Introduction

Mesenteric ischaemia was first described by Antonio Beniviene in the 15th century and later by Virchow in the 19th century (2). The incidence of AMI is increasing parallel to the ageing patient population. The autopsy series in 1967 of OTTINGER and AU TNT reported a rate of 8.8 cases of AMI per 10,000 hospital admissions (3). STONEY and CUNNINGHAM in later years observed an incidence rate of 1 in 1000 hospital admissions (4). Mortality rates for elderly population over the age of 60 carries a relative risk ratio of 3.0 compared to younger patients (5). Early intervention is crucial and the potential for intestinal viability is 100% when symptoms are less than 12 hours, 56% if symptoms are 12 to 24 hours and only 18% if symptoms have been neglected for over 24 hours before diagnosis (6).

Anatomical Considerations

The arterial circulation to the gut is served by three unpaired anterior branches of aorta namely coeliac, superior and inferior mesenteric arteries (Fig. 1 & 2). They can develop extensive collaterals to supply areas of relative ischaemia in the mesenteric circulation, as often happens with chronic mesenteric ischaemia. This explains why vascular occlusion is well tolerated as evidenced by the lack of clinically significant intestinal ischaemia despite the high prevalence of atherosclerotic disease of the aorta and visceral arteries quoted in upto 35-70% of unselected patients in autopsy series (7). Clinical ischaemia results when the Superior Mesenteric Artery (SMA) is occluded in combination with one of the other two arteries. However, these collaterals are insufficient when an acute embolic insult occurs in mesenteric circulation. Certain collateral patterns are well recognised. When either the coeliac or superior mesenteric artery is compromised, the main collateral circulation is by the gastro duodenal or pancreaticoduodenal arteries. The main collateral channels between the SMA and Inferior Mesenteric Artery (IMA) occur in the region of the splenic flexure and middle colic arteries through the marginal artery of Drummond and the arch of Riolan (an ascending branch of the left colic artery anastomosing with the branches of SMA). In presence of an IMA occlusion, another important collateral circulation is between internal iliac artery and the left colic artery via the superior haemorrhoidal arteries. An embolus lodges preferentially into the SMA due to its oblique angle of emergence from the aorta. The occlusion is usually distal to the origin of pancreaticoduodenal and the middle colic branches, which allows some blood flow to the midgut to be maintained. The stomach, duodenum and proximal jejunum are spared with ischaemia extending to the mid transverse colon. The combination of proximal SMA pulsation and jejunal sparing are important intraoperative findings of prognostic significance. On the other hand, thrombosis of SMA occurs at the origin of the artery. In contrast to the embolic occlusion, the proximal SMA pulse is absent.
Acute Mesenteric Ischaemia

and the distribution of ischaemia is much more extensive. Only the stomach, duodenum and the distal colon are spared.

Physiological considerations

About 10 to 20% of the resting cardiac output flows through the visceral circulation (500 to 1200 ml) and three fourth of this blood flow preferentially supplies the mucosa due to its high metabolic activity. The remaining 25% supplies the submucosal and serosal layers of the gut. When the blood flow becomes critical, mucosal ischaemia results first and serosal ischaemia occurs later. Mucosal ischaemia leads to severe unrelenting periumbilical or epigastric visceral pain explaining the paucity of clinical signs initially i.e. a soft abdomen and this can be associated with increased bowel irritability leading to explosive diarrhoea and copious vomiting. Biochemically, this gives rise to early metabolic acido-sis, high anion gap, increased serum lactate and hyper-amylasemia. This mucosal ischaemia triggers transloca-
tion of bacteria initiating sepsis with fever and marked leucocytosis, proceeding rapidly to multiorgan failure. Successful reperfusion releases free radicals causing an inflammatory response with release of many cytokines, activated leucocytes and inflammatory mediators. This results in local mucosal and hepatic inflammation and systemic effects leading to multiorgan failure (8).

Mesenteric vasoconstriction is a normal physiological response to shock mediated largely by the renin angiotensin axis (9). In the face of persistent and prolonged hypotension, mesenteric ischaemia can result in the absence of any mechanical obstruction. This distinct entity is termed as Non Occlusive Mesenteric Ischaemia (NOMI).

Aetiology

Mesenteric ischaemia could be thrombotic or non-
thrombotic in origin. Non occlusive mesenteric ischaemia, the dominant cause of non-thrombotic mesenteric ischaemia, results from low flow states whereas thrombotic causes include arterial embolism, arterial thrombosis and mesenteric venous thrombosis. Most cases are caused by emboli (64%), followed by arterial thrombosis (27%), venous thrombosis (3.5%) and NOMI (4.8%) (10). The disease usually presents after the sixth decade and is three times more common in women and it is likely that the patient would have atherosclerotic disease in other vascular beds. A summary of causes is presented in Table 1.

Arterial emboli

The SMA is the most common site of embolic occlusion although the coeliac artery can also be affected. The
The commonest source of an embolus is cardiac usