternatively, segmental resection can be used.[4]

Emerging

Splanchnic near-infrared spectroscopy (NIRS)

One meta-analysis of studies in newborns, which assessed the use of abdominal NIRS as a marker of bowel ischaemia in newborns, showed that NIRS accurately detects local tissue oxygenation mismatch and can be used to monitor gastrointestinal oxygenation and detect bowel ischaemia in this patient group.[89] Low gastrointestinal regional blood flow was consistently associated with bowel ischaemia.[89] Further investigation is needed to confirm the utility of NIRS as a biomarker for bowel ischaemia in adults.

Primary prevention

Given the contribution of cardiovascular disease to many cases of ischaemic bowel, it is reasonable to suggest that careful long-term lifestyle and medical management of cardiovascular risk factors may reduce the risk of developing ischaemic bowel disease. However, robust supporting data are lacking.

Secondary prevention

Patients surviving acute mesenteric ischaemia should be counselled about smoking cessation and commenced on statin and antiplatelet (after endovascular intervention) or anticoagulant (in non-valvular atrial fibrillation) therapy.[9] [38]

Patient discussions

Emphasise the importance of regular medical follow-up and adherence to medication, to ensure that the underlying aetiology of the ischaemic bowel disease is managed in the optimal manner to help prevent future ischaemic episodes.

The patient should seek urgent medical attention if symptoms such as abdominal pain, persistent nausea or vomiting, bloating, or bloody stools occur.

Monitoring

Monitoring

Patients with transient colonic ischaemia who have not undergone surgery should have endoscopy performed following resolution of the acute ischaemic event, in order to identify strictures and/or confirm resolution of the colonic ischaemia.

All patients who have undergone any form of surgery require regular follow-up to ensure satisfactory healing and recovery from surgery.

In order to minimise the chance of recurrence of bowel ischaemia, patients should have regular medical check-ups to optimise and ensure adherence to medical treatment of any comorbidities that may contribute to the development of ischaemic bowel disease.

Complications

Complications	Timeframe	Likelihood
sitophobia (food fear)	**variable**	**high**
Patients with chronic mesenteric ischaemia frequently develop a fear of eating, as consumption of food often acts as the trigger for increased physiological demand for blood in the bowel, bringing with it symptoms of ischaemia. Significant weight loss and malnutrition can occur as a result.		
stricture	**variable**	**medium**
Patients whose episodes of ischaemic bowel disease resolve with medical management carry a significant risk for developing strictures. Surgical strictureplasty or more commonly bowel resection is the mainstay of treatment, as strictures are normally of a length that precludes endoscopic balloon dilatation.		
short bowel syndrome	**variable**	**medium**
Develops in patients who have undergone significant bowel resection. Management requires permanent parenteral nutrition or small bowel transplantation.		

Prognosis

Acute mesenteric ischaemia

Despite advances in knowledge in diagnosis, pathophysiology and treatment, the outlook for patients with acute mesenteric ischaemia remains poor.[29] [38] Acute mesenteric ischaemia results in mortality rates of approximately 50% to 60% according to several large series.[8] [29] [90] [91] Pooled mortality estimates for different treatment modalities confirm this high mortality rate, with a meta-analysis of observational studies and case series from 2000 to 2001 showing a relatively static mortality rate of 40% for open surgery, 26% for endovascular surgery, and 32% for open retrograde mesenteric stenting.[92]

Mortality is correlated with the mechanism of ischaemia; arterial mesenteric infarction and non-occlusive mesenteric ischaemia (NOMI) are three times more likely to result in death than venous infarction.[38] In general, the relatively slow process of mesenteric venous thrombosis is much less lethal than the very sudden interruption to blood supply caused by superior mesenteric artery embolus. Predictors of poor outcome include advanced age, chronic kidney disease, diabetes, large bowel involvement, evidence of organ hypoperfusion (e.g., creatinine and/or lactate rise), and delayed surgery.[93] Historically, NOMI has resulted in 70% to 80% mortality; however, more recent data suggest mortality rates from NOMI are more like 30% to 55%.[91] [94] [95] [96]

Diagnosis before the occurrence of intestinal infarction is the most important factor in improving survival for patients with acute mesenteric ischaemia. This is supported by several retrospective studies in which diagnosis within 24 hours of presentation to a physician, or before any significant bowel infarction occurred, resulted in markedly improved survival. In a report of 21 patients with superior mesenteric artery embolus, intestinal viability was achieved in 100% of patients if the duration of symptoms was <12 hours, in 56% if it was between 12 and 24 hours, and in only 18% if symptoms were >24 hours in duration before diagnosis.[97]

Chronic mesenteric ischaemia

Mortality rates for surgical revascularisation tend towards the lower end of a range from 0% to 16%, with success rates of >90%, and recurrence rates generally <10%.[65]

Several long-term studies have shown that patients who survive surgical revascularisation have cumulative 5-year survival rates of 81% to 86%.[65]

Colonic ischaemia

Colonic ischaemia carries the most favourable prognosis of the varying forms of bowel ischaemia; nevertheless, 20% will develop chronic ulcerating ischaemic colitis.[21] Poor prognosis is associated with male sex, right-sided ischaemia, and lack of rectal bleeding at presentation.[33]

Diagnostic guidelines

Europe

Management of the diseases of mesenteric arteries and veins

Published by: European Society of Vascular Surgery **Last published:** 2017

International

Acute mesenteric ischemia

Published by: World Society of Emergency Surgery **Last published:** 2022

North America

ACR appropriateness criteria: radiologic management of mesenteric ischemia

Published by: American College of Radiology **Last published:** 2022

ACR appropriateness criteria: imaging of mesenteric ischemia

Published by: American College of Radiology **Last published:** 2018

ACG clinical guideline: epidemiology, risk factors, patterns of presentation, diagnosis, and management of colon ischemia

Published by: American College of Gastroenterology **Last published:** 2015

Treatment guidelines

Europe

Management of the diseases of mesenteric arteries and veins

Published by: European Society of Vascular Surgery **Last published:** 2017

ESTES guidelines: acute mesenteric ischaemia

Published by: European Society for Trauma and Emergency Surgery **Last published:** 2016

International

Acute mesenteric ischemia

Published by: World Society of Emergency Surgery **Last published:** 2022

North America

Chronic mesenteric ischemia: clinical practice guidelines from the Society for Vascular Surgery

Published by: Society for Vascular Surgery **Last published:** 2021

ACG clinical guideline: epidemiology, risk factors, patterns of presentation, diagnosis, and management of colon ischemia

Published by: American College of Gastroenterology **Last published:** 2015

Key articles

- Bala M, Catena F, Kashuk J, et al. Acute mesenteric ischemia: updated guidelines of the World Society of Emergency Surgery. World J Emerg Surg. 2022 Oct 19;17(1):54. Full text Abstract
- Tilsed JV, Casamassima A, Kurihara H, et al. ESTES guidelines: acute mesenteric ischaemia. Eur J Trauma Emerg Surg. 2016 Apr;42(2):253-70. Full text Abstract
- 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 May;8(4):371-95. Full text Abstract

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Images

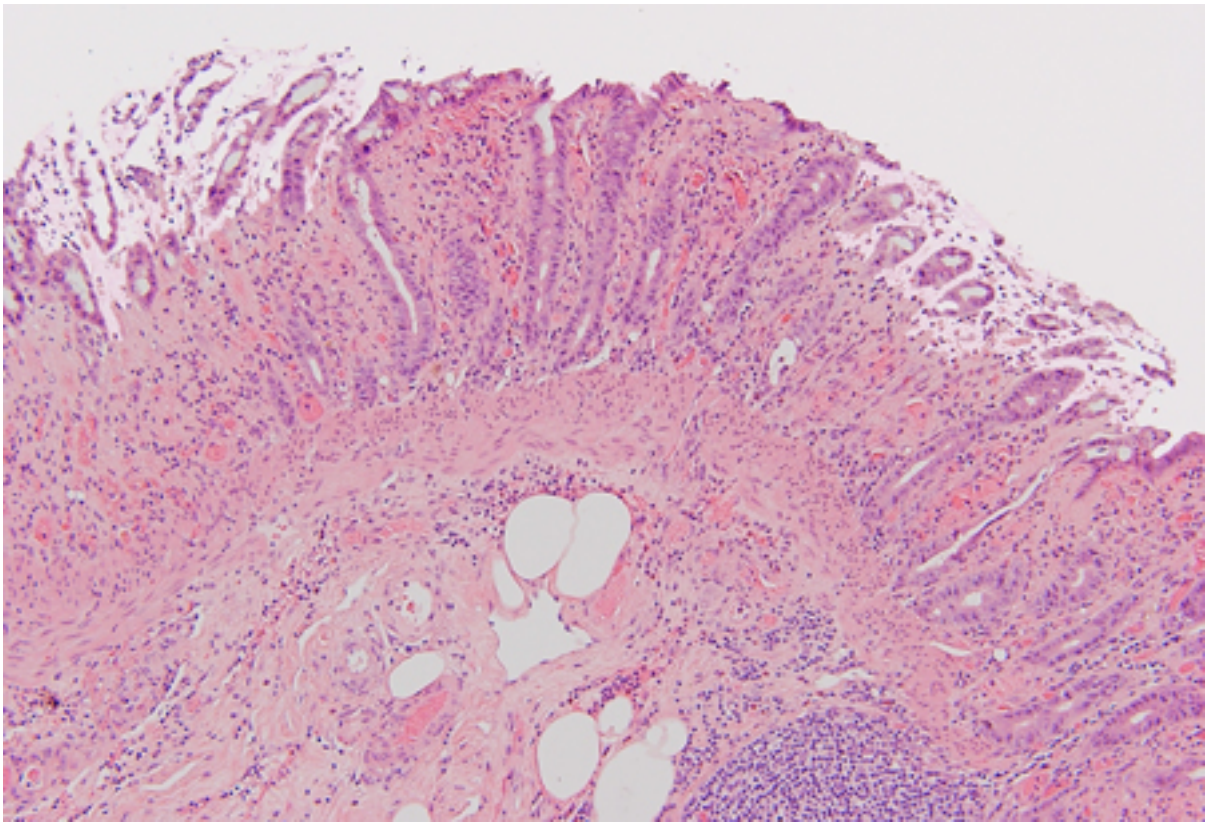

Figure 1: Histopathology of intestinal ischaemia

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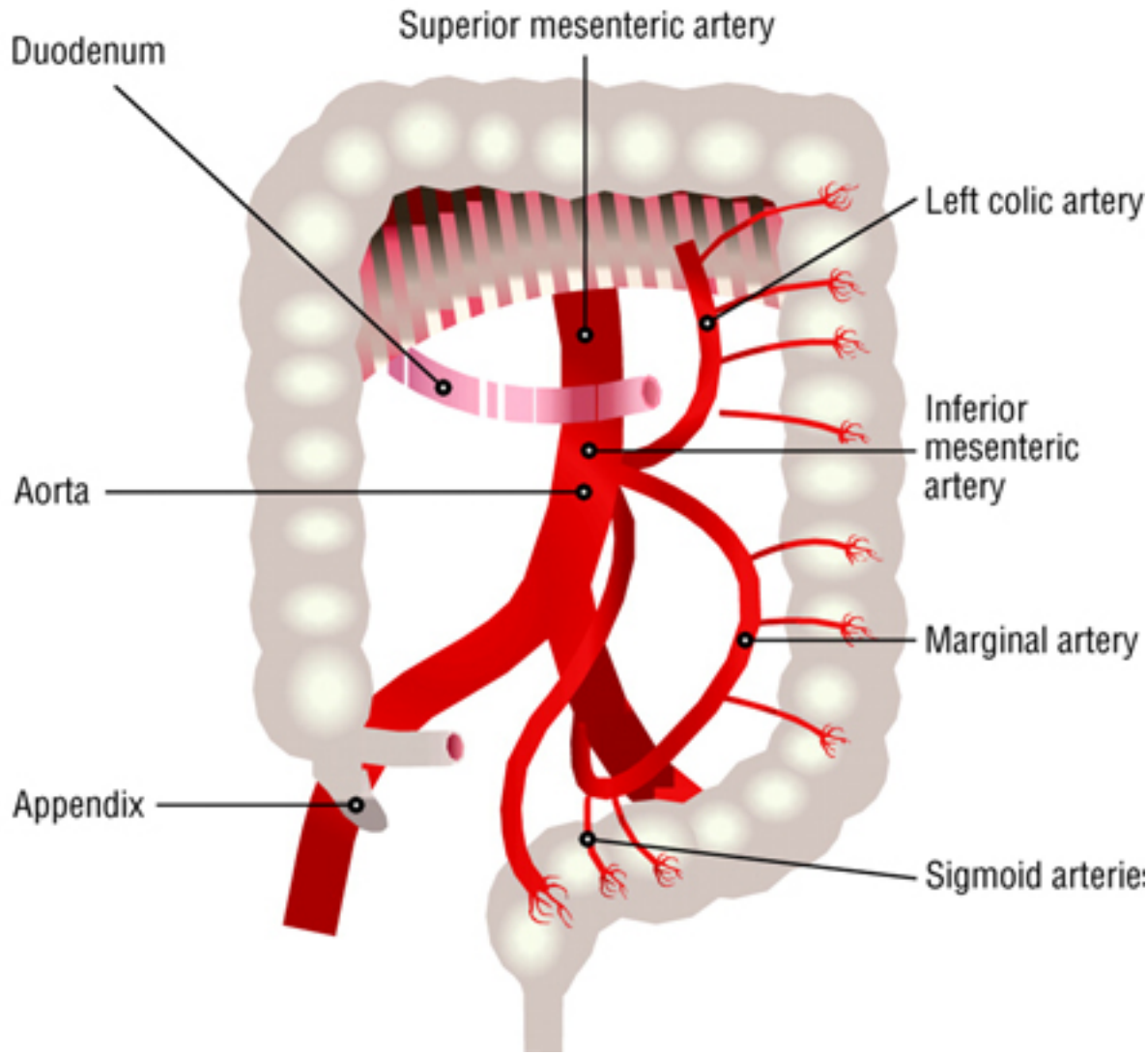

