Mesenteric ischaemia

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Abstract

Mesenteric ischaemia is an uncommon but potentially life-threatening condition encompassing a range of pathology and symptoms. This article considers the spectrum of acute mesenteric ischaemia, venous infarction, acute colonic ischaemia, chronic mesenteric ischaemia and ischaemic colitis.

Keywords Bowel infarction; bowel ischaemia; colonic ischaemia; ischaemic colitis; mesenteric angina; mesenteric embolus; mesenteric ischaemia

Acute mesenteric ischaemia

Acute mesenteric ischaemia (AMI) is a surgical emergency with published mortality rates varying between 50% and 100%.[1]

Epidemiology

Acute mesenteric ischaemia is a relatively uncommon condition but it has been difficult to establish incidence as AMI is usually diagnosed at autopsy (post-mortem rates vary), or during operative intervention. A recent meta-analysis has suggested that the annual incidence of AMI lies between 0.09% and 0.2% per patient per year.[1] Median age at presentation is approximately 70 years, with an exponential increase in incidence of AMI with age.[2] Correction for age within the population reveals no gender difference.[2]

Aetiology

There are four principal causes of mesenteric ischaemia:

- Embolus into to the coeliac axis or the superior mesenteric artery (SMA; usually due to atrial fibrillation (AF) or mural thrombus following myocardial infarction) (Figure 1)
- Thrombosis (on pre-existing atherosclerosis of the coeliac axis or more commonly SMA) (Figure 2).
- Mesenteric venous thrombosis.
- Non-occlusive mesenteric ischaemia (NOMI) resulting from low output states, vasoconstriction, or both.

Other rare causes include aortic dissection, aneurysmal disease and vasculitis. The most common cause of AMI has previously been reported to be emboli (accounting for approximately 50% of cases) but more recent reports suggest thrombosis may be becoming more common,[3] reflecting the increasing burden of atherosclerosis (and possibly reduction in emboli due to rheumatic valve disease, or better management of AF).[4,5] NOMI accounts for approximately 15% of acute mesenteric ischaemia.[2] NOMI occurs as a result of splanchnic hypoperfusion due to congestive cardiac failure, myocardial infarction, aortic insufficiency and hepatic or renal diseases.[5] Risk factors for NOMI include old age, concurrent atherosclerotic disease of the coeliac axis and SMA, hypotension, dehydration, low cardiac output, alpha-agonists, beta-blockers and digoxin.

Pathology

The main arterial supply to the bowel is based on the embryological development of the gut as follows:

- coeliac axis — to the foregut (distal oesophagus to 2nd part of duodenum)
- superior mesenteric artery — to the midgut (3rd part of duodenum to mid-transverse colon)
- inferior mesenteric artery — to the hindgut (mid-transverse colon to rectum).

The SMA is the vessel most commonly affected by emboli (due to the oblique angle of origin from the aorta compared to the coeliac axis). Five per cent of all peripheral emboli lodge in the SMA. Twenty per cent of patients with SMA emboli have clinical evidence of simultaneous emboli elsewhere, whilst post-mortem studies indicate two thirds of patients have concurrent emboli.[2] At a normal range of blood pressures splanchnic autoregulation preserves mesenteric blood flow, which accounts for 10—20% of resting cardiac output, increasing to 35% following a meal.[6,7] Once blood pressure dips below 70 mmHg there is a linear correlation with mesenteric flow and at pressures less than 40 mmHg the gut becomes ischaemic. After 15 minutes of absolute ischaemia, changes in villi can be seen; after 3 hours the mucosa sloughs and after 6 hours, transmural necrosis occurs (Figure 3), resulting in perforation, sepsis and death. Effects of arterial occlusion may be mitigated by the presence of collaterals, more commonly in thrombosis than emboli, since they have time to develop as a result of the underlying stenotic disease. Thrombotic occlusion tends to occur more proximally than embolic occlusion, and the extent of bowel infarction is more severe.[2] Up to 75% of patients with thrombotic occlusion of the SMA may have previous symptoms of chronic mesenteric ischaemia.[2]

Presentation

There may be considerable variation in the presentation of AMI. Main symptoms and signs include:[8]

- abdominal pain — the predominant symptom in AMI, characterized as 'pain out of proportion to signs'. Pain usually starts as intermittent spasmodic discomfort in the umbilical or epigastrium region before becoming constant
- nausea and/or vomiting
- diarrhoea or, less commonly, constipation
- per-rectal bleeding — usually a late sign indicating significant mucosal injury
- tachycardia with hypotension
- abdominal distention
- peritonitis is a late sign associated with bowel infarction
- bowel sounds may be present or absent.

