Small bowel ischaemia

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Abstract

Ischaemia in the small bowel can present as an acute surgical emergency or more insidiously with relatively non-specific symptoms. Acute small bowel ischaemia is associated with a mortality rate of 60–80%. While other more common causes of abdominal pain should be excluded, a high index of clinical suspicion is needed. Cross-sectioning imaging with CT scanning is sensitive and specific for making the diagnosis. Prompt intervention (revascularization) is needed to restore mesenteric blood flow in order to prevent bowel necrosis and sepsis. Non-viable small bowel needs surgical resection, and subsequent re-anastomosis is possible in some patients. Chronic small bowel ischaemia presents as reproducible pain on eating, with weight loss, and should be considered in patients with vascular risk factors; it can be diagnosed by CT angiography. Angioplasty and stenting give good short-term outcomes, but surgical bypass is still associated with more durable results, even though it has higher operative morbidity and mortality.

Keywords Mesenteric ischaemia; MRCP; small bowel ischaemia

Key points

- Small bowel ischaemia can be acute or chronic
- Acute small bowel ischaemia is a surgical emergency associated with 60–80% mortality
- Chronic small bowel ischaemia is typified by reproducible postprandial abdominal pain and weight loss
- In acute small bowel ischaemia, prompt intervention is needed to limit necrosis and small bowel loss
- In chronic small bowel ischaemia angioplasty, endarterectomy and bypass are treatment options

Introduction

Mesenteric ischaemia is a group of disorders characterized by reduced blood supply to the small bowel.[1]

Acute mesenteric ischaemia (AMI) is defined as the abrupt interruption of mesenteric blood flow whose onset varies from minutes to hours. The most characteristic symptom is acute onset of severe abdominal pain that can develop, if left untreated, into necrosis of the gastrointestinal tract, perforation and subsequent peritonitis. In contrast, chronic mesenteric ischaemia (CMI) is defined as insufficient mesenteric blood flow for at least 3 months. The most common symptoms are postprandial abdominal pain, weight loss and diarrhoea.

Although mesenteric ischaemia is a rare cause of abdominal pain, it is an important differential diagnosis that needs to be appropriately investigated and managed.

Causes of mesenteric ischaemia

Causes of mesenteric ischaemia can be divided into: (1) obstruction of veins and/or arteries, and (2) vasoconstriction of structurally normal vessels, so-called non-occlusive mesenteric ischaemia (NOMI) (Figure 1).

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Arterial obstruction

The major causes of arterial obstruction are thrombosis, atherosclerotic disease, dissection, extrinsic compression and embolism. The predominant source of arterial embolism is the heart in the context of atrial fibrillation. Although rare, mesenteric vasculitis can also lead to ischaemia. Extrinsic compression of the mesenteric vasculature can occur as a result of local progression of cancer, particularly with pancreatic carcinoma. In addition, compression of the coeliac axis as a result of median arcuate ligament syndrome, a type of fibromuscular dysplasia, can result in mesenteric ischaemia.

True mesenteric arterial aneurysms are rare but can become symptomatic by thrombosis, embolism or rupture. Pseudoaneurysm can result from any injury to the arterial wall, such as atherosclerotic ulceration, or from localized inflammation in an adjacent structure (e.g. pancreatitis).

Venous obstruction

Venous mesenteric ischaemia is most commonly the result of venous thrombosis, the causes of which include, but are not limited to, intra-abdominal malignancies, inflammatory disorders, thrombophilic states and myeloproliferative disorders. Isolated thrombosis in intra-abdominal veins can occur, such as Budd–Chiari syndrome, but is rare. NOMI is the end-stage result of circulatory failure in the context of all states of shock, and in critically ill patients where blood flow to the brain, heart and kidneys is maintained at the expenses of the mesenteric circulation.

Acute ischaemia

The reported annual incidence of AMI is 7.3 per 100,000. The classical presentation is acute-onset diffuse abdominal pain with a relative paucity of clinical signs. Peritonism and circulatory compromise are late signs signifying perforation. Although not confirmatory, raised white cell count, inflammatory markers and lactate concentration provide support for small bowel ischaemia, particularly in a patient with identifiable risk factors such as atrial fibrillation. D-Dimer can be used as a highly sensitive early marker, but it has low specificity.

Cross-sectional imaging with computed tomography (CT) angiography is the initial investigation of choice, with excellent

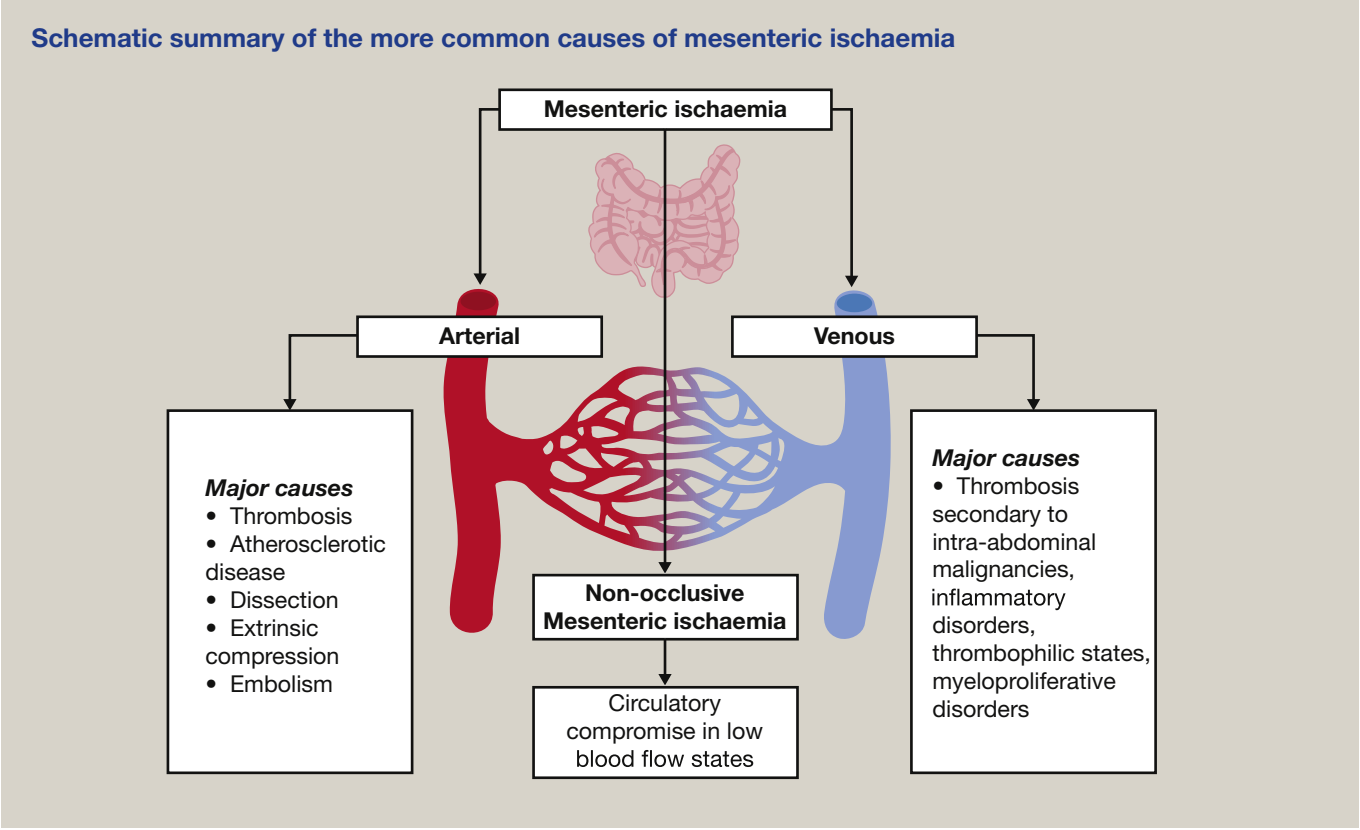

Figure 1

specificity and clinically acceptable sensitivity.[2] However, a diagnostic laparotomy is often required to establish a definitive diagnosis, with formal resection of all non-viable bowel. Revascularization should be performed before bowel surgery. Primary anastomosis is normally delayed, with an early second-look procedure that allows further assessment of viability of the remaining bowel and possible re-anastomosis.[3] Postoperatively, patients with embolic disease should be anticoagulated, and those with atherosclerotic disease should be on best medical therapy including an antiplatelet agent and statin. A high-output stoma, short bowel syndrome and intestinal failure are possible outcomes.

The management of mesenteric venous thrombosis is very challenging. Anticoagulation is the mainstay of treat for patients without features of peritonitis, with laparotomy and bowel resection for those with signs of complications. Thrombectomy is associated with suboptimal outcomes, although the highly selective use of thrombolysis has an emerging role. An underlying thrombophilia is often identifiable in these patients. In those with

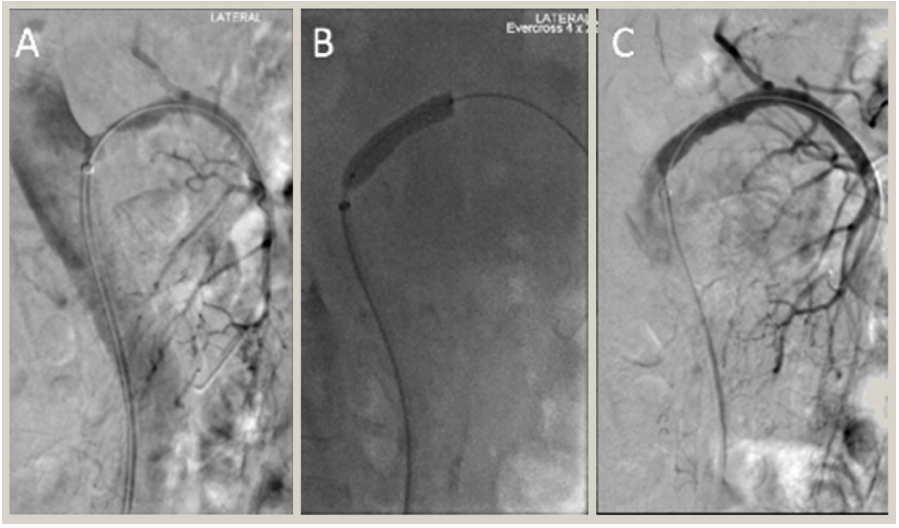

Figure 2 Superior mesenteric artery stenosis. (**a**) Lateral view of the aorta, showing significant superior mesenteric artery (SMA) stenosis. (**b**) Angioplasty balloon and stent inflated. (**c**) After angioplasty and stenting of the SMA.

NOMI, cardiovascular resuscitation needs to be undertaken before proceeding to surgical intervention. Despite improvements in diagnosis, intervention and supportive care, outcomes remain poor, with a reported mortality of up to 75%.[4]

Chronic mesenteric ischaemia

Owing to the extensive nature of the collateral supply, stenosis or frank occlusion of the mesenteric vessels results in symptoms in only a small proportion of patients. Given the broad nature of the differential diagnosis of abdominal pain and weight loss, CMI is frequently overlooked as a differential diagnosis. CMI should be considered as a possible diagnosis in arteriopathic patients with associated risk factors, such as smoking, and in those with established vascular disease. The prototypical symptom is diffuse and reproducible postprandial pain, which can lead to a fear of food and weight loss. At the end stage of the disease, patients can also develop nausea, vomiting and diarrhoea.

Given the non-specific nature of symptoms, patients usually undergo a standard work-up with transabdominal ultrasound, cross-sectional imaging and gastrointestinal endoscopy. In experienced hands, duplex ultrasonography can accurately identify superior mesenteric artery stenosis, although CT angiography is required for a definitive diagnosis.

The goals of interventional mesenteric revascularization are: (1) reducing symptoms, (2) improving nutritional status, and (3) reducing the risk of death by preventing infarction. When considering revascularization, the clinician needs to undertake an individualized assessment balancing potential risks and potential benefits. Surgical mesenteric bypass procedures still have better patency rates than endovascular procedures, but are associated with higher peri-procedural morbidity and mortality. Endovascular intervention with angioplasty and stenting (with covered stents) is therefore attractive, particularly for short focal stenosis or occlusion (Figure 2). However, endovascular treatment often needs to be repeated because of re-stenosis, to maintain patency rates.[5] These patients therefore require long-term follow-up and antiplatelet therapy. ◆

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TEST YOURSELF

To test your knowledge based on the article you have just read, please complete the questions below. The answers can be found at the end of the issue or online here.

Question 1

A 63 year old man presented with a 3 month history of recurrent abdominal pain. He has a background history of ischaemic heart disease and diabetes.

Which of the following clinical features would be supportive of the most likely diagnosis?

A. weight gain
B. history of irritable bowel syndrome
C. constipation
D. Bloating
E. pain after eating

Question 2

A 78-year-old man admitted to the intensive therapy unit after a myocardial infarction complains of severe central abdominal pain.

Which of the following is the most accurate diagnostic study?

A. Plain abdominal radiograph
B. Ultrasonography
C. CT angiography
D. Upper GI endoscopy
E. C reactive protein

Question 3

An 83-year-old man presented with recurrent abdominal pain after meals. On investigation, he was found to have chronic mesenteric ischaemia. He had a 30-year history of productive cough and a 5-year history of wheezy breathlessness. Exercise tolerance was limited to 100 m on the flat by pain in the right calf.

In context of the clinical history what would be the most appropriate treatment for his mesenteric ischaemia?

A. Endovascular angioplasty and stenting
B. Thrombolysis
C. Bypass surgery
D. Anti-platelet therapy
E. Surgical endarterectomy