Intestinal ischaemia

S Shetty, G Morris-Stiff, MH Lewis

Over the past three decades major advances have been made in the diagnosis and management of mesenteric ischaemia. Early identification and treatment of this condition has reduced its mortality and morbidity. This review discusses the causes, preliminary and definitive investigations, and treatment protocols relating to gut ischaemia.

Intestinal ischaemia is a devastating disease process, which without early diagnosis and timely intervention, leads to gangrene and death. Over the last 30 years the mortality in patients with this condition has decreased from around 85% to 60%, mainly as a result of early and aggressive management (Levy et al, 1990). The incidence of intestinal ischaemia has increased during this time, probably as a result of a combination of heightened awareness of the condition, a more elderly population, and the increased number of critically ill patients in hospital. Intestinal ischaemia should be classified as acute, where there are no pre-existing ischaemia gut symptoms, or chronic, when the patient presents with a long illness associated with weight loss, abdominal pain and negative standard investigations. It is important to differentiate between these two types of ischaemia and the causative factors involved, as the treatment is different for the two conditions.

The key to accurate diagnosis and successful management of patients with acute or chronic mesenteric ischaemia includes a detailed history, focusing on the quality and temporal relationship of symptoms, an accurate vascular assessment and physical examination with attention directed at ruling out non-vascular causes for their ill health. The important ischaemia-related factors to be considered include bowel obstruction, neoplasia, embolism, arterial thrombosis, mesenteric venous thrombosis, arteritis, aortic aneurysm surgery and other abdominal inflammatory conditions.

APPLIED ANATOMY OF THE MESENTERIC VESSELS

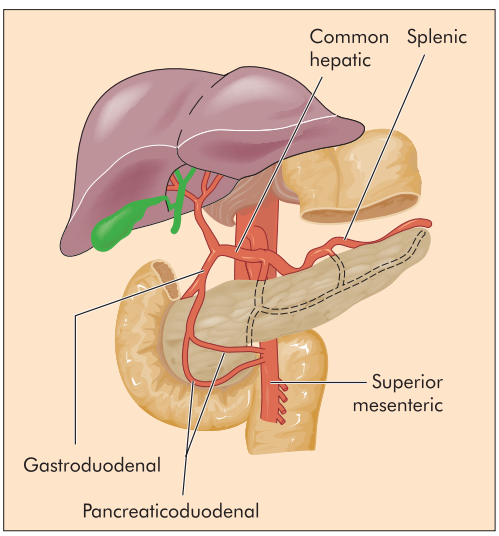
The superior mesenteric artery (SMA) is the principal arterial supply to the jejunum, ileum and colon up to the splenic flexure. Although potential collaterals exist, they may be inadequate to prevent infarction in the supplied area if occlusion occurs acutely. A decrease in arterial flow initially produces a physiological vasodilatory response in order to maintain adequate intestinal perfusion, but if diminished flow is prolonged, reactive vasoconstriction occurs. In patients with non-occlusive mesenteric ischaemia, it is the persistent vasospasm that causes gut ischaemia.

The important channels of collateral circulation between the coeliac and superior mesenteric arteries are the superior and inferior pancreaticoduodenal arteries. The collateral channels between the superior mesenteric and the inferior mesenteric vessels are the vasa recta, the marginal arteries of Drummond and the arc of Riolan (*Figures 1* and *2*).

PATHOPHYSIOLOGICAL RESPONSE TO ACUTE MESENTERIC ISCHAEMIA

The initial response to an acute ischaemic event is a marked increase in bowel activity. This leads to rapid bowel evacuation and an increase in the oxygen demands of the affected intestine. Later, the bowel motility ceases and within hours, the bowel becomes haemorrhagic and oedematous as

Figure 1. Collateral circulation between the coeliac artery and the superior mesenteric artery.



Mr S Shetty is Senior House Officer, **Mr G Morris-Stiff** is Specialist Registrar and **Mr MH Lewis** is Consultant Vascular Surgeon in the Department of General Surgery, Royal Glamorgan Hospital, Llantrisant CF72 8XR

Correspondence to: Mr MH Lewis

capillary integrity is compromised. The invasion of microorganisms into the bowel wall with production of toxic metabolites and utilization of the available oxygen supply further adds to the ischaemic insult. The shift of intravascular volume into the bowel wall leads to severe haemoconcentration and hypovolaemic shock. Significant microvascular and parenchymal wall injury is also caused by reperfusion injury sustained following an ischaemic event. It appears to be mediated by highly reactive oxygen free radicals and activated polymorphonuclear cells, which release proteases and cause physical disruption of the endothelial barrier. The vasoactive mediators and the endotoxins released in the inflammatory process, along with neutrophil-mediated endothelial injury, have been implicated in the aetiology of cardiac depression, septic shock and acute renal failure (Brophy, 1993).

INTESTINAL ISCHAEMIA

Two types of ischaemia present in the clinical setting, acute and chronic intestinal ischaemia. Their presentation, pathological sequelae and treatment are different and therefore are presented separately.

ACUTE INTESTINAL ISCHAEMIA

Based on the aetiology and pathogenesis intestinal ischaemia can be classified as occlusive and non-occlusive.

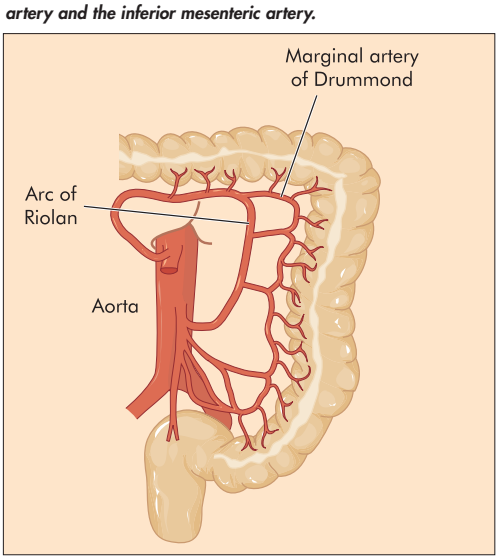
Occlusive intestinal ischaemia

The primary vascular occlusive causes are SMA atherosclerosis, mesenteric emboli, mesenteric vein thrombosis and occlusive arteritis (*Figure 3*) (*Table 1*). SMA embolus is the most common cause of acute vascular ischaemia and accounts for almost 50% of the cases, while 10–15% are caused by mesenteric venous thrombosis (Reinus et al, 1990).

Mesenteric arterial occlusion is mainly caused by arterial thrombosis and emboli. The SMA origin is commonly involved in the atherosclerotic narrowing and it occurs proximally causing extensive bowel infarction. The patient may give a history of chronic symptoms related to the mesenteric ischaemia. Emboli usually originate from the left atrium or the left ventricular mural thrombus. They usually lodge distal to the middle colic artery or at the origin of the right colic and ileocaecal artery.

In mesenteric venous occlusion, short segments of the proximal and middle bowel segments may be affected. The bowel appears dusky from the onset because of venous engorgement and no clear demarcation between viable and non-viable tissue can be observed. Necrosis of the bowel is more prominent than in arterial occlusion. The

Figure 2. Collateral circulation between the superior mesenteric artery and the inferior mesenteric artery.



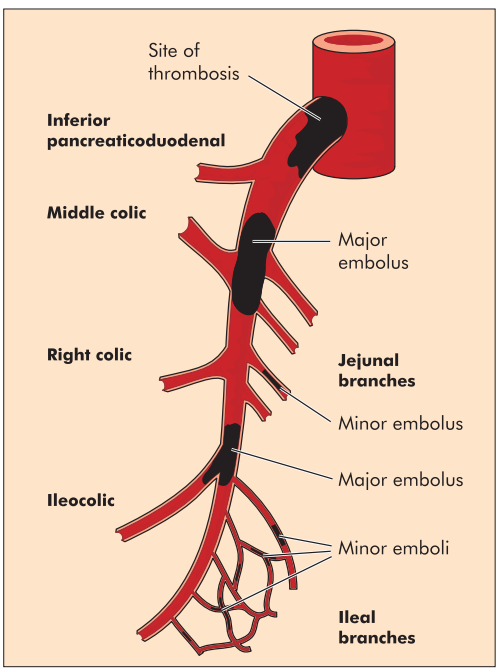


Figure 3. Common sites of thrombosis and embolus in the superior mesenteric artery.