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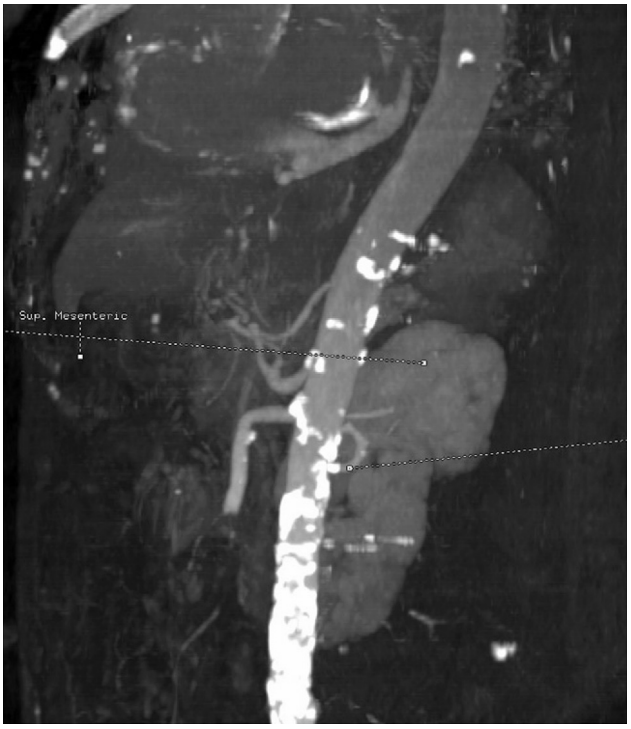

Figure 1 MRA showing the characteristic convex shape of apex of an SMA embolus with distal occlusion.

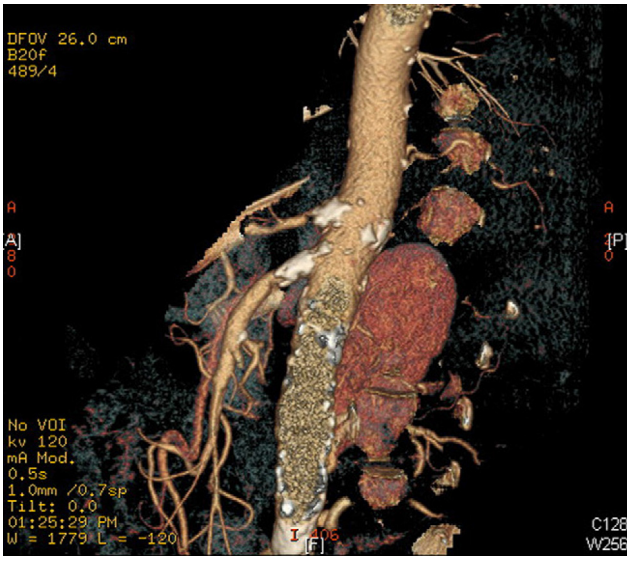

Figure 2 Contrast CTA of aorta with reconstruction, demonstrating marked atherosclerosis at SMA and coeliac axis origins.

Investigations

Diagnosis is guided by clinical suspicion, often correlated with CT findings, as other investigations are likely to yield non-specific abnormalities.

Blood tests: a blood gas sample is likely to be available before any other investigation. This may show an elevated lactate and low pH. Severity of derangement may not correlate with the extent of injured bowel but lactate is a predictor of mortality.[9]

Raised white cell count (e.g. >20,000) is common but a normal white cell count does not exclude the diagnosis. It is a poor prognostic factor, as are elevated serum enzymes (ALP, AST and LDH), phosphate, D-dimers and lipase.[1,10]

Radiology: findings on plain abdominal X-ray are often non-specific and may even be normal. The film may reveal bowel wall oedema with distended loops of small bowel, and possible pneumatosis intestinalis or portal venous gas.

Computed tomography angiography (CTA) is advocated as the first-line imaging modality in assessment of both AMI and chronic mesenteric ischaemia. CTA is non-invasive, readily available and less operator dependent than other imaging modalities. CTA was reported to have high sensitivity (93%) and specificity (96%) in a recent meta-analysis.[11] The diagnosis is based on a combination of the following signs:[8,12]

- filling defect in coeliac axis or SMA
- mesenteric venous filling defect/venous engorgement
- portal vein gas
- differential mural enhancement/mural thickening
- pneumointestinalis (gas in bowel wall)
- solid organ infarction
- mesenteric fat stranding.

Management

Emergency resuscitation: mesenteric ischaemia is associated with significant mortality and patients often have a poor physiological reserve with significant co-morbidities. Initial management should comprise administration of oxygen and rapid infusion of intravenous fluids. Prophylactic intravenous antibiotics should be given. A loading dose of 5000 units of unfractionated heparin (UFH) followed by maintenance infusion of UFH should be commenced, unless contraindicated. A nasogastric tube should be inserted to facilitate bowel rest. Patients require invasive monitoring of haemodynamic parameters by arterial line and a central venous catheter. A urinary catheter should be inserted to measure hourly urine output.

Surgical management: if a surgical approach is decided upon then the patient should be transferred promptly to the operating theatre. Laparotomy is performed through a midline incision. If bowel infarction is confirmed, a decision should be made about the necessary extent of bowel resection (and whether this is compatible with life), and the possibility of revascularization.

Frankly ischaemic bowel appears grey or black and is foul-smelling. More subtle changes include duskiness and loss of peristalsis. If bowel appears salvageable it may be possible to consider revascularization. This should be performed before bowel resection.

SMA embolectomy involves transverse arteriotomy across the SMA and passage of a 3—4 Fr Fogarty catheter to remove the thrombus. The arteriotomy is then closed and SMA flow is assessed by intraoperative Doppler. If the SMA has significant atherosclerotic disease then a mesenteric bypass may be considered from infra-renal aorta or iliac artery, to a suitable section of the SMA, using reversed vein.[13]