Figure 2: Distribution of blood flow to the colon originating from the inferior mesenteric artery, branches of which include the left colic, marginal, and sigmoid arteries and supply the left colon and superior portion of the rectum

BMJ 2003; 326 doi: 10.1136/bmj.326.7403.1372

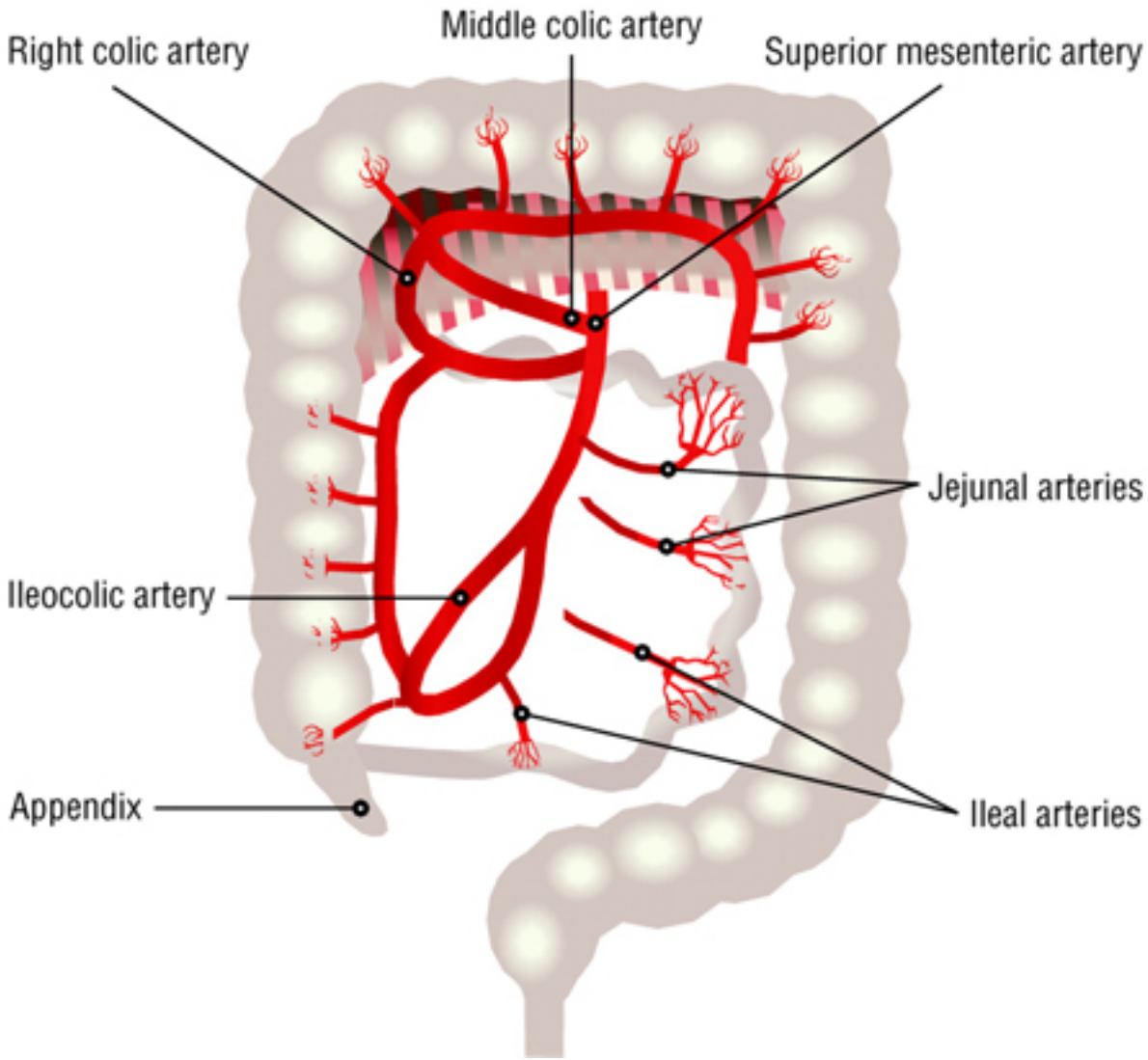

Figure 3: Distribution of blood supply to the small intestine and colon from the superior mesenteric artery, branches of which include the middle, right, and ileocolic arteries as well as jejunal and ileal arteries and arterioles

BMJ 2003; 326 doi: 10.1136/bmj.326.7403.1372

	Acute mesenteric ischaemia	Chronic mesenteric ischaemia	Colonic ischaemia
Site	• Periumbilical pain • Upper abdominal or generalised	Epigastric or mid abdominal	Left lower quadrant or lower abdominal
Onset	Sudden	Insidious	Sudden
Character	• Sharp or colicky • Pain is out of proportion to the exam	Dull	Dull
Radiation	No radiation	No radiation	Radiates to back
Associations	• Nausea, vomiting, diarrhoea • May have sudden forceful bloody bowel evacuation	Nausea, vomiting	Bloody bowel movements
Timing, duration, frequency	Acute	Chronic	Acute
Exacerbating and relieving factors	No association with meals, pain not relieved	Worse after meals, resolving over hours	None
Severity	Severe	Mild	Mild-to-moderate
Abdominal examination	• Soft, non-tender with hyperactive bowel sound - early phase • Rebound tenderness, rigidity with absent bowel sound - late phase	Abdominal bruit	LLQ or lower abdominal tenderness
Cardiovascular exam	May have a fibrillation or other arrhythmia, evidence of vascular disease	Atherosclerosis, peripheral vascular disease	May have a fibrillation or other arrhythmia, atherosclerosis, evidence of peripheral vascular disease
Laboratory test results	• Leukocytosis • Elevated serum amylase • Lactic acidosis		
Imaging	• Thumbprinting on plain x-rays • Mesenteric occlusion on angiography • Subdiaphragmatic air if perforated • Pneumatosis intestinalis, or air in portal vessels when bowel necrosis present • Use contrast-enhanced CT to diagnose mesenteric venous thrombosis	Angiography demonstrates severe occlusion of at least 2 of the 3 splanchnic vessels	Angiography has no role
Typical patient characteristics	• Older patients with cardiovascular disease • Younger patients with collagen vascular disease, vasculitis, hypercoagulable state, vasoactive medication use, or cocaine use	Older women Smoker Cardiovascular disease Sitophobia	Older patients with cardiovascular disease or atrial fibrillation

IMAGES

Figure 4: Comparison of symptoms/signs and investigations for the three types of ischaemic bowel disease

Designed by BMJ Knowledge Centre, with input from Dr Amir Bastawrous

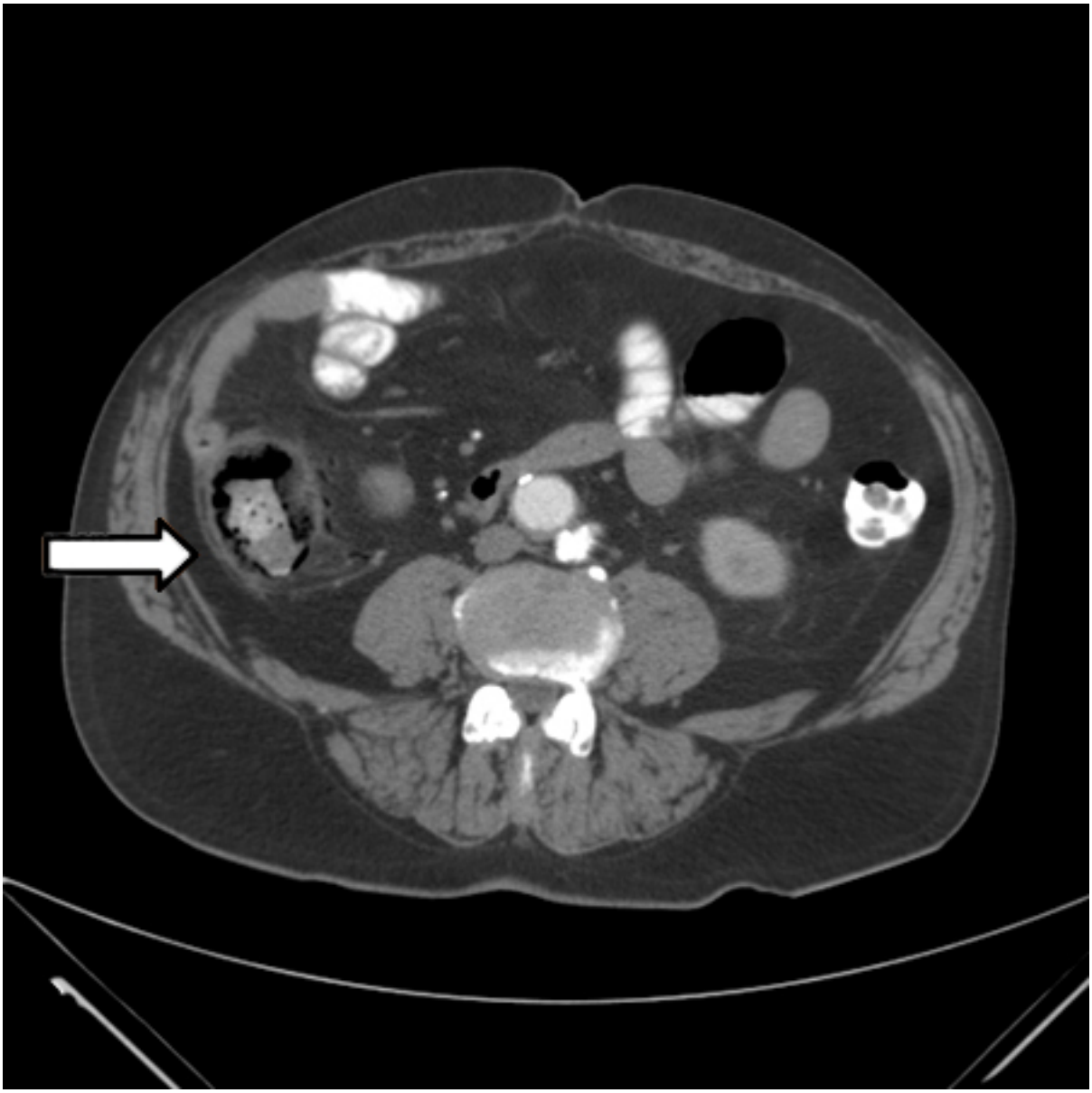

Figure 5: CT scan: colonic thickening with pneumatosis intestinalis

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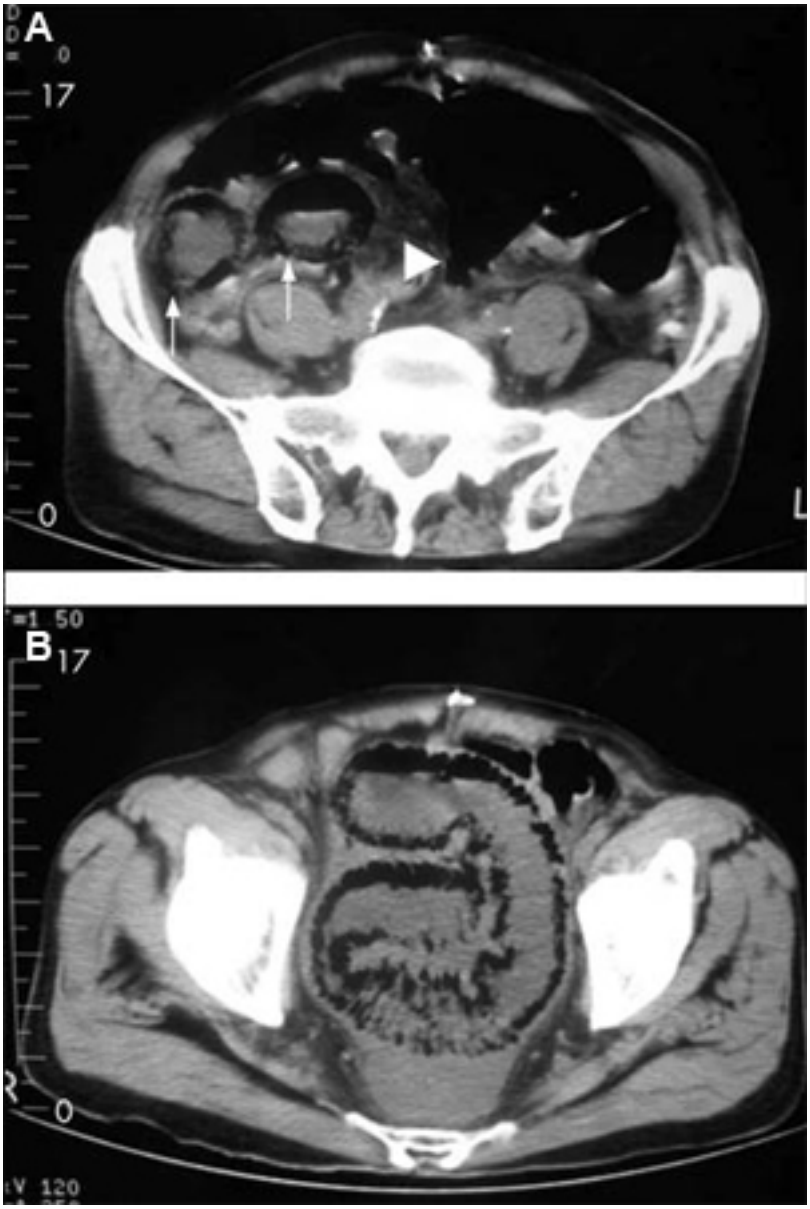

Figure 6: 84-year-old man presenting with symptoms suggestive of ischaemic bowel disease: (A) Abdominal CT revealing a massive circumferential and band-like air formation as intestinal pneumatosis (arrows) and pronounced oedema of mesenteric fat (arrowhead) around necrotic bowel loops; (B) Another slice of abdominal CT showing long segmental pneumatosis of the small bowel

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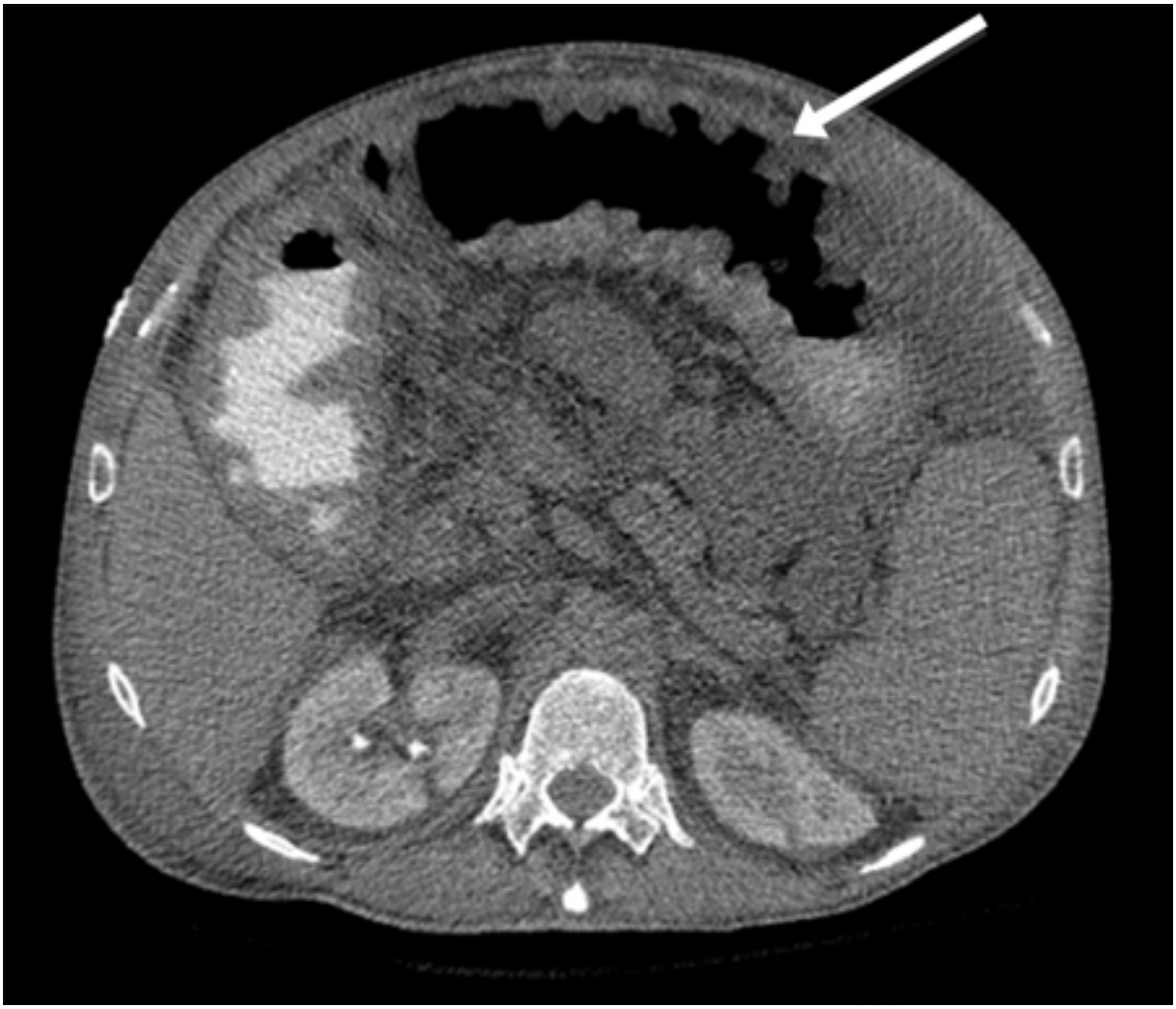

Figure 7: CT scan: circumferential wall thickening of the transverse colon; white arrow shows thumbprinting

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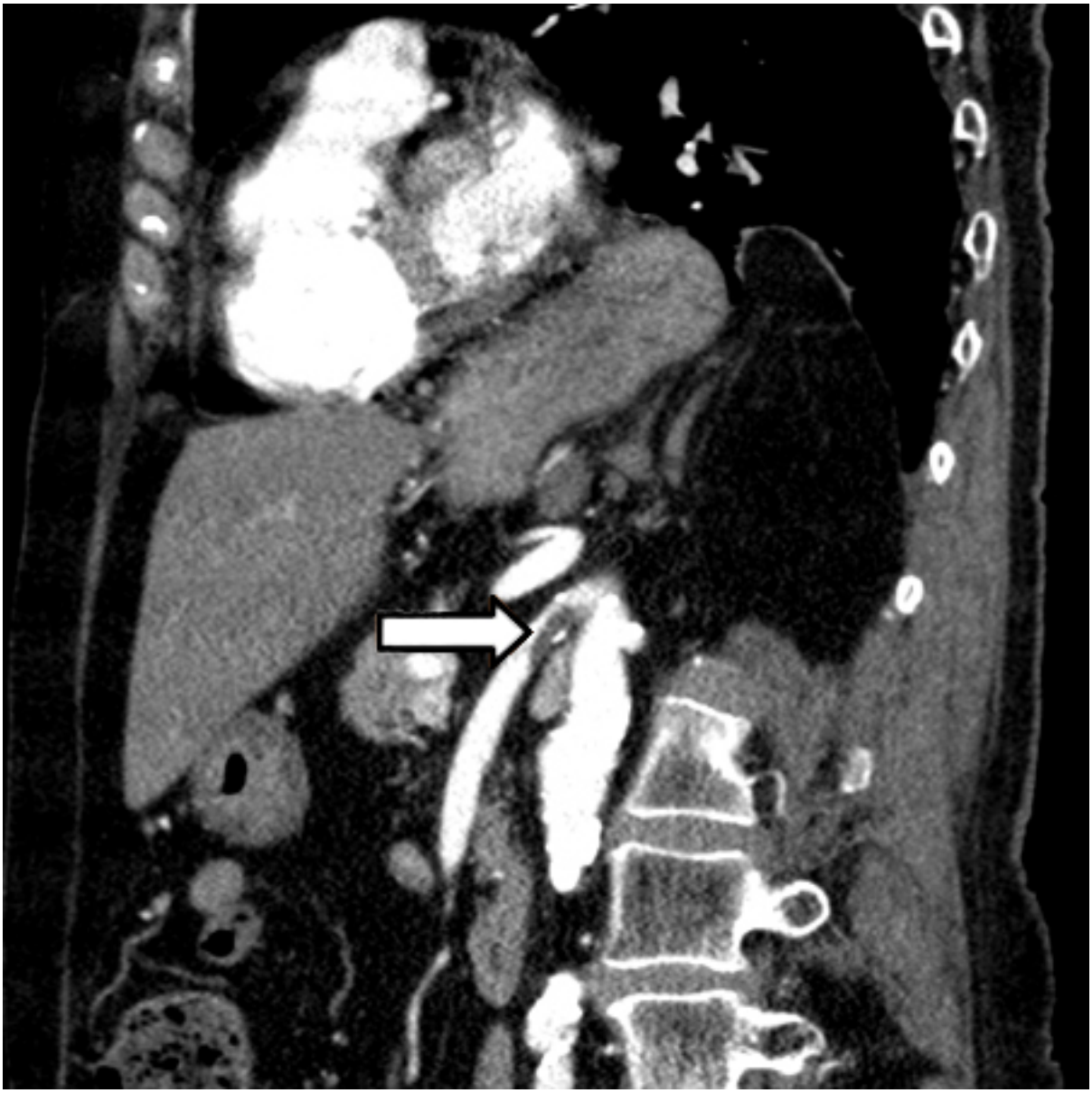

Figure 8: CT angiogram: Acute superior mesenteric artery thrombus

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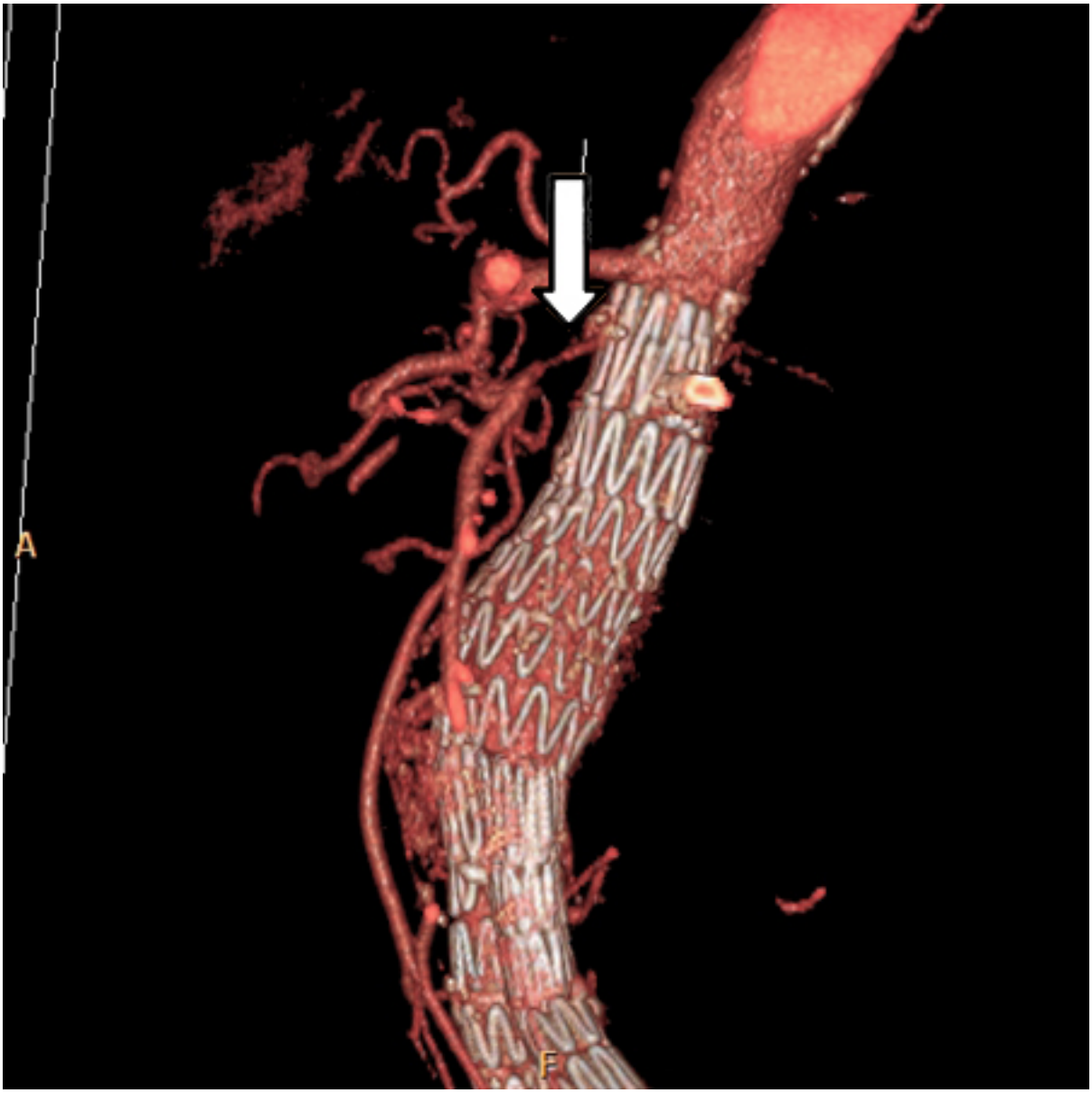

Figure 9: CT angiography: 3-dimensional reconstruction with superior mesenteric artery stenosis from severe atherosclerotic plaque in a patient on follow-up imaging for endovascular aneurysm repair