TABLE 1.
Factors associated with occlusive intestinal ischaemia

Causes of acute intestinal ischaemia	Percentage of cases
Superior mesenteric artery embolus	45%
Superior mesenteric artery thrombosis	10%
Mesenteric venous thrombosis	10%
Non-occlusive ischaemia	20%
Other causes	15%

primary venous occlusions have no underlying cause, whereas the secondary thromboses are associated with portal venous stasis, splanchnic venous injury or hypercoaguable states.

Colonic ischaemia is most commonly seen in the elderly population; 90% of patients are over 60 years of age and have evidence of systemic atherosclerosis, and 20% of patients may have an obstructing lesion of the colon. The clinical presentation may vary with the severity of the ischaemic insult. The range of symptoms may vary from mild abdominal pain, bloody diarrhoea or stricture to frank peritonitis. There is a 2% risk of acute colonic ischaemia after abdominal aortic aneurysm repair. The warning symptoms include bloody diarrhoea after surgery, increased fluid requirement, pyrexia, leukocytosis, abdominal distension and acidosis. Localized non-occlusive ischaemia involves the watershed areas of the colon, namely the splenic flexure (Griffith's point) and the junction of sigmoid and rectum.

Non-occlusive ischaemia is usually secondary to cardiovascular events and mainly involves the small intestine. Non-occlusive mesenteric ischaemia accounts for 15–20% of acute cases and is mostly seen in patients who are already critically ill (*Table 2*). It may also result from intense visceral vasoconstriction in patients with ergot and cocaine intoxication. These patients are usually on the intensive care or the high dependency units. To diagnose the condition, clinicians need to have a high index of suspicion in patients whose general condition further deteriorates with symptoms and signs referred to the abdomen.

TABLE 2.
Factors associated with non-occlusive ischaemia

Acute myocardial infarction
Congestive cardiac failure
Cardiac arrhythmia
Sepsis
Acute pancreatitis
Haemorrhagic shock
Repair of coarctation of aorta

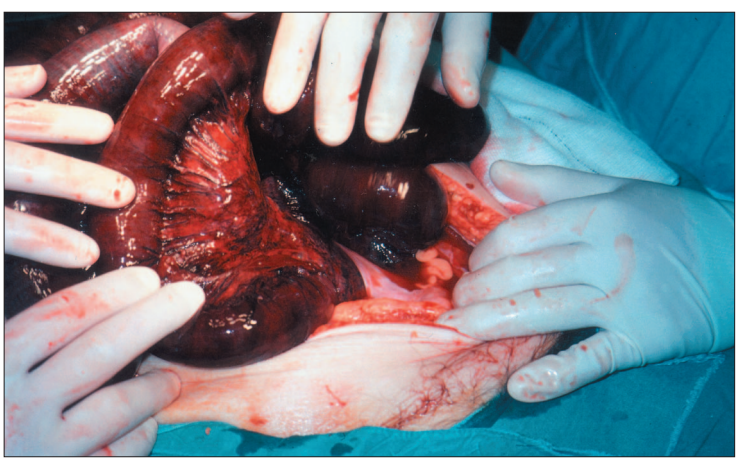


Figure 4. Gangrenous bowel found at laparotomy.

Clinical features

Patients with acute intestinal ischaemia present with symptoms which far exceed their physical signs. The patient complains of severe abdominal pain, although pain may be less prominent in colonic ischaemia. Sudden forceful gut emptying with vomiting and/or defecation may occur. An early positive sign is severe abdominal distension even when peristalsis is present. A patient with sudden onset abdominal pain, underlying cardiac disease and forceful gut emptying should be considered to potentially have embolic mesenteric ischaemia until proven otherwise. In acute SMA thrombosis, symptoms are variable and patients may complain of either steady or crampy pain in the central abdomen with radiation to the back. As necrosis develops abdominal tenderness, rebound tenderness and guarding becomes more prominent. Peritoneal signs and gastrointestinal bleeding are usually late and are associated with bowel infarction (*Figure 4*).

Diagnosis

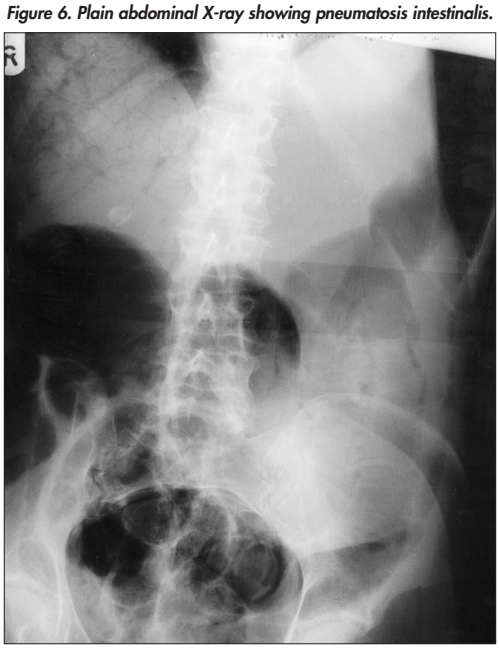
Since patients with acute ischaemia present with a wide variety of non-specific symptoms, the diagnosis of acute mesenteric ischaemia is a challenging problem. Once gut infarction sets in the mortality rate is 70–90% (Mansour, 1999). No simple non-invasive test has proved to be sensitive and specific enough to prove the diagnosis of acute mesenteric ischaemia. A thorough vascular assessment is essential on initial examination. Leucocytosis and metabolic acidosis may suggest the presence of intestinal ischaemia and serum lactate levels are elevated in the later stages. Selective mesenteric angiography has been described as the gold standard in the diagnosis of ischaemia. It allows for differentiation between the occlusive and non-occlusive ischaemia, demonstrates the site and nature of occlusion, the distal vascular bed and the mesenteric veins (Vicente and Kazmers, 1999).

Following initial resuscitation, signs of peritonitis often become evident. Patients usually undergo laparotomy with a diagnosis of peritonitis of unknown origin. It is often only then that the diagnosis of gut ischaemia is con-

firmed. After the initial resuscitation of the patient, plain abdominal X-rays should be performed mainly to exclude other causes of an acute abdomen. Non-specific changes seen on X-ray in patients with acute mesenteric ischaemia include adynamic ileus, a gasless abdomen, small bowel pseudo-obstruction and splenic flexure cut-off signs. More specific signs include thumbprinting and rigid formless loops, which are seen in colonic ischaemia.

Figure 5. Plain abdominal X-ray showing air in the biliary tree.

Figure 6. Plain abdominal X-ray showing pneumatosis intestinalis.



Once infarction sets in, linear pneumatosis intestinalis and portal venous gas can be seen on plain abdominal films (*Figures 5* and *6*).

The use of other radiological investigations is becoming more prominent and these include computed tomography (CT) scan and duplex ultrasound scanning. The CT scan can demonstrate occlusion of the mesenteric vessels, bowel wall thickening, poor enhancement of the bowel wall and intramural pneumatosis (Rha et al, 2000) (*Figure 7*).

Barium contrast studies in the acute clinical settings are rarely performed, but may be useful in colonic ischaemia, with thumbprinting and the rapidly changing pattern of the enema being characteristic findings. Wide-mouthed deep flask-shaped ulcers are also characteristic of ischaemic colitis.

Treatment

The initial treatment for patients suffering with acute gut ischaemia is active resuscitation, with correction of cardiac failure, control of arrhythmias and replacement of blood volume. The correction of plasma volume deficits, along with gastric decompression and parenteral antibiotics, is important to prevent progression of the ischaemia. An early laparotomy, rapid volume expansion and anticoagulation will be needed as dictated by the patient's vital signs (Kaleya et al, 1992).

In acute gut ischaemia the diagnosis is at best 'suspected' and not infrequently seen in cases of peritonitis of 'unknown cause'. Unlike chronic visceral ischaemia the diagnosis is often made on opening the abdomen and the findings of already irreversible ischaemic gut. In the presence of black irreversibly damaged gut there is only one option: to resect the appropriate piece of bowel, usually a large proportion of the small bowel and the right colon. In younger patients, when the small bowel resection extends up to the proximal jejunum, a primary anastomosis of this to the descending colon is done to prevent post-

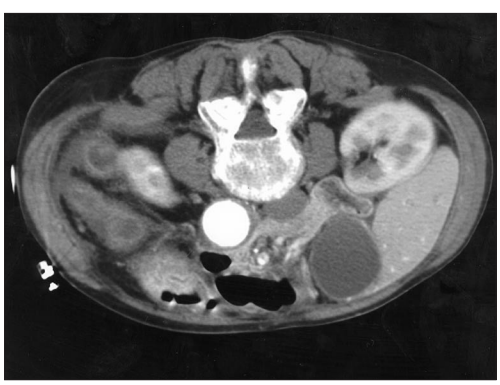


Figure 7. Computed tomography scan showing the abrupt cut-off of contrast in the superior mesenteric artery.

operative dehydration from a high output small bowel fistula. When the ischaemia is more distal, small bowel can be brought out as an ileostomy and the large bowel brought out as mucous fistulae, and a delayed anastomosis is valuable. Any progression of the ischaemia is apparent from the stoma visible above the closed abdominal wall.

A second look laparotomy should be planned in 24–48 hours, regardless of the patient's general condition. Monitoring levels of serum lactate has been described in making the decision for second look laparotomy (Brager et al, 2000). The use of vasodilators and gradual reinstitution of feeding avoids postoperative mesenteric vasospasm (Clavien et al, 1987). Postoperatively these patients require intensive care with invasive haemodynamic monitoring. Acute intestinal failure may occur as a consequence of massive intestinal resection especially in patients with less than 100 cm of jejunum. These patients will require long-term nutritional support to provide for the basal requirements of energy and nitrogen and to prevent dehydration. Central venous total parenteral nutrition is the preferred method of nutrition. If the patient can independently manage the parenteral feed before discharge from hospital, then home parenteral nutrition can be considered and this can be given in a cyclical basis, allowing day-time freedom and improving the patient's quality of life.

Small bowel transplantation is an option for such patients who are on long-term home parenteral nutrition and have an increased risk of complications secondary to home parenteral nutrition. However, only 78% of those who survive transplant are able to completely stop parenteral nutrition (Messing et al, 2001).

In cases where the patient is in atrial fibrillation and the cause appears to be an embolus to the SMA, an embolectomy can be undertaken. However, once a successful embolectomy has been undertaken via one of the major tributaries of the SMA, the vessel should be closed using a vein patch.

In the case of thrombosis of the SMA, which is more commonly seen in patients with a previous history of chronic mesenteric ischaemia, an aortomesenteric or ileo-mesenteric bypass using polytetrafluoroethylene or reversed saphenous vein graft may be contemplated.

In patients with mesenteric venous thrombosis immediate anticoagulation and volume expansion is mandatory to limit the thrombotic process. Such a diagnosis is rarely made preoperatively. However, in the presence of peritoneal signs or sepsis an urgent laparotomy is indicated. In these cases portomesentric thrombectomy should be considered and anticoagulation is continued postoperatively (Mansour, 1999; Vicente and Kazmers, 1999).

In patients with non-occlusive disease a conservative treatment approach with vasoactive drugs is indicated in the initial phase. Treatment involves management of the precipitating cardiac events and maintaining haemodynamic stability before diagnostic evaluation and definitive therapy.

CHRONIC INTESTINAL ISCHAEMIA

In this form of ischaemia the viability of the gut is often not compromised, but the flow is inadequate to support the function and demands of the intestine. As such the term abdominal migraine has been used. Colonic ischaemia was first reported as a disease process by Councilman in 1894. In 1957, Mikkelson coined the term intestinal angina for the triad of postprandial pain, weight loss and altered intestinal motility. In 1966 Marston et al used the term ischaemic colitis to describe a spectrum of ischaemic injuries to the colon (Boley et al, 1997). Because of the potential for multiple collateral flow, this condition only occurs when at least two of the three major vessels are occluded or contain critical stenoses. Atherosclerotic narrowing and gradually evolving mesenteric venous thrombosis are the common forms of chronic ischaemia. In younger patients, the median arcuate ligament compression syndrome has been described, as a result of extrinsic compression of the coeliac

TABLE 3.
Conditions associated with chronic intestinal ischaemia

Visceral artery atherosclerosis
Neurofibromatosis
Visceral artery dissection
Polyarteritis nodosa
Neurofibromatosis
Cogan's syndrome
Fibromuscular hyperplasia
Ergot poisoning
Rheumatoid arthritis
Buergers disease
Radiation injury
Systemic lupus erythematosus
Cocaine abuse
Coarctation repair

From Taylor (2000)

artery rather than an atheromatous process (Williams, 1971). Visceral ischaemic syndromes have also been described secondary to collagen vascular disorders and external beam radiation (*Table 3*).

Clinical features

A typical patient suffering with chronic intestinal ischaemia is often investigated by numerous physicians and surgeons with no definitive diagnosis made for the symptoms. The patient is usually a heavy cigarette smoker and describes pain which occurs 15–30 minutes after meals and persists for a period of 1–4 hours postprandially. The pain is described as dull and crampy in quality, located in the epigastric and periumbilical regions. Nausea, vomiting, diarrhoea and constipation are also frequent complaints. Over a period of time, the patient develops food avoidance, which is followed by considerable weight loss, in order to reduce the pain associated with the ischaemia. The average age of these patients is between 50 and 60 years with women being more commonly affected than men. Often other stigmata such as peripheral vascular disease, angina and previous myocardial infarction are seen (*Figure 8*).

Diagnosis

Unlike acute intestinal ischaemia, the chronic form can be frequently diagnosed preoperatively. A definitive diagnosis is best made on selective mesenteric angiography in both the anteroposterior and lateral projection to visualize the collaterals and the occlusion respectively (*Figure 9*).

Figure 8. A typical patient with chronic intestinal ischaemia on total parenteral nutrition.

Excellent images of the mesenteric vessels can also be obtained with three-dimensional contrast enhanced magnetic resonance angiography (MRA) and cine CT scanning. MRA has a sensitivity of 100% and specificity of 95% for the diagnosis of mesenteric ischaemia (Meaney, 1999) (*Figure 10*).

Treatment

The patient experiences relief of pain by limiting food intake, but this leads to profound weight loss. This can be offset by the administration of

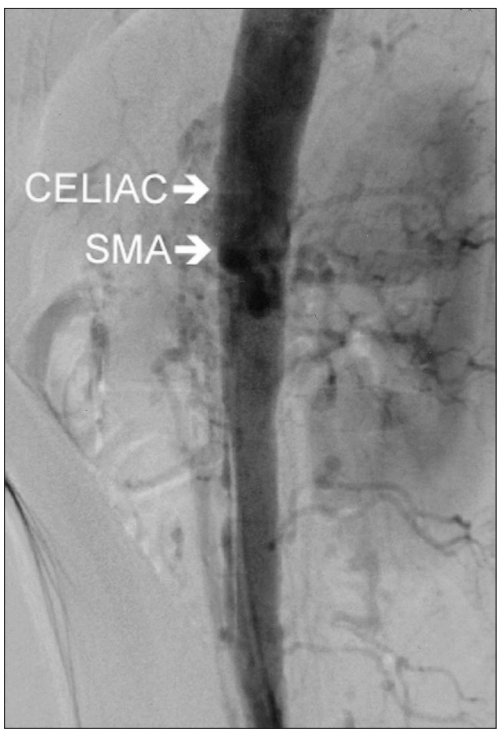


Figure 9. Arteriogram demonstrating the thrombosis in both the coeliac and the superior mesenteric artery (SMA) at its origin from the aorta.

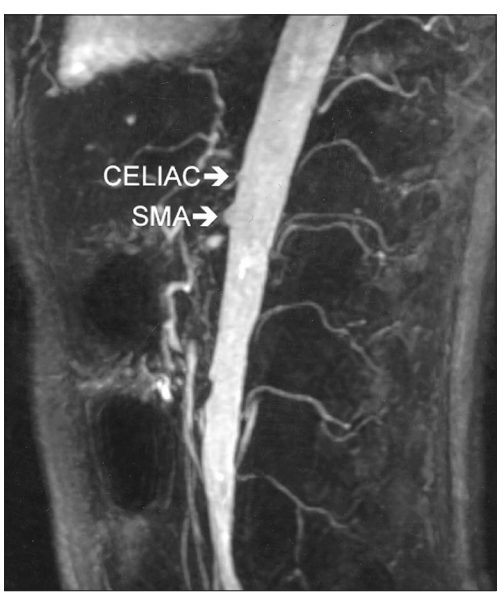


Figure 10. Magnetic resonance angiography of the same patient showing the blockage in both the coeliac and the superior mesenteric artery (SMA).

