BMJ Best Practice

Ischaemic bowel disease

The right clinical information, right where it's needed

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Summary

- Encompasses a heterogeneous group of disorders caused by acute or chronic processes, arising from occlusive or non-occlusive aetiologies that result in decreased blood flow to the gastrointestinal tract.
- Intestinal ischaemia can be classified into 3 types: acute mesenteric ischaemia, chronic mesenteric ischaemia, and colonic ischaemia. Colonic ischaemia is the most common type and has the most favourable prognosis.
- It 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 sub-acute 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.

BASICS

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.
[Fig-1]

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. 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, and other factors increase the risk of developing colonic ischaemia 2- to 4-fold.[5] [6]

Acute mesenteric ischaemia accounts for approximately 0.1% of hospital admissions.[7] One study in Sweden found that between 1970 and 1982 the overall incidence of acute thrombo-embolic 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 aged >85 years.[8] It also occurs more commonly in those with comorbidities, most notably atrial fibrillation, MI, and atherosclerosis. Non-occlusive mesenteric ischaemia (NOMI) accounts for 20% to 30% of acute mesenteric ischaemia.[9] [10] The overall incidence of NOMI is 2 cases per 100,000 person-years, increasing to 40 per 100,000 in patients aged >80 years.[11]

Aetiology

Arterial compromise:

- Embolism
    - Responsible for approximately 50% of acute mesenteric ischaemia events. 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.[9] [12] [13] [14] Interventional radiological procedures are the usual cause of iatrogenic 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.[9] [15] Mesenteric atherosclerotic plaques may rupture with associated acute thrombosis of the vessel. Sub-acute or chronic ischaemia may result from partial occlusion of the vessel.
- Vasculitis
    - Rheumatoid arthritis, polyarteritis nodosa, SLE, 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.[16]
- 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 approximately 5% of cases of acute mesenteric ischaemia. Frequently involves the superior mesenteric vein.
  - Usually associated with cirrhosis or portal hypertension; other potential associations include inheritable hyper-coagulable 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 DVT or pulmonary embolus.[16]

Hypo-perfusion (i.e., non-occlusive ischaemia):

- Accounts for 20% to 30% of cases of acute mesenteric ischaemia.
- Shock, or hypotension, or relative mesenteric hypotension (from any aetiology). Prominent causes include:
  - Heart failure.
  - Dialysis.
  - Drug-related
    - Such as digitalis, oestrogen, contraceptives, vasopressin, vasopressors, danazol, flutamide, glycerin enema, alosetron, immunosuppressives, psychotropics, imipramine, adrenaline (epinephrine), sumatriptan, non-steroidal anti-inflammatory drugs (NSAIDs), ergot, diconal, laxatives, pegylated interferon, methamphetamines, and cocaine.[17]
  - Recent surgery
    - Such as aortic aneurysm repair, aorto-iliac bypass, colectomy, colonoscopy.
  - Infection
    - Such as CMV, 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 (CA) 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 2 watershed areas where collateralisation of blood flow may be limited.
[Fig-2]

[Fig-3]

Ischaemia occurs secondary to hypo-perfusion of an intestinal segment. When hypo-perfusion occurs, collateral blood flow may preclude or minimise 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. Re-perfusion injury can also occur if blood supply is re-established after a prolonged interruption. Segments of bowel which do not cause acute necrosis or perforation can heal with stenosis or stricture. These can cause ischaemic bowel disease with long-term sequelae, which is either mild and chronic or acute and resolved.

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 while the other vessels form more oblique angles from the aorta.

Classification

American Gastroenterological Association[1]

Intestinal ischaemia can be classified into 3 broadly defined types:

- 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.

Primary prevention

Given the contribution of cardiovascular disease to many cases of ischaemic bowel disease, 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, although good supporting data are lacking.

Case history

Case history #1

A 72-year-old male presents to the emergency department with sudden-onset, diffuse abdominal pain that began 18 hours ago. He has not been vomiting, but he has had several episodes of diarrhoea, the last of which was bloody. He was hospitalised 1 week ago for an acute MI.

Case history #2

A 48-year-old female complains of intermittent diffuse abdominal pain, worse after eating meals. The pain has been present for the previous 6 months, but has worsened recently. She has had significant weight loss since the onset of symptoms. Her past medical history includes SLE, which has been difficult to manage medically.

Other presentations

Up to 6.7% of patients who have undergone open or endovascular cardiac or major vascular procedures develop colonic ischaemia, and the mortality may be as high as 67% 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 hypo-perfusion in the postoperative period, and a relatively high incidence of heart failure in these patients.

Step-by-step diagnostic approach

Clinicians must maintain a high index of suspicion for ischaemic bowel disease, because the signs and symptoms are relatively non-specific yet the condition has significant morbidity and mortality. Early recognition, appropriate diagnostic studies, and aggressive treatment are necessary to improve outcome.

In the absence of highly specific or definitive signs and symptoms, a history and physical examination alone are generally not sufficient to make the diagnosis; usually some form of imaging is required. However, when fulminant ischaemic bowel disease is present, extensive diagnostic testing may not be appropriate in order that surgical intervention can proceed without delay.

Where clinically indicated, resuscitation should be administered in parallel with the diagnostic work-up in order to minimise the risk of ischaemia progressing. Resuscitation should include administration of supplemental oxygen, adequate fluid replacement, and correction of acute heart failure or arrhythmias.

Clinical presentation and history

Presenting features and history may vary widely, as ischaemic bowel disease encompasses a wide spectrum of disorders. History taking needs to be thorough in order to be able to exclude other potential diagnoses confidently. The history should explore the key characteristics of the abdominal pain, using the mnemonic SOCRATES:

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

Sudden onset of diffuse abdominal pain suggests acute mesenteric ischaemia or non-occlusive mesenteric ischaemia. However, chronic symptoms of vague, diffuse abdominal pain may be indicative of chronic mesenteric ischaemia. In contrast, colonic ischaemia may cause focal or diffuse abdominal pain and often has a more insidious onset, over several hours or days.

Other important elements to elicit include smoking history, cardiovascular risk factors, comorbidities, and past medical history. Suggestive findings for each of the possible types of ischaemic bowel disease are as follows:

Acute mesenteric ischaemia

- Older patients with long-standing congestive heart failure, cardiac arrhythmias, recent MI, hypotension, or peripheral vascular disease.
- Younger patients with history of collagen vascular disease, vasculitis, hyper-coagulable state, vasoactive medicine or cocaine use.
- Patients with arterial embolus who describe sudden, severe abdominal pain with rapid, forceful bowel evacuation, possibly containing blood.
- 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.[20]
- Pain generally persists for >2 or 3 hours. Pain often peri-umbilical.
- Patients usually appear severely ill, particularly if presentation has been delayed.
- Clinical picture does not suggest some other abdominal pathology.[21]

Chronic mesenteric ischaemia

- Usually occurs in older people.
- Women affected more than men (ratio 3:1).
- Patients frequently give history of heavy smoking and other symptoms associated with atherosclerosis.
- Insidious onset with repeated, mild, transient, episodes over many months, becoming progressively more severe over time.
- Pain often occurs after meals, gradually resolving over a few hours.
- Patients may have associated nausea, and diarrhoea with or without blood.
- Infarction of bowel uncommon, as insidious onset allows some collateral circulation to develop.
- Pain poorly localised.
- May be significant weight loss, giving the patient a cachectic appearance.
- Patients may describe having become fearful of eating (sitophobia).

Colonic ischaemia

- Represents the most common form of intestinal ischaemia.[21] About 90% of cases occur in patients >60 years of age.
- More than 80% of cases resolve spontaneously or with conservative measures, but surgery may be required acutely, sub-acutely or in chronic cases.
- Soon after the onset of ischaemia, there is pain with frequent bloody, loose stools, reflecting mucosal or sub-mucosal damage. However, 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 which is usually felt laterally, in contrast to the pain of acute mesenteric ischaemia which is often described as peri-umbilical.
- Tenderness to palpation over the affected bowel from early in the course of ischaemia, in contrast to acute mesenteric ischaemia where tenderness is a relatively late sign.
- 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.
- The following have been identified as important risk factors for colonic ischaemia: age >60 years, haemodialysis, hypertension, hypo-albuminaemia, diabetes mellitus, constipation-inducing medicines.[22]
- Patients do not generally appear severely ill, unless fulminant ischaemia present.
- Increasingly identified in younger people, associated with strenuous and prolonged physical exertion (e.g., long-distance running), various medicines (e.g., oral contraceptives), cocaine use, and coagulopathies (e.g., protein C and S deficiencies, anti-thrombin III deficiency, activated protein C resistance).[1]
- Other circumstances when colonic ischaemia may occur include:
    - following aortic or cardiac bypass surgery
    - in association with vasculitides such as SLE or polyarteritis nodosa, infections (e.g., CMV, *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 barium enema; mesenteric angiography plays little role in diagnosis unless only the right side of the colon is affected or the patient reports more pain than is usual.[21]

Non-occlusive ischaemia (mesenteric or colonic)

- May present in patients with underlying hypotension and volume deficits, which may be related to congestive heart failure, hypo-volaemia, sepsis, and cardiac arrhythmias, or haemodialysis.[20]

Coeliac compression syndrome

- Occurs due to the median arcuate ligament compressing the coeliac axis.
- Consider this diagnosis particularly in younger patients (especially women) with unexplained abdominal pain and normal upper endoscopy, normal liver, pancreatic and gastric laboratory studies, particularly in those patients who have an abdominal bruit (from partially obstructed flow in the coeliac axis).
- Can be considered part of the chronic mesenteric ischaemia category.

	Acute mesenteric ischaemia	Chronic mesenteric ischaemia	Colonic ischaemia
Site	Periumbilical pain. Focal pain if necrosis present.	Poorly localised.	Lateral abdomen or flanks. Focal pain if necrosis present.
Onset	Sudden.	Insidious.	Sudden. May become continuous and diffuse if it progresses.
Character	Sharp or colicky. Pain is out of proportion to the exam.	Repeated, transient episodes of pain, progressing with time.	Dull.
Radiation	No radiation.	No radiation.	Radiates to back.
Associations	Nausea, vomiting, diarrhoea. May have sudden forceful bloody bowel evacuation.	Nausea, vomiting.	Nausea, vomiting, diarrhoea. Passage of maroon stools.
Timing, duration, frequency	2-3 hours (arterial) or 5 to more than 30 days (venous).	Months.	Acute, subacute, or chronic.
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	Epigastric bruit and distention.		Abdominal distention and no bowel sounds as ischaemia progresses.
Cardiovascular exam	May have a fibrillation or other arrhythmia, evidence of peripheral vascular disease.	Atherosclerosis, peripheral vascular disease.	May have a fibrillation or other arrhythmia, atherosclerosis, evidence of peripheral vascular disease.
Laboratory test results	Leukocytosis, metabolic acidemia, and elevated serum amylase.		
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. Barium enema can be used if colonoscopy is not available (cobblestone appearance, thumbprinting, stricture).
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 Evidence Centre, with input from Dr Amir Bastawrous

Physical examination

Examination should begin with an assessment of vital signs in order to determine if immediate resuscitation measures are required. This should be followed by full and thorough examination of all systems, particularly focusing on the abdomen and cardiovascular system, for clues that may help secure the diagnosis.

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, which 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 re-bound, 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.[20]
- Auscultation of the abdomen reveals an epigastric bruit (indicative of turbulent flow through an area of vascular narrowing) in 48% to 63% of patients.[16]
- Rectal examination may demonstrate gross blood per rectum or microscopic blood upon testing for occult haemorrhage.
- Peritonitis indicates need for urgent surgical intervention.

Cardiovascular examination:

- Identifies murmurs or arrhythmias.
- May reveal bruits on carotid auscultation, along with skin changes, absent hair, and absent distal pulses on the limbs, consistent with advanced atherosclerotic disease.
- When ischaemic bowel disease is associated with vasculitis or specific disease entities, characteristic dermatological, musculoskeletal, or further findings specific to the disease may be present.

Laboratory tests

Initial blood work should include:

- FBC
- Chemistry panel
- Serum amylase
- Arterial blood gases.

These results may direct initial resuscitation, help assess the severity of any ischaemia, and provide clues to alternative diagnoses.

There are no specific laboratory tests for the diagnosis of acute mesenteric ischaemia or intestinal infarction, but leukocytosis, acidosis, and elevated serum amylase usually occur late in the course of ischaemic bowel. On admission, approximately 75% of patients with acute mesenteric ischaemia have a leukocytosis of >15,000 cells/mm^3, and about 50% have metabolic acidaemia.[20]

Imaging

Patients being investigated for potential ischaemic bowel disease should have plain x-rays and/or CT scans of the abdomen as guided by clinical findings. As well as providing clues towards a diagnosis of ischaemia, these investigations help exclude other potential diagnoses.

If no alternative diagnosis is made following these studies, then selective angiography is indicated. Based on the angiographic findings, the patient should be treated according to the specific cause of the ischaemia.

In cases of acute ischaemia, if angiography is not immediately available, prompt exploratory laparotomy is indicated in patients with suspected ischaemic bowel. Laparotomy without prior angiography may be indicated in unstable patients with peritoneal signs.

Abdominal x-ray:

- Plain x-rays are often normal early in the course of ischaemia or when ischaemia is mild. In one series, 6 of 23 patients (26%) with confirmed acute mesenteric ischaemia had normal abdominal x-rays.[23]
- With worsening ischaemia, plain x-rays may show formless loops of bowel, ileus, or thickening of the bowel wall with thumb-printing sign suggestive of sub-mucosal oedema or haemorrhage.
  [Fig-5]

Erect chest x-ray:

- May show sub-diaphragmatic air, indicative of perforation of the bowel requiring prompt surgical intervention.

CT scanning:

- Helpful in diagnosing acute mesenteric ischaemia, but findings can be non-specific in early ischaemia; late signs indicate necrotic bowel. 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.[20] [24]CT abdomen: circumferential wall thickening of the transverse colon; white arrow shows thumbprinting.May show thickening of the bowel wall with thumb-printing sign suggestive of submucosal oedema or haemorrhage.
  [Fig-6]

  [Fig-7]

  [Fig-8]
- 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 (SMV), 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.[20]
- In many centres, CT angiography is replacing conventional angiography as standard practice for evaluation of the mesenteric vasculature and diagnosis of acute mesenteric ischaemia; however, mesenteric angiography currently remains the definitive test.
  [Fig-9]

  [Fig-10]

Mesenteric angiography:[20]

- The definitive investigation for assessing occlusive and non-occlusive forms of ischaemia. Provides direct imaging of the splanchnic vasculature.
- Sensitivity is 74% to 100%, specificity 100%.
- The only method that can diagnose non-occlusive mesenteric ischaemia (NOMI) before infarction occurs. Four criteria are used for this purpose:

  1. Narrowing of the origins of the superior mesenteric artery (SMA) branches
  2. Irregularities in these branches

3. Spasm of the mesenteric arcades
4. Impaired filling of the intra-mural vessels.

- Enables treatment by infusion of vasodilators or thrombolytic agents (which have been shown to improve outcome).
- 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.[1]
- Mesenteric angiography plays little role in the diagnosis of colonic ischaemia unless only the right-side colon is affected or the patient reports more pain than is usual.[21]

Colonoscopy:

- The best test to establish the diagnosis of colonic ischaemia and establish severity. However, if urgent surgical intervention is required due to the condition of the patient, surgery should not be delayed to carry out this investigation.
  [Fig-11]

  [Fig-12]

  [Fig-13]
- Repeated clinical evaluation and serial endoscopic studies of the colon are often required to establish the diagnosis of colonic ischaemia, as symptoms (and accompanying signs) are frequently transient and resolve spontaneously.
- Mesenteric angiography plays little role in the diagnosis of colonic ischaemia unless only the right-side colon is affected or the patient reports more pain than is usual.[21]

Barium enema:

- May be used for the diagnosis of colonic ischaemia if colonoscopy is not available. Classic early findings of colonic ischaemia include thumb-printing (due to sub-mucosal oedema and haemorrhage) and pseudo-tumours. Other potential findings include cobble-stoning and stricture formation.

Mesenteric duplex ultrasound:

- 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.

Magnetic resonance angiograph (MRA):

- May have a role in making the diagnosis of chronic mesenteric ischaemia. The time required to perform MRI examinations, and the possible need for bowel stimulation with a meal, limit the usefulness of MRI in the diagnosis of acute mesenteric ischaemia. Overall, CT angiography is likely a better examination than MRI for the diagnosis of chronic mesenteric ischaemia (CMI) because of its capacity for higher resolution in combination with faster scans.

Risk factors

Strong

old age

- Older people frequently suffer from medical comorbidities such as atrial fibrillation, MI, advanced atherosclerosis, and heart failure, which are significant contributory factors to the development of ischaemic bowel disease.[14]

history of smoking

- A history of smoking, often in combination with peripheral vascular disease and hypertension, is also frequently present.[16]

hyper-coagulable states

- A strong risk factor for mesenteric venous thrombosis (MVT). Approximately 50% of patients presenting with MVT have had a DVT or pulmonary embolus in the past.[16] Common causes of hyper-coagulability include cirrhosis or portal hypertension; inheritable hyper-coagulable states such as factor V Leiden, protein C deficiency, or prothrombin G20210A mutation; oral contraceptive use; malignancy; pancreatitis; and a history of recent surgery.[16]

atrial fibrillation

- Untreated, atrial fibrillation can result in the formation of thrombi within the heart, which then embolise to the mesenteric vasculature.[14]

myocardial infarction

- Impaired wall motion secondary to MI can act as a nidus for thrombus formation, 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, SLE, 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, and the mortality may be as high as 67% in this population.[2] [3] Factors that may underlie these figures include emboli arising from cross-clamping of the aorta, a risk of intestinal hypo-perfusion in the postoperative period, and a relatively high incidence of heart failure in these patients.

shock

- Hypo-perfusion 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. When atherosclerosis occurs in more distant vessels, it can act as a source of emboli.
- Severe atherosclerosis in vessels supplying the gut also makes individuals more vulnerable to bowel ischaemia arising from reductions in perfusion arising from congestive heart failure or shock.

previous ileostomy

- A previous ileostomy for any reason is associated with a 3.8-fold increased risk of developing colonic ischaemia.[6] This association is likely due to the underlying reason for the ileostomy itself - such as ulcerative colitis or infectious colitis (which may result in the need for ileostomy) may in itself increase the risk of ischaemic bowel disease.

irritable bowel syndrome

- The diagnosis of irritable bowel syndrome is associated with a 2-fold increased risk of developing colonic ischaemia.[6] 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, diverticulitis or volvulus. The proximal colonic distension leads to intra-luminal dilation and increased pressure that may result in decreased mucosal perfusion.

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.[18] [19]

use of vasopressors, digitalis, cocaine

- Especially in the setting of severe atherosclerosis, use of vasopressors, digitalis and cocaine have been shown to exacerbate non-occlusive mesenteric ischaemia.[16]

History & examination factors

Key diagnostic factors

abdominal pain (common)

- The majority of patients with ischaemic bowel experience pain, which can vary depending on the type and segment of bowel involved.

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 melaena or haematochezia.
- If bleeding is severe, this can potentially cause further hypo-perfusion and worsening of ischaemia.

diarrhoea (common)

- Mucosal sloughing occurs due to intestinal ischaemia, frequently causing episodes of diarrhoea.

abdominal tenderness (common)

- Perceived pain may be out of proportion to tenderness appreciated on physical examination.

weight loss (common)

- This is a notable feature of chronic mesenteric ischaemia (CMI), which is usually related to sitophobia (food fear) in these patients.[16]

abdominal bruit (common)

- Physical examination may reveal an epigastric bruit in 48% to 63% of patients with ischaemic bowel disease, indicative of turbulent flow through an area of vascular narrowing.[16]

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, and food fear may develop. This should not be confused with acute anorexia due to acute onset of pain or discomfort.

Diagnostic tests

1st test to order

Test	Result
FBC • On admission, approximately 75% of patients with acute mesenteric ischaemia have a leukocytosis >15,000 cells/mm^3 and about 50% have metabolic acidaemia.[20] • May reveal anaemia (often as a result of repeated episodes of melaena) that exacerbates ischaemia.	**leukocytosis, anaemia, evidence of haemoconcentration**

Test	Result
chemistry panel • Acidosis and elevated serum amylase usually occur late in the course of ischaemic bowel. • Also helps assess renal dysfunction and dehydration, frequently present in these patients.	**acidosis, uraemia, elevated creatinine, amylasaemia**
arterial blood gas/lactate level • The degree of acidosis aids in determination of the severity of illness. Although lactate is not specific, severely elevated levels and trends may aid in determination of the extent of illness.	**acidosis, elevated lactate**
ECG • May demonstrate arrhythmias or acute infarction that may be the aetiology of intestinal ischaemia.	**atrial fibrillation, arrhythmia, acute MI**
erect CXR • If perforation has occurred due to bowel necrosis, free air may be identified under the diaphragm. Immediate peri-operative resuscitation, empiric intravenous antibiotics and surgery are indicated if free air is identified.	**free air if perforation present**
abdominal x-rays • May demonstrate air-fluid levels or bowel dilation that may be the result of ischaemia or may indicate the aetiology of ischaemia, such as a distal obstruction. • May demonstrate distension or pneumatosis in advanced colonic ischaemia. • Presence of thumb-printing sign, indicative of mucosal oedema, suggests a worse prognosis. [Fig-5]	**air-fluid levels, bowel dilation, bowel wall thickening, pneumatosis**
sigmoidoscopy or colonoscopy • The best test to establish the diagnosis of colonic ischaemia and assess severity. May be repeated to follow disease progression or resolution. [Fig-13] [Fig-12] [Fig-11] • Not necessary to perform in the setting of an acute abdomen with planned emergent operative intervention.	**mucosal sloughing or friability; mucosal petechiae; sub-mucosal haemorrhagic nodules, erosions, or ulcerations; sub-mucosal oedema; luminal narrowing; necrosis, gangrene**

Other tests to consider

Test	Result
mesenteric angiography • The definitive test for diagnosing mesenteric ischaemia. Requires both anterior and lateral views. • Occlusive ischaemia demonstrates a proximal defect in the angiogram without distal filling of the mesenteric arcades. • Non-occlusive ischaemia may cause vasoconstriction of all mesenteric arcades.	**proximal defect of a mesenteric vessel or vasoconstriction of all mesenteric arcades**

Test	Result
CT scan with contrast/CT angiogram • CT provides evidence for the extent of bowel compromise from ischaemia.[25] [26] [Fig-6] [Fig-7] • Presence of thumb-printing sign, indicative of mucosal oedema, suggests a worse prognosis. [Fig-8] • CT angiography is replacing conventional angiography as standard practice for diagnosis of acute mesenteric ischaemia; however, mesenteric angiography currently remains the definitive test. [Fig-10] [Fig-9] • Finding is often non-specific and non-diagnostic for colonic ischaemia; colonoscopy is usually required.	**bowel wall thickening, bowel dilation, pneumatosis intestinalis, portal venous gas, occlusion of the mesenteric vasculature, bowel wall thickening with thumb-printing sign suggestive of submucosal oedema or haemorrhage**
barium enema • May be used to aid in the diagnosis of sub-acute or chronic colonic ischaemia. • Largely superseded by CT and colonoscopy. • Do not use in the acute setting because of the risk of perforation.	**thumb-printing, oedema, cobble-stoning, stricture**
mesenteric duplex ultrasound • 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.	**reduced or lack of blood flow through proximal mesenteric vessels**
magnetic resonance angiograph (MRA) • May have a role in making the diagnosis of chronic mesenteric ischaemia. The time required to perform MRI examinations, and the possible need for bowel stimulation with a meal, limit the usefulness of MRI in the diagnosis of acute mesenteric ischaemia. Overall, CT angiography is likely a better examination than MRI for the diagnosis of chronic mesenteric ischaemia because of its capacity for higher resolution in combination with faster scans.	**narrowing or obstruction of mesenteric vasculature; decreased bowel wall enhancement**

Differential diagnosis

Condition	Differentiating signs / symptoms	Differentiating tests
Infectious colitis	• May have similar clinical features.	• Colonoscopy will demonstrate if ischaemia is present. • Stool cultures may reveal causative organism. • CT may show marked thickening of colon with *Clostridium difficile* .

Condition	Differentiating signs / symptoms	Differentiating tests
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 ischaemic bowel disease. 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.	• 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 discomfort. Nausea and vomiting are common. Usually symptoms are less acute.	• Oesophagogastroduodenoscopy will demonstrate gastritis and ulcers.
Small bowel obstruction	• Often have a history of previous abdominal surgery. Nausea, vomiting and abdominal distension are the predominant features.	• X-ray studies will show air-fluid levels and dilated small bowel. CT will show dilated proximal small bowel with distal decompression, with a possible transition point.

Condition	Differentiating signs / symptoms	Differentiating tests
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.

Step-by-step treatment approach

Treatment for ischaemic bowel disease depends on the anatomical location and severity of ischaemia, its underlying pathophysiology and time course. Thorough assessment and prompt, appropriate intervention is essential to alleviate symptoms and improve outcome.[27] Surgical consultation should not be delayed if ischaemia of the bowel is suspected or verified.

Acute mesenteric ischaemia

Adequate fluid resuscitation and supplemental oxygen should be administered to optimise tissue perfusion and oxygenation. Inotropic support may be required, in which case agents such as low-dose dopamine, dobutamine, or milrinone are preferred, as these agents carry less risk of exacerbating visceral ischaemia. Initial resuscitation should also aim to relieve any acute heart failure and correct any cardiac arrhythmias. Invasive monitoring may be appropriate. NPO status should be enforced, with nasogastric tube decompression for symptomatic relief.

Empiric antibiotics suitable for enteric coverage (e.g., a third-generation cephalosporin or quinolone plus metronidazole) are administered to all patients, as ischaemia can lead to significant bacterial translocation due to damage to the normal intestinal mucosal barrier.

If there are clinical signs of peritonitis, or radiographic or laboratory evidence suggestive of infarction or perforation, exploratory laparotomy or laparoscopy must proceed urgently and include resection of non-viable intestine. Ideally, re-vascularisation procedures should be completed prior to any bowel resection, as borderline ischaemic bowel may recover satisfactorily after re-vascularisation. Second-look operations may be necessary to evaluate progression of ischaemia or re-perfusion injury that may result in more 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, a number of interventions may be appropriate:

- Proximal embolisation or thrombosis may be amenable to re-vascularisation with embolectomy or thrombectomy at the level of arterial occlusion.
- If arterial occlusion is due to severe and widespread atherosclerotic disease and the patient is stable enough to tolerate increased operative duration, systemic-mesenteric bypass is recommended.
- Distal mesenteric vasculature spasm or non-occlusive mesenteric ischaemia may be treated with trans-catheter papaverine infusion.
- If mesenteric venous thrombosis is identified, venous thrombectomy and/or anticoagulation postoperatively may be appropriate.
- If a source of sepsis is identified, appropriate swabs and cultures allow identification of causative organisms and subsequent targeting of antibiotic therapy.
- If vasculitis is identified as a contributory cause (e.g., by thickened blood vessels on CT scan, or the presence of other vasculitic symptoms, or a previous diagnosis) postoperative corticosteroid therapy may be considered.

Papaverine infusions may be used as an adjunct to surgery in cases of acute mesenteric ischaemia, both in cases arising from obstruction by thrombosis or embolus, and in cases of non-obstructive mesenteric

ischaemia. A papaverine infusion is administered before, during, and after surgery until there is no more angiographic or clinical evidence of persistent vasoconstriction.

Heparinisation is also 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 intra-luminal bleeding from damaged bowel, while others advocate immediate heparinisation. Another suggested approach has been immediate anticoagulation if no infarction is present at surgery, but delayed anticoagulation if intestinal infarction was found. Good data supporting any of these approaches are lacking.[1] [20]

In patients with a superior mesenteric artery (SMA) embolus where there is no evidence of infarction, perforation or peritonitis requiring urgent surgical intervention, consideration can be given to local thrombolytic therapy if there are no other contraindications. This is usually administered in conjunction with a papaverine infusion. If lysis of the embolus cannot be demonstrated within 4 hours, or there is evidence of ischaemia progression, patients should then undergo exploratory laparotomy with a view to conventional surgical embolectomy.

Patients with an SMA thrombosis and no evidence of infarction, perforation, or peritonitis, may be treated with percutaneous angioplasty and stenting, although this is a technically difficult procedure. Heparinisation and a papaverine infusion are maintained before, during and after this procedure until there is no evidence of persistent vasoconstriction.

Chronic mesenteric ischaemia

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

Endovascular treatment should be considered, especially in those with severe malnutrition, as it is associated with less morbidity and mortality.[28] 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 SMA, and trans-arterial and trans-aortic mesenteric endarterectomy may all have a role.[1] Open surgery is better for long-term patency when compared with endovascular approaches for chronic mesenteric ischaemia.[28] [29]

In patients unable to undergo an open procedure, percutaneous trans-luminal mesenteric angioplasty (PTMA) alone or with stent insertion may be an option.[30]

Colonic ischaemia

This is the most common form of intestinal ischaemia and comprises a spectrum of disorders covering:[31]

- Reversible colonopathy
- Transient colonic ischaemia
- Chronic colonic ischaemia
- Stricture
- Gangrene
- Fulminant universal colitis.

Most patients with colonic ischaemia do not have any identifiable, specific, and precipitating cause,[1] and treatment varies with severity of presentation. Most cases resolve spontaneously (reversible ischaemic colonopathy or transient colonic ischaemic changes). Severe or continuing symptoms necessitate hospitalisation, supportive measures, bowel rest, and investigation and correction of precipitants.

Patients with moderate or severe acute presentations of colonic ischaemia routinely receive antibiotic therapy, although good evidence of benefit is lacking. The practice is based on a number of old studies and the theoretical protection it gives against the bacterial translocation that occurs with loss of mucosal integrity.[1]

Indications for surgery in colonic ischaemia include:[1]

- Acute indications:
    - Peritoneal signs, suggestive of necrosis or perforation
    - Massive bleeding (rare): may require sub-total colectomy
    - Universal fulminant colitis with or without toxic megacolon.
- Sub-acute 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 with recurrent bouts of sepsis.
- Chronic indications:
    - Symptomatic colon stricture: may be treated with trans-endoscopic dilation or segmental resection
    - Symptomatic segmental ischaemic colitis.

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

Patients with acute transient or mild ischaemia have physical findings without peritonitis, a CT scan or mesenteric angiography demonstrating perfusion to the intestines, and no evidence for full thickness necrosis. If colonoscopy and imaging suggest only mucosal to sub-mucosal involvement, conservative measures may be employed. These include:

- NPO status
- Fluid resuscitation and possible inotropic support
- Antibiotics for enteric coverage
- Nasogastric tube decompression for symptomatic relief.

Antibiotics suitable for enteric coverage (such as a third-generation cephalosporin or quinolone plus metronidazole) should be given to all patients, as bacterial translocation may be significant due to the loss of the normal intestinal mucosal barrier.

The underlying cause should be treated promptly:

- Anticoagulation for mesenteric venous thrombosis.
- Tailored antibiotic therapy when an infectious cause is identified.

- Corticosteroids for vasculitis.
- Fluid resuscitation and cardiac optimisation for shock.

Diligent and frequent re-assessment of the patient must be undertaken to detect those patients for whom conservative management fails and who then require operative intervention due to evidence of peritonitis or infarction.

[VIDEO: Female urethral catheterisation animated demonstration]

[VIDEO: Male urethral catheterisation animated demonstration]

[VIDEO: Central venous catheter insertion animated demonstration]

[VIDEO: Peripheral venous cannulation animated demonstration]

Treatment details overview

Consult your local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing. (see Disclaimer)

Acute (summary)

Patient group	Tx line	Treatment
evidence of infarction, perforation, or peritonitis	**1st**	**resuscitation and supportive measures**
	plus	**empiric antibiotics**
	plus	**exploratory laparotomy or laparoscopy**
■ **superior mesenteric artery (SMA) embolus**	**plus**	**papaverine infusion + embolectomy or arterial bypass ± bowel resection**
■ **acute superior mesenteric artery (SMA) thrombosis**	**plus**	**papaverine infusion + arterial reconstruction or bypass ± bowel resection**
■ **acute superior mesenteric artery (SMA) thrombosis**	**plus**	**postoperative heparinisation**
■ **non-occlusive mesenteric ischaemia**	**plus**	**papaverine infusion ± bowel resection**
■ **mesenteric vein thrombosis**	**plus**	**venous thrombectomy ± bowel resection**
■ **mesenteric vein thrombosis**	**plus**	**anticoagulation**
■ **mesenteric vein thrombosis**	**adjunct**	**papaverine infusion**
■ **fulminant ischaemic colitis**	**plus**	**sub-total or total colectomy**

Acute (summary)

no evidence of infarction, perforation or peritonitis	1st	supportive measures
	plus	empiric antibiotics
■ superior mesenteric artery (SMA) embolus	plus	papaverine infusion ± thrombolysis
■ superior mesenteric artery (SMA) thrombosis	plus	heparinisation
■ superior mesenteric artery (SMA) thrombosis	plus	papaverine infusion + re-vascularisation by percutaneous trans-luminal mesenteric angioplasty (PTMA)
■ non-occlusive mesenteric ischaemia	plus	papaverine infusion + observation
■ mesenteric vein thrombosis	plus	anticoagulation + observation
■ vasculitis associated mesenteric ischaemia	plus	corticosteroid therapy

Ongoing (summary)

Patient group	Tx line	Treatment
■ surgical candidate	1st	medical optimisation + surgical systemic-mesenteric bypass
■ non-surgical candidate	1st	medical optimisation + percutaneous angioplasty and stenting
■ 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 options

Acute

Patient group	Tx line	Treatment
evidence of infarction, perforation, or peritonitis	**1st**	**resuscitation and supportive measures** » Initial measures include supplemental oxygen via a mask, correction of hypotension with fluids and inotropic support if required, assigning NPO status, inserting a nasogastric tube for decompression, and correction of any heart arrhythmias. » Monitoring should be appropriate for the clinical condition of the patient, which may include invasive monitoring.
	plus	**empiric antibiotics** » Antibiotics suitable for enteric coverage (e.g., third-generation cephalosporin or quinolone plus metronidazole) should be given to all patients, as bacterial translocation may be significant due to the loss of the normal intestinal mucosal barrier. **Primary options** » ceftriaxone: 1 g intravenously every 24 hours **-or-** » levofloxacin: 500 mg intravenously every 24 hours **--AND--** » metronidazole: 500 mg intravenously every 8 hours
	plus	**exploratory laparotomy or laparoscopy** » The presence of infarction, perforation, or peritonitis warrants urgent exploratory laparotomy or laparoscopy. The exact nature of the subsequent procedures will depend on preoperative investigations and intraoperative findings.
■ **superior mesenteric artery (SMA) embolus**	**plus**	**papaverine infusion + embolectomy or arterial bypass ± bowel resection** » Responsible for approximately 50% of acute mesenteric ischaemia events. Emboli typically originate from the heart and lodge at points of normal anatomic tapering, usually just distal to the origin of a major branch.

Acute

Patient group	Tx line	Treatment
		» If not amenable to embolectomy, arterial bypass may be required. An embolus in the SMA causes severe vasoconstriction of both the obstructed and unobstructed branches of the SMA. If not corrected promptly this vasoconstriction can become irreversible and persist following removal of the embolus. » Consequently, a papaverine infusion is used preoperatively, intra-operatively, and postoperatively until there is no clinical or radiographic evidence of continuing vasoconstriction. **Primary options** » papaverine: 30-60 mg/hour as an intra-arterial infusion for 24 hours followed by angiographic evaluation; infusion may be re-started for additional 24-hour periods followed by repeat angiograms until resolution of vasoconstriction and clinical signs and symptoms; infusions have been maintained for as long as 5 days
acute superior mesenteric artery (SMA) thrombosis	**plus**	**papaverine infusion + arterial reconstruction or bypass ± bowel resection** » 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 superior mesenteric artery, and trans-arterial and trans-aortic mesenteric endarterectomy.[1] » Papaverine infusions are continued before, during and after surgery until there is no more angiographic or clinical evidence of persistent vasoconstriction. **Primary options** » papaverine: 30-60 mg/hour as an intra-arterial infusion for 24 hours followed by angiographic evaluation; infusion may be re-started for additional 24-hour periods followed by repeat angiograms until resolution of vasoconstriction and clinical signs and symptoms; infusions have been maintained for as long as 5 days

Acute

Patient group	Tx line	Treatment
acute superior mesenteric artery (SMA) thrombosis	**plus**	**postoperative heparinisation** » Timing of postoperative heparinisation is controversial, although it is generally recognised as being beneficial. » Some authorities recommend a delay of 48 hours because of the risk of intra-luminal bleeding from damaged bowel, while others advocate immediate heparinisation. Another suggested approach has been immediate anticoagulation if no infarction was present, but delayed anticoagulation if intestinal infarction was present. Good data supporting any of these approaches are lacking.[1] [20] **Primary options** » heparin: see local protocol for dosing guidelines
non-occlusive mesenteric ischaemia	**plus**	**papaverine infusion ± bowel resection** » Responsible for 20% to 30% of cases of acute mesenteric ischaemia (AMI) and results from mesenteric vasoconstriction following hypo-perfusion of the gut. » Hypo-perfusion may be precipitated by congestive heart failure, cardiac arrhythmia, shock, or by large volume shifts such as occur during haemodialysis. » Papaverine infusion should be continued before, during and after surgery until there is no angiographic or clinical evidence of persistent vasoconstriction. » Bowel of questionable viability should be preserved unless necrosis is clear; borderline viable bowel often responds to papaverine and, by using frequent re-explorations, intestinal resection can be kept to a minimum. **Primary options** » papaverine: 30-60 mg/hour as an intra-arterial infusion for 24 hours followed by angiographic evaluation; infusion may be re-started for additional 24-hour periods followed by repeat angiograms until resolution of vasoconstriction and clinical signs and symptoms; infusions have been maintained for as long as 5 days
mesenteric vein thrombosis	**plus**	**venous thrombectomy ± bowel resection**

Acute

Patient group	Tx line	Treatment
		» The presence of infarction, perforation, or peritonitis mandates exploratory laparotomy. If surgically feasible, venous thrombectomy can be considered. Ideally, thrombectomy should be done prior to resection of any bowel as borderline ischaemic bowel may recover following the procedure.
mesenteric vein thrombosis	**plus**	**anticoagulation** » Whether or not a thrombectomy is undertaken, anticoagulation with heparin should be used routinely following surgery in patients with mesenteric vein thrombosis. Immediate heparinisation for 7 to 10 days has been shown to decrease thrombus recurrence and progression, and to improve survival. In patients who receive heparin the recurrence rate is lowered from 25% to 13% and mortality is reduced from 50% to 13%.[20] Once patients are stable, and able to tolerate oral medicine, they can be converted to warfarin, which should then be administered for 3 to 6 months. **Primary options** » heparin: see local protocol for dosing guidelines **OR** **Primary options** » warfarin: see local protocol for dosing guidelines
mesenteric vein thrombosis	**adjunct**	**papaverine infusion** » Papaverine may be infused into the superior mesenteric artery to relieve any associated arterial spasm that may be contributing to the ischaemic injury. **Primary options** » papaverine: 30-60 mg/hour as an intra-arterial infusion for 24 hours followed by angiographic evaluation; infusion may be re-started for additional 24-hour periods followed by repeat angiograms until resolution of vasoconstriction and clinical signs and symptoms; infusions have been maintained for as long as 5 days
fulminant ischaemic colitis	**plus**	**sub-total or total colectomy** » These patients usually appear toxic and are unresponsive to medical therapy.

Acute

Patient group	Tx line	Treatment
		» Ischaemia and necrosis of the right-side colon can be treated by right hemi-colectomy with primary anastomosis. If there is perforation and peritonitis, resection with terminal ileostomy and a colonic mucocutaneous fistula would be indicated. » Left-sided colonic involvement may require a proximal stoma and distal mucous fistula or Hartmann's procedure. » If most of the colon and rectum are involved in the ischaemia, total colectomy with terminal ileostomy is indicated. » Depending on the findings of the initial surgery, a second-look operation within 12 to 24 hours to re-assess bowel viability may be indicated.
no evidence of infarction, perforation or peritonitis	**1st**	**supportive measures** » General measures should include bowel rest; nasogastric tube decompression; NPO status; intravenous fluids; supplemental oxygen; and correction of hypotension, heart failure and arrhythmias. » Diligent and repeated re-assessment of vital signs, physical examination and laboratory values is required 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).
	plus	**empiric antibiotics** » Patients with colonic ischaemia routinely receive antibiotic therapy suitable for enteric coverage to protect against bacterial translocation, although good evidence of benefit is lacking. The practice is based on a number of old studies and the theoretical protection it gives against the bacterial translocation that occurs with loss of mucosal integrity.[1] **Primary options** » ceftriaxone: 1 g intravenously every 24 hours **-or-**

Acute

Patient group	Tx line	Treatment
		» levofloxacin: 500 mg intravenously every 24 hours **--AND--** » metronidazole: 500 mg intravenously every 8 hours
superior mesenteric artery (SMA) embolus	**plus**	**papaverine infusion ± thrombolysis** » Embolus in the SMA induces profound vasoconstriction of both the obstructed and unobstructed branches of the SMA. Such vasoconstriction can become irreversible and persist if not corrected promptly with a papaverine infusion. » If there are no other contraindications, consideration can be given to concomitant local thrombolytic therapy.[32] [33] The most suitable cases are those where the embolus is a minor one (i.e., distal to the origin of the ileocolic artery), when there is only partial occlusion and when it is given within 12 hours of the onset of symptoms. » If there is no evidence of lysis of the embolus within 4 hours or the patient develops evidence of worsening ischaemia, exploratory laparotomy should be performed with a view to conventional surgical embolectomy. **Primary options** » papaverine: 30-60 mg/hour as an intra-arterial infusion for 24 hours followed by angiographic evaluation; infusion may be re-started for additional 24-hour periods followed by repeat angiograms until resolution of vasoconstriction and clinical signs and symptoms; infusions have been maintained for as long as 5 days
superior mesenteric artery (SMA) thrombosis	**plus**	**heparinisation** » 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 PTT prolongation to 1.5 to 2.5 times normal PTT levels is recommended. **Primary options** » heparin: see local protocol for dosing guidelines

Acute

Patient group	Tx line	Treatment
superior mesenteric artery (SMA) thrombosis	**plus**	**papaverine infusion + re-vascularisation by percutaneous trans-luminal mesenteric angioplasty (PTMA)** » In this circumstance it is reasonable to attempt re-vascularisation by PTMA plus stenting, although it is a technically challenging procedure. » A papaverine infusion is continued before, during and after the procedure until there is no more angiographic or clinical evidence of persistent vasoconstriction. **Primary options** » papaverine: 30-60 mg/hour as an intra-arterial infusion for 24 hours followed by angiographic evaluation; infusion may be re-started for additional 24-hour periods followed by repeat angiograms until resolution of vasoconstriction and clinical signs and symptoms; infusions have been maintained for as long as 5 days
non-occlusive mesenteric ischaemia	**plus**	**papaverine infusion + observation** » Responsible for 20% to 30% of cases of acute mesenteric ischaemia and results from mesenteric vasoconstriction following hypoperfusion of the gut. » Hypo-perfusion may be precipitated by congestive heart failure, cardiac arrhythmia, shock, or by large volume shifts such as occur during haemodialysis. » Papaverine infusion should be continued until there is no angiographic or clinical evidence of persistent vasoconstriction. **Primary options** » papaverine: 30-60 mg/hour as an intra-arterial infusion for 24 hours followed by angiographic evaluation; infusion may be re-started for additional 24-hour periods followed by repeat angiograms until resolution of vasoconstriction and clinical signs and symptoms; infusions have been maintained for as long as 5 days
mesenteric vein thrombosis	**plus**	**anticoagulation + observation** » In patients who have evidence of a mesenteric vein thrombus on CT scan, but do not have signs of infarction, perforation or peritonitis, immediate anticoagulation with heparin and a period of

Acute

Patient group	Tx line	Treatment
		observation is appropriate. These patients need close clinical observation, and surgery is indicated if signs of peritonitis develop. If patients remain stable and symptom free, they may be converted to warfarin for 3 to 6 months. » If a mesenteric vein thrombus is discovered incidentally in an asymptomatic patient who undergoes a CT scan for another reason besides abdominal pain, then a 3- to 6-month course of warfarin is reasonable, especially if a predisposing hyper-coagulable state or concomitant DVT can be identified.[1] **Primary options** » heparin: see local protocol for dosing guidelines **OR** **Primary options** » warfarin: see local protocol for dosing guidelines
vasculitis associated mesenteric ischaemia	**plus**	**corticosteroid therapy** » If vasculitis is identified as a contributory cause of the ischaemia (e.g., by thickened blood vessels on CT scan, the presence of other vasculitic symptoms or a previous diagnosis), postoperative corticosteroid therapy may be considered. **Primary options** » methylprednisolone: 40-500 mg/day intravenously given in 1 to 4 divided doses

Ongoing

Patient group	Tx line	Treatment
surgical candidate	**1st**	**medical optimisation + surgical systemic-mesenteric bypass** » Endovascular treatment should be considered, especially in those with severe malnutrition, as it is associated with less morbidity and mortality.[28] 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.

Ongoing

Patient group	Tx line	Treatment
		» Antegrade and retrograde bypass grafting, aortic re-implantation of the superior mesenteric artery, and trans-arterial and trans-aortic mesenteric endarterectomy may all have a role.[1] Open surgery is better for long-term patency when compared with endovascular approaches for chronic mesenteric ischaemia.[28] [29]
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 trans-luminal mesenteric angioplasty (PTMA) alone or with stent insertion may be an option.[30]
segmental colitis symptomatic for >2 weeks, or protein-losing colonopathy	**1st**	**segmental colectomy** » Patients who have an acute episode of colonic ischaemia which 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.[1]
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.[1]
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. Trans-endoscopic dilation may be successful in less severe cases. Alternatively, segmental resection can be used.[1]

Emerging

Thrombolysis for superior mesenteric vein (SMV) thrombosis

Small numbers of cases have been reported of successful venous thrombolysis with streptokinase, urokinase, and tissue plasminogen activator.[34] [35] [36] However, currently, thrombolysis for SMV thrombosis should be considered experimental.

Recommendations

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.

Patient instructions

Emphasise the importance of regular medical follow-up and adherence to medicine, 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.

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 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 stricturoplasty or more commonly bowel resection is the mainstay of treatment, as strictures are normally of a length that precludes endoscopic balloon dilatation.		
short gut syndrome	**variable**	**medium**
Develops in patients who have undergone significant bowel resection. Management requires permanent intravenous hyper-alimentation or small bowel transplantation.		

FOLLOW UP

Prognosis

Colonic ischaemia

Colonic ischaemia carries the most favourable prognosis of the ischaemic bowel diseases; nevertheless, 20% will develop chronic ulcerating ischaemic colitis.[17]

Acute mesenteric ischaemia

Despite advances in knowledge in diagnosis, pathophysiology and treatment, the outlook for patients with acute mesenteric ischaemia remains poor. Acute mesenteric ischaemia results in mortality rates of between 60% and 100% in several large series.[7] [9] [23] [37] Non-occlusive mesenteric ischaemia results in 70% to 80% mortality.[37] [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.

Diagnosis before the occurrence of intestinal infarction is the most important factor in improving survival for patients with acute mesenteric ischaemia.[1] 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.1[B]Evidence

Chronic mesenteric ischaemia

Mortality rates for surgical re-vascularisation tend towards the lower end of a range from 0% to 16%, with success rates of >90%, and recurrence rates generally <10%.[1]

Several long-term studies have shown that patients who survive surgical re-vascularisation have cumulative 5-year survival rates of 81% to 86%.[1]

Diagnostic guidelines

North America

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

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

Summary: Literature review and recommendations of the American College of Gastroenterology Practice Parameter Committee. Comprehensive diagnosis recommendations with a focus on colonic ischaemia.

ACR Appropriateness Criteria: imaging of mesenteric ischemia

Published by: American College of Radiology **Last published:** 2012

Summary: Recommendations for the imaging studies of choice for the variants of mesenteric ischaemia.

Technical review on intestinal ischemia

Published by: American Gastroenterological Association **Last published:** 2000

Summary: Literature review and recommendations prepared for the American Gastroenterological Association Clinical Practice and Practice Economics Committee. Structured around a number of clinical questions.

Medical position statement: guidelines on intestinal ischemia

Published by: American Gastroenterological Association **Last published:** 2000

Summary: Short summary paper, based on the above technical review, outlining main findings and recommendations.

Treatment guidelines

North America

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

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

Summary: Literature review and recommendations of the American College of Gastroenterology Practice Parameter Committee. Comprehensive treatment recommendations with a focus on colonic ischaemia.

Technical review on intestinal ischemia

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Summary: Literature review and recommendations prepared for the American Gastroenterological Association Clinical Practice and Practice Economics Committee. Structured around a number of clinical questions.

North America

Medical position statement: guidelines on intestinal ischemia

Published by: American Gastroenterological Association **Last published:** 2000

Summary: Short summary paper, based on the above technical review, outlining main findings and recommendations.

Evidence scores

1. Increased survival in acute mesenteric ischaemia: there is medium-quality evidence from several retrospective studies that diagnosis within 24 hours of presentation to a physician, or before any significant bowel infarction occurred, resulted in markedly improved survival.[1]
Evidence level B: Randomized controlled trials (RCTs) of <200 participants, methodologically flawed RCTs of >200 participants, methodologically flawed systematic reviews (SRs) or good quality observational (cohort) studies.

Key articles

- Brandt LJ, Boley SJ. AGA technical review on intestinal ischemia. Gastroenterology. 2000;118:954-968. Full text Abstract
- Herbert GS, Steele SR. Acute and chronic mesenteric ischemia. Surg Clin North Am. 2007;87:1115-1134. Abstract
- American Gastroenterological Association Medical Position Statement: guidelines on intestinal ischemia. Gastroenterology. 2000;118:951-953. [Erratum in: Gastroenterology. 2000;119:280-281.] Full text Abstract

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Images

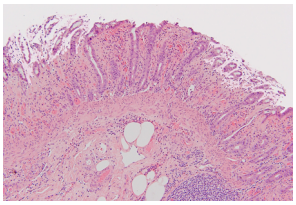

Figure 1: Histopathology of intestinal ischaemia

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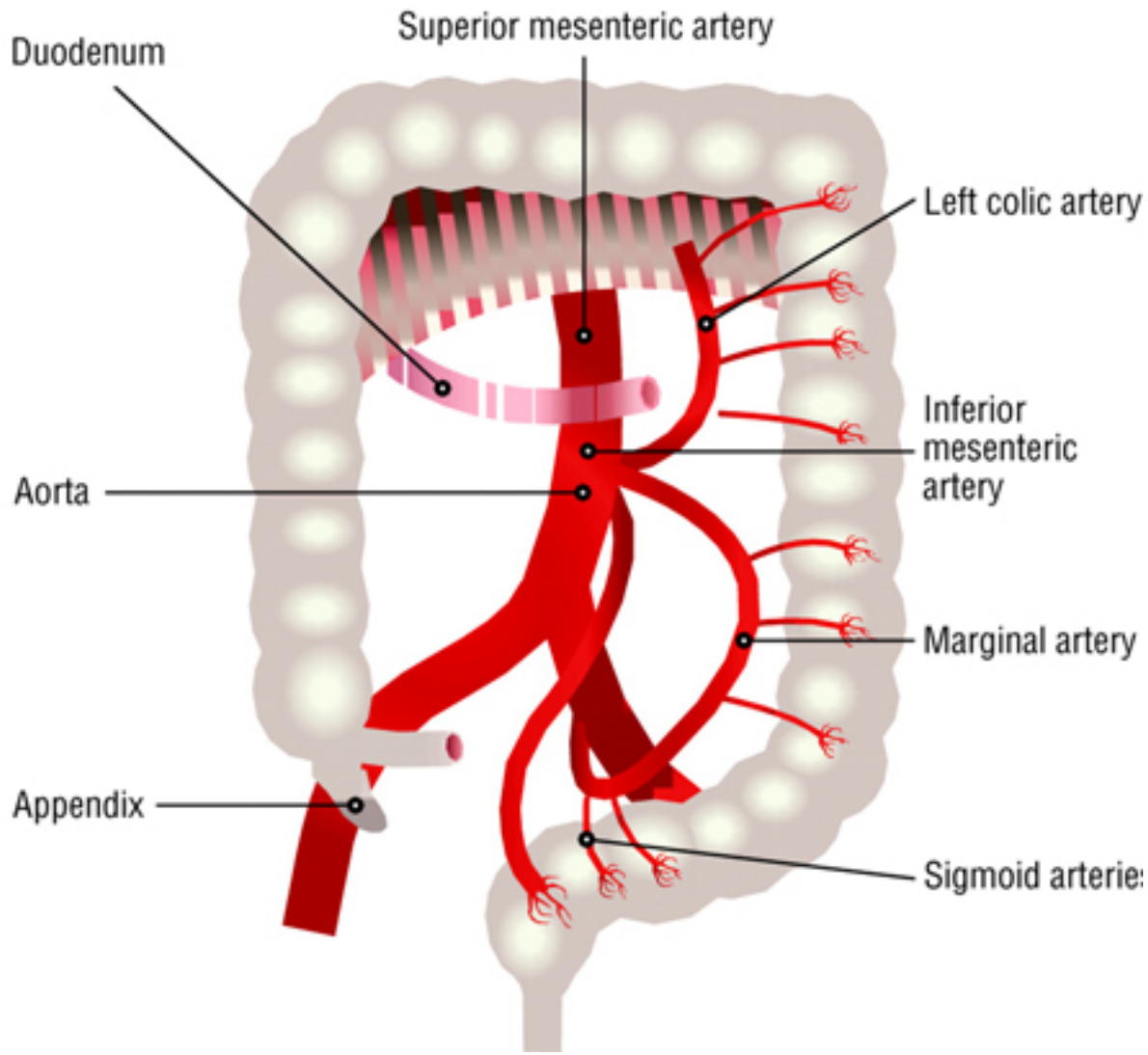

Figure 2: Distribution of blood flow to the colon originating from the inferior mesenteric artery, branches of which include the left colic, marginal, and sigmoid arteries and supply the left colon and superior portion of the rectum

BMJ 2003; 326 doi: 10.1136/bmj.326.7403.1372

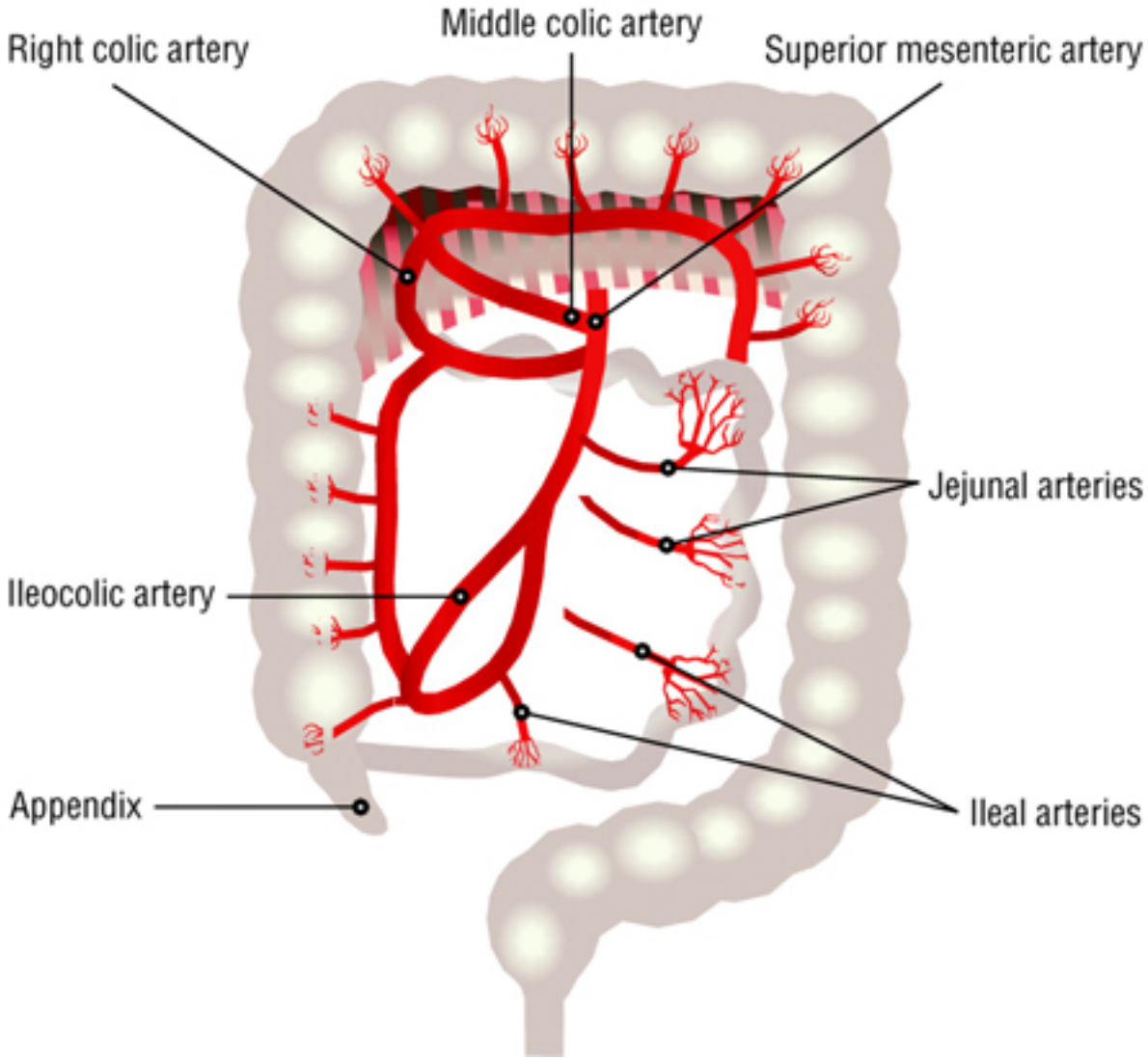

Figure 3: Distribution of blood supply to the small intestine and colon from the superior mesenteric artery, branches of which include the middle, right, and ileocolic arteries as well as jejunal and ileal arteries and arterioles

BMJ 2003; 326 doi: 10.1136/bmj.326.7403.1372

	Acute mesenteric ischaemia	Chronic mesenteric ischaemia	Colonic ischaemia
Site	Periumbilical pain. Focal pain if necrosis present.	Poorly localised.	Lateral abdomen or flanks. Focal pain if necrosis present.
Onset	Sudden.	Insidious.	Sudden. May become continuous and diffuse if it progresses.
Character	Sharp or colicky. Pain is out of proportion to the exam.	Repeated, transient episodes of pain, progressing with time.	Dull.
Radiation	No radiation.	No radiation.	Radiates to back.
Associations	Nausea, vomiting, diarrhoea. May have sudden forceful bloody bowel evacuation.	Nausea, vomiting.	Nausea, vomiting, diarrhoea. Passage of maroon stools.
Timing, duration, frequency	2-3 hours (arterial) or 5 to more than 30 days (venous).	Months.	Acute, subacute, or chronic.
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	Epigastric bruit and distention.		Abdominal distention and no bowel sounds as ischaemia progresses.
Cardiovascular exam	May have a fibrillation or other arrhythmia, evidence of peripheral vascular disease.	Atherosclerosis, peripheral vascular disease.	May have a fibrillation or other arrhythmia, atherosclerosis, evidence of peripheral vascular disease.
Laboratory test results	Leukocytosis, metabolic acidemia, and elevated serum amylase.		
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. Barium enema can be used if colonoscopy is not available (cobblestone appearance, thumbprinting, stricture).
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.

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

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

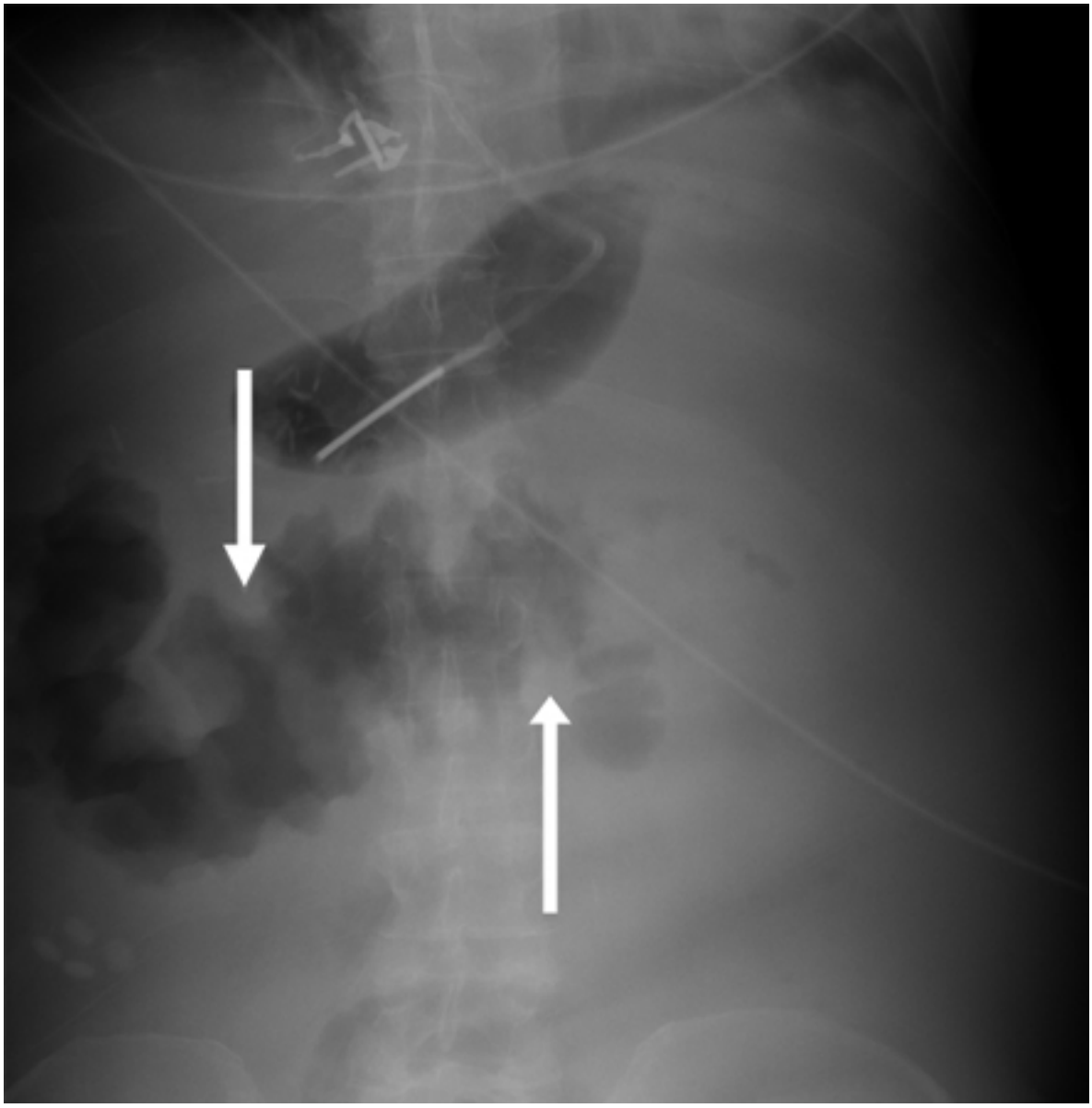

Figure 5: Plain abdominal x-ray: shows marked wall thickening of the transverse colon compatible with the finding of thumbprinting (white arrows)

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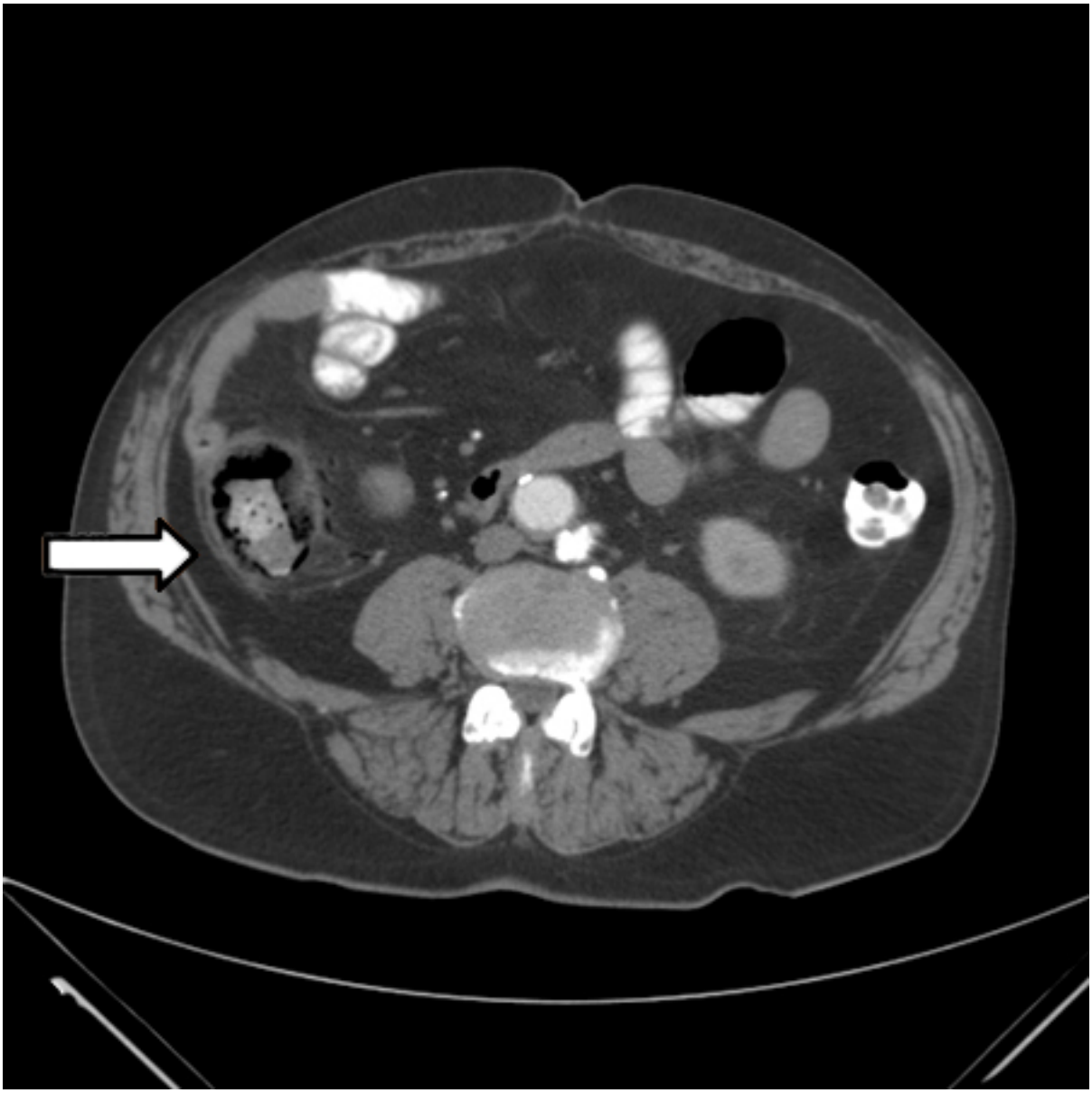