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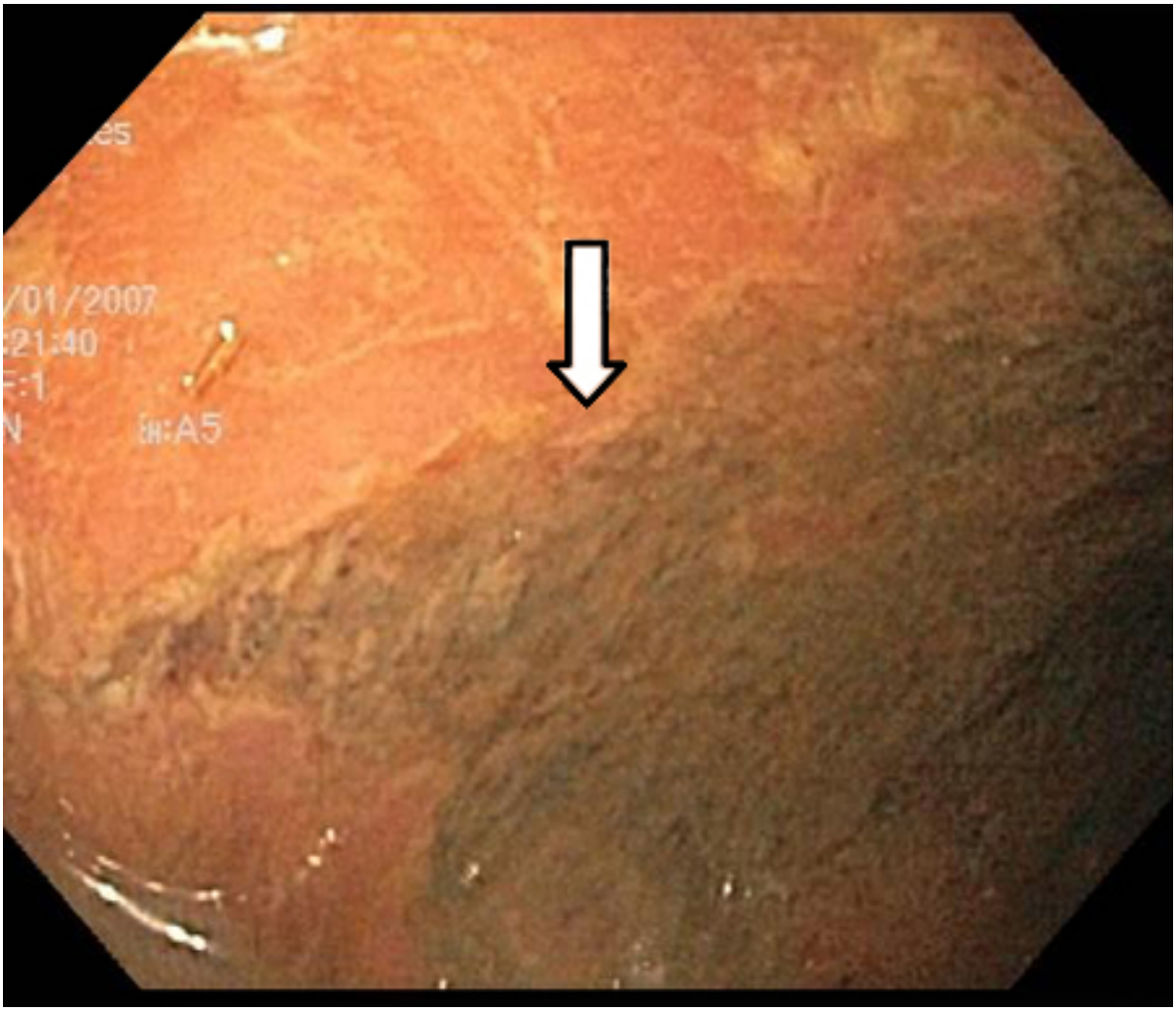

Figure 10: Colonoscopy: demarcation between ischaemic and normal colon

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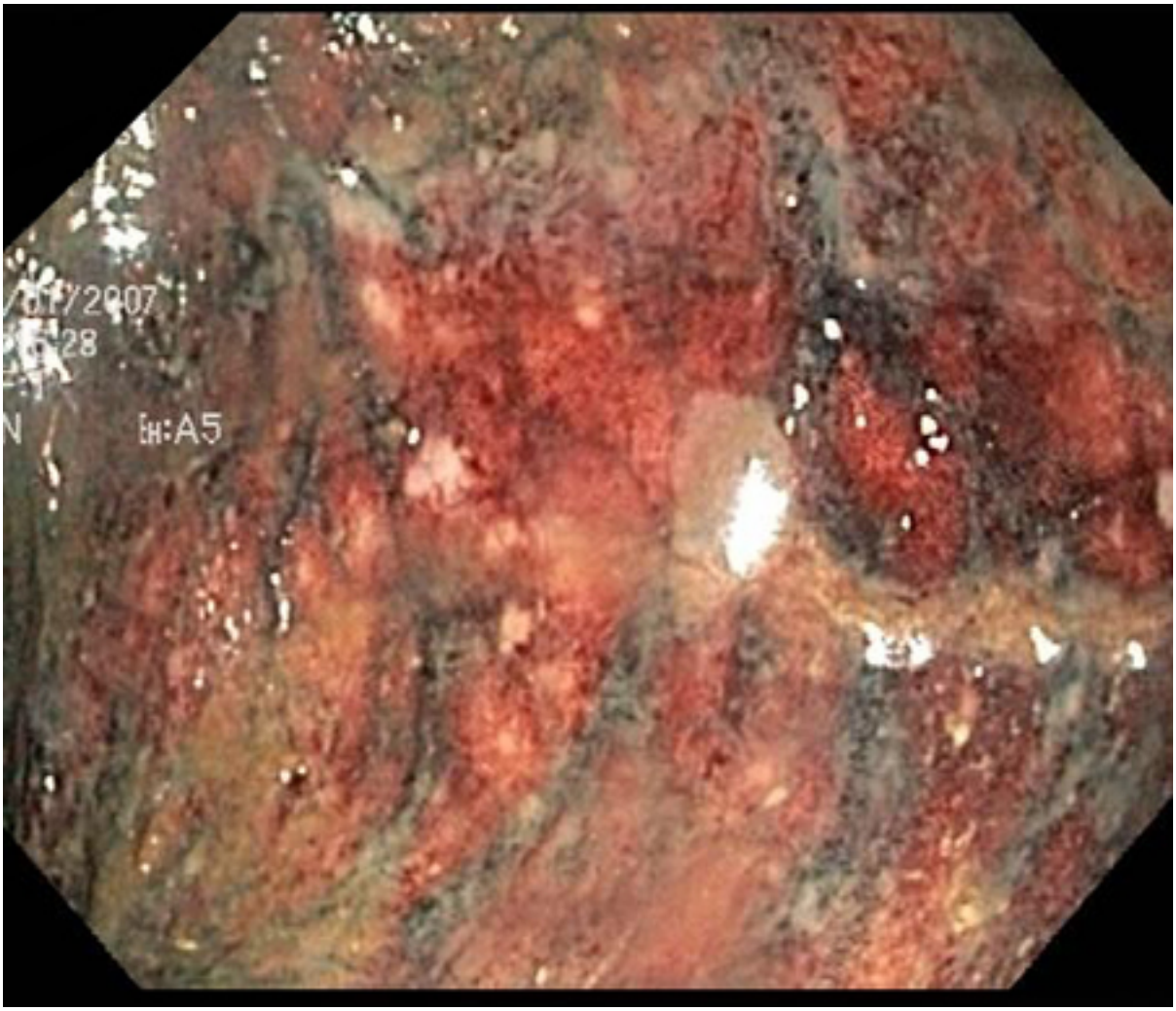

Figure 11: Colonoscopy: denudation of colonic mucosa

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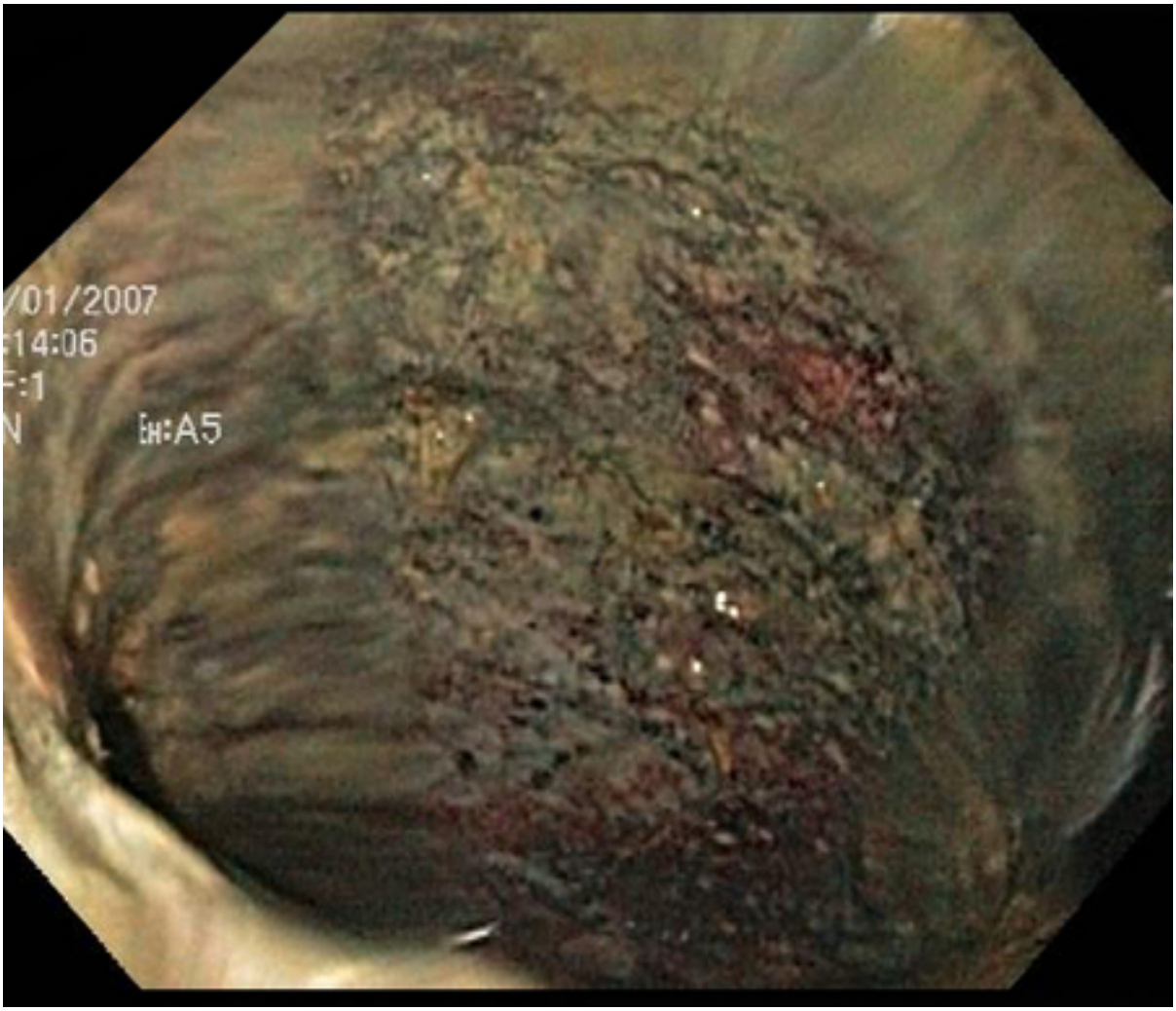

Figure 12: Colonoscopy: mucosal sloughing and likely to be non-viable colon

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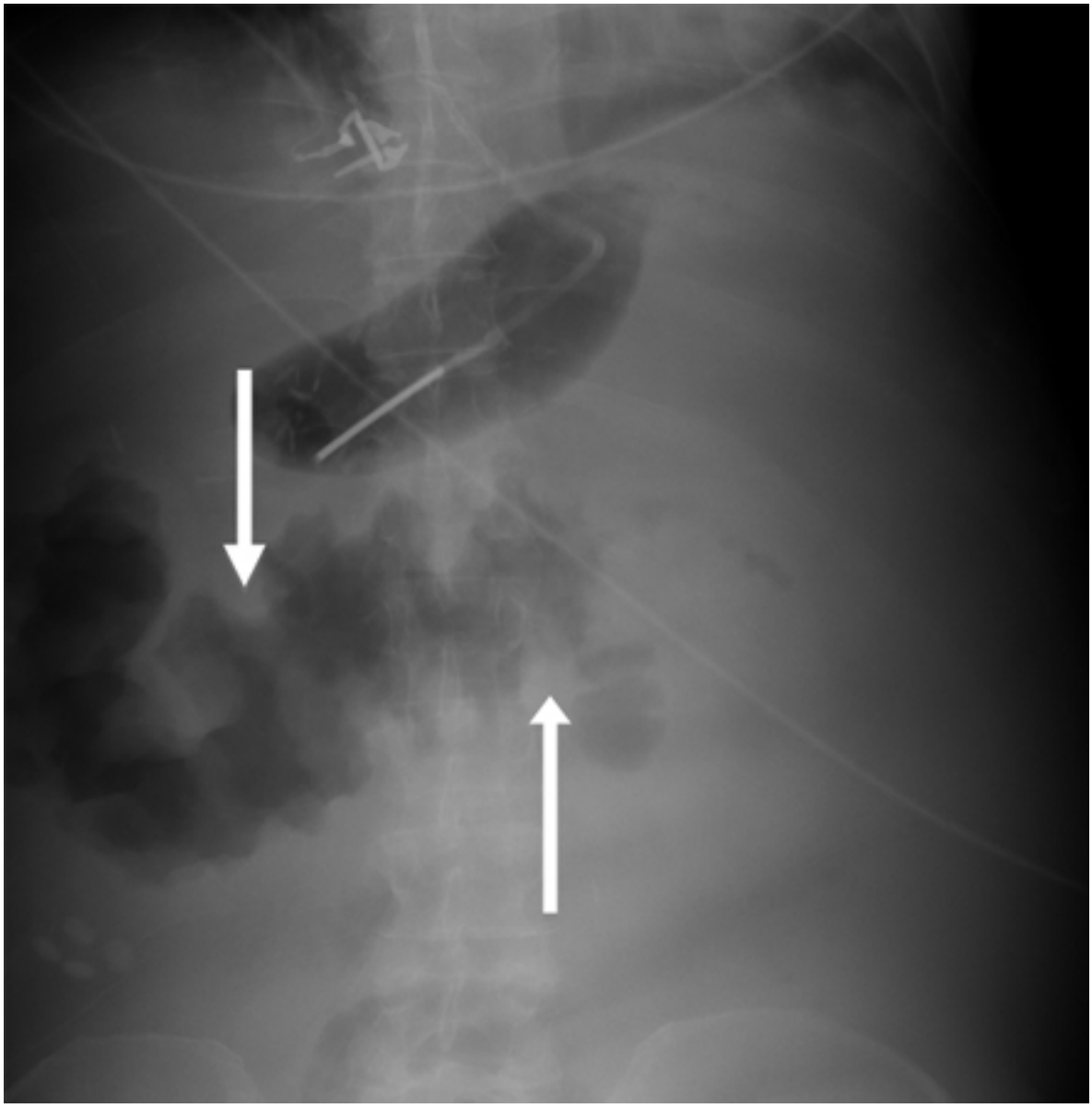

Figure 13: Plain abdominal x-ray: shows marked wall thickening of the transverse colon compatible with the finding of thumbprinting (white arrows)

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Figure 1 – BMJ Best Practice Numeral Style

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