parenteral nutrition, which relieves the symptoms and allows for more definitive surgical treatment to be instituted. The aim of surgical treatment is to achieve restoration of mesenteric blood flow and maintain the bowel viability with relief of symptoms. Shaw and Maynard first described the technique of thromboendarterectomy for the treatment of intestinal ischaemia in 1957. In 1962, Morris and Crawford used a retrograde Dacron bypass graft from the infrarenal aorta to the mesenteric vessel. Stoney and Wylie, in 1966, introduced the operation of anterograde visceral bypass and transaortic visceral thromboendarterectomy, which is now routinely used in the treatment of chronic intestinal ischaemia. The results of single vessel bypass grafting to the SMA compares favourably to other techniques of bypass grafting to multiple visceral arteries (Gentile et al, 1994; Mateo et al, 1999; Foley et al, 2000). Percutaneous angioplasty and stenting has shown some improvement, but has a higher incidence of recurrent symptoms compared to patients undergoing formal bypass (Kasirajan et al, 2001) (*Figure 11*).

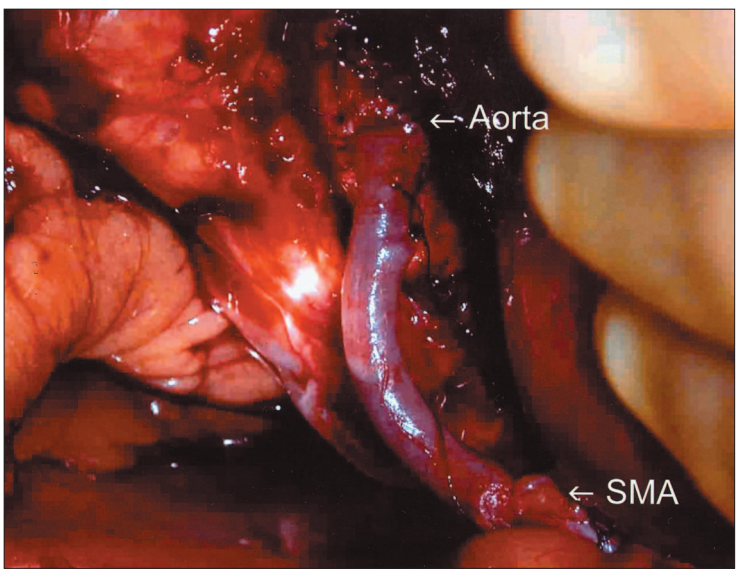


Figure 11. The reverse saphenous vein graft used for an aortomesenteric vascular graft. SMA = superior mesenteric artery.

KEY POINTS

- A high index of suspicion is needed in the diagnosis of acute intestinal ischaemia.
- The patient's symptoms far exceed the physical signs on clinical examination.
- Rule out non-occlusive causes for the abdominal symptoms.
- The presence of abdominal signs usually indicates bowel necrosis and necessitates urgent laparotomy.
- Visceral angiography is the gold standard in the diagnosis of acute intestinal ischaemia, however, contrast-enhanced magnetic resonance angiography is a good alternative investigation in chronic intestinal ischaemia.
- In general, visceral revascularization should precede intestinal resection in all patients with occlusive acute mesenteric ischaemia.

CONCLUSION

A more aggressive therapeutic approach is needed to reduce the mortality and morbidity associated with acute intestinal ischaemia. The increased mortality and morbidity is related to late presentation, delayed diagnosis along with inadequate radiological investigation and lack of institution of adjuvant treatment by the clinician. Early use of angiography or MRA along with the use of preoperative vasodilators is indicated to reduce postoperative morbidity and mortality. The surgical approach should take into account the cause of bowel ischaemia. HM

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Conflict of interest: none.

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