Figure 6: CT scan: colonic thickening with pneumatosis intestinalis

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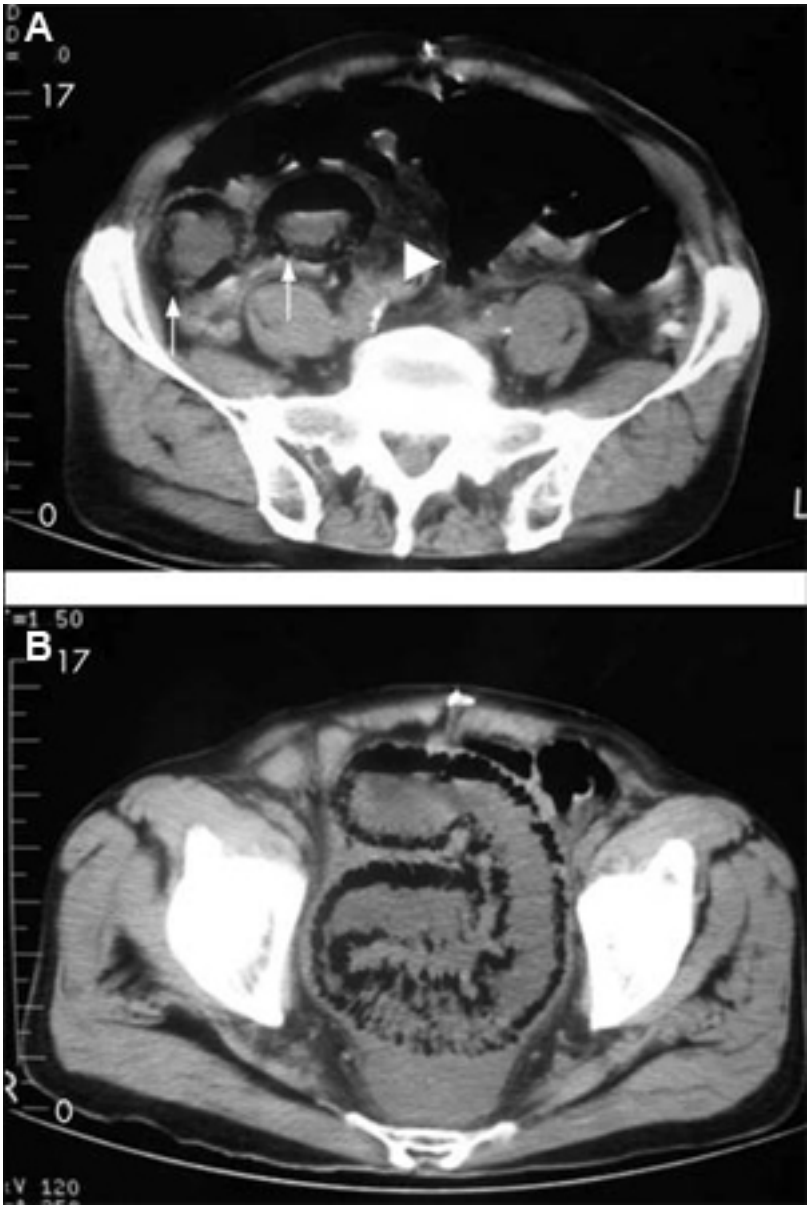

Figure 7: 84-year-old man presenting with symptoms suggestive of ischaemic bowel disease: (A) Abdominal CT revealing a massive circumferential and band-like air formation as intestinal pneumatosis (arrows) and pronounced oedema of mesenteric fat (arrowhead) around necrotic bowel loops; (B) Another slice of abdominal CT showing long segmental pneumatosis of the small bowel

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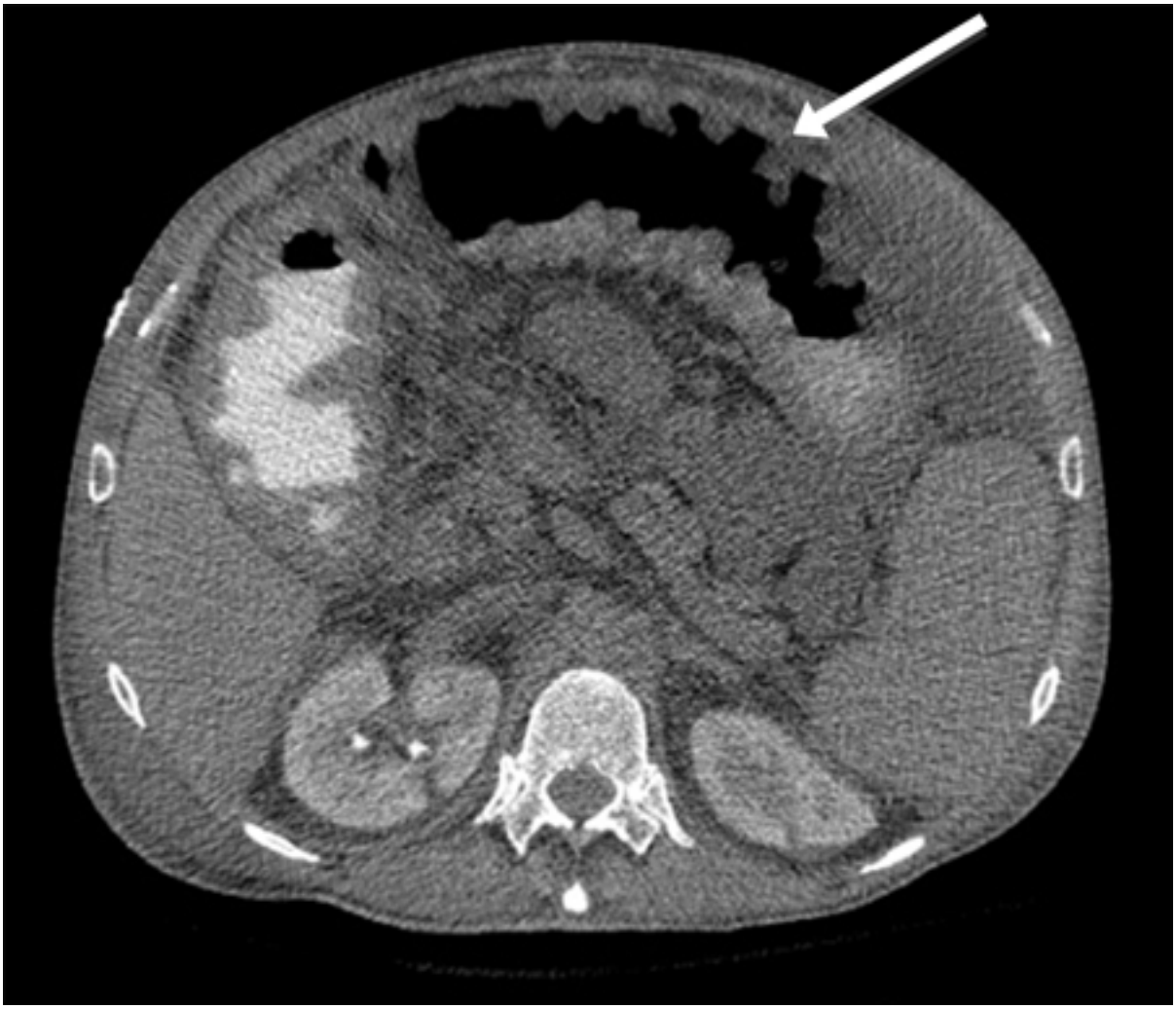

Figure 8: CT scan: circumferential wall thickening of the transverse colon; white arrow shows thumbprinting

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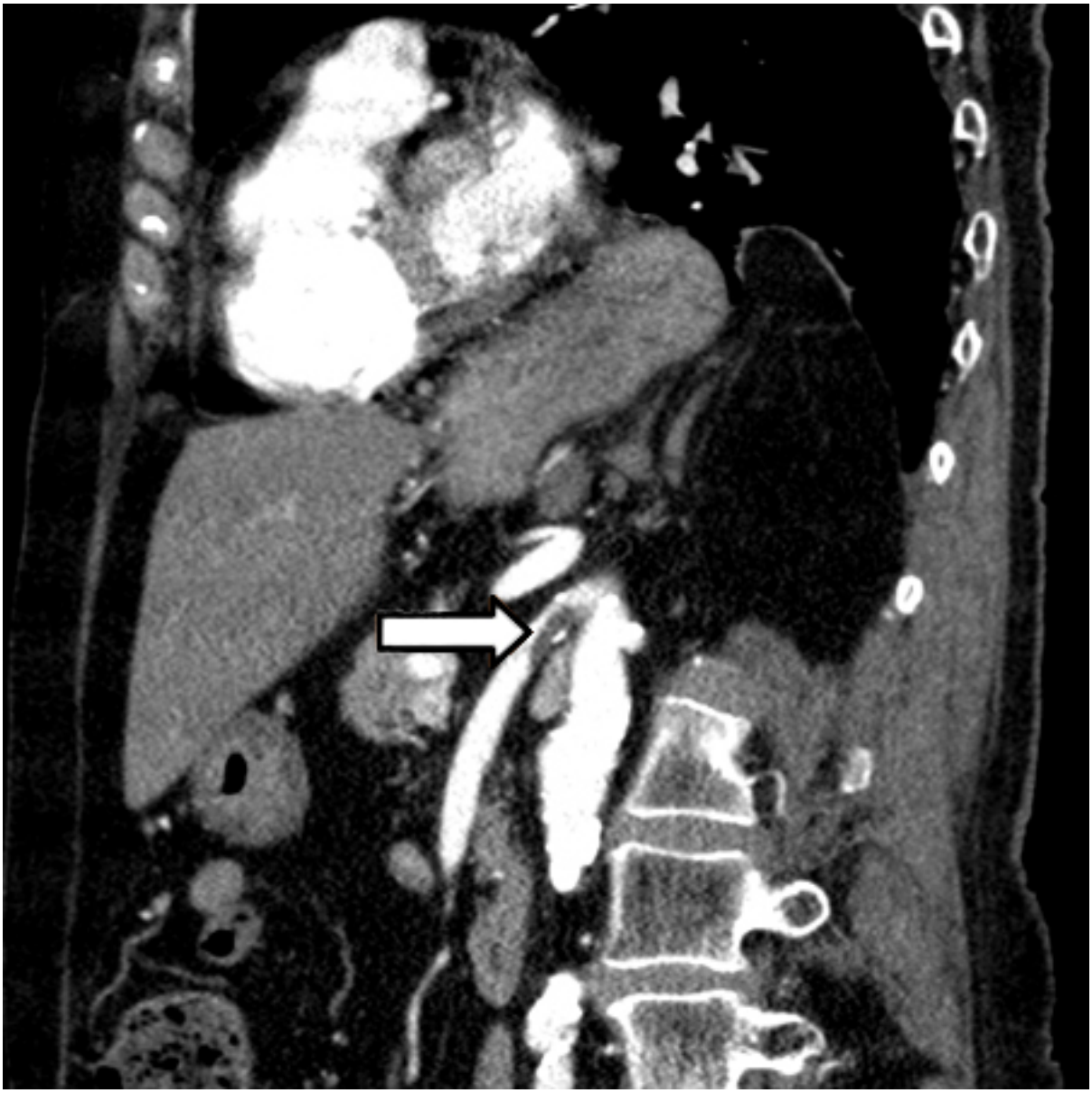

Figure 9: CT angiogram: Acute superior mesenteric artery thrombus

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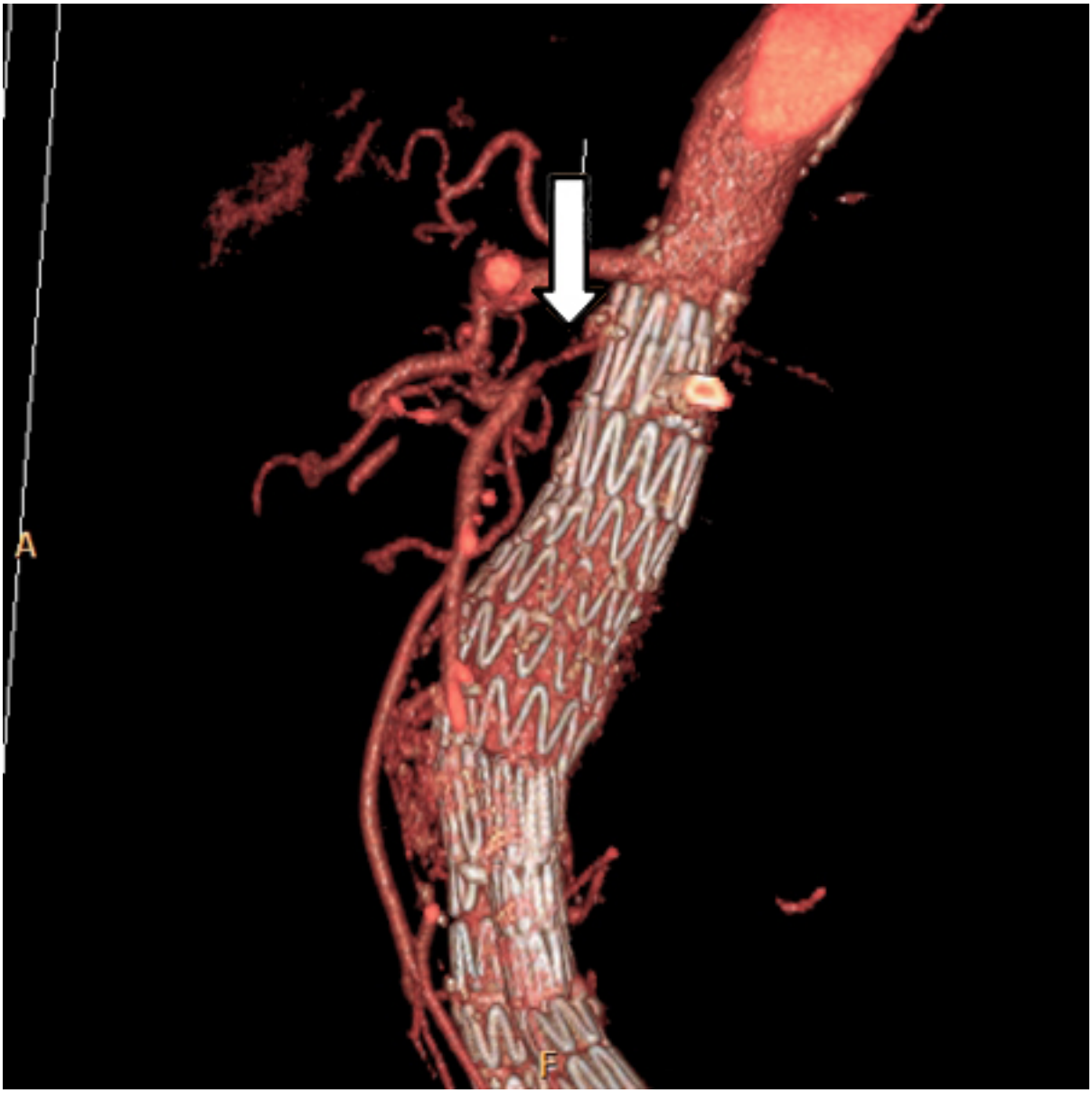

Figure 10: CT angiography: 3-dimensional reconstruction with superior mesenteric artery stenosis from severe atherosclerotic plaque in a patient on follow-up imaging for endovascular aneurysm repair

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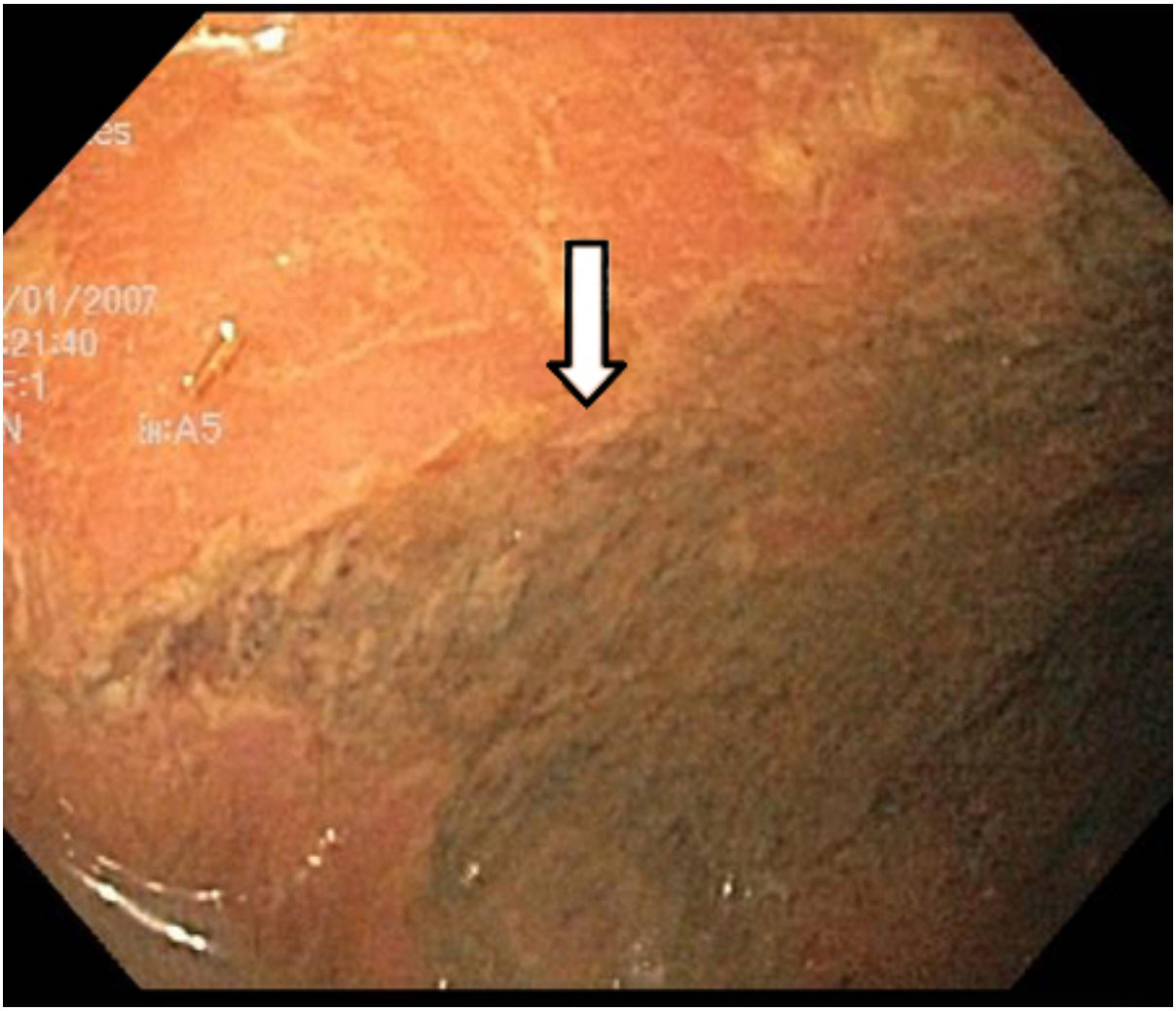

Figure 11: Colonoscopy: demarcation between ischaemic and normal colon

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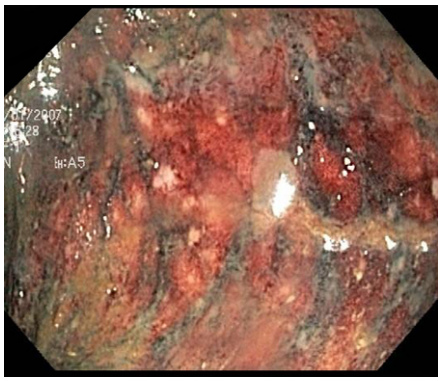

Figure 12: Colonoscopy: denudation of colonic mucosa

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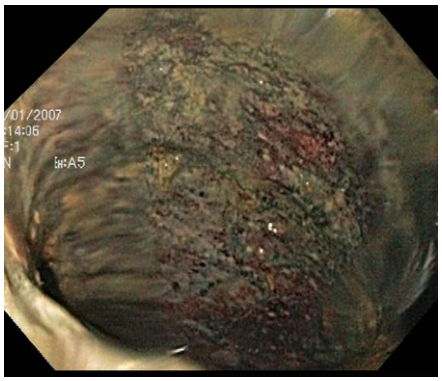

Figure 13: Colonoscopy: mucosal sloughing and likely to be non-viable colon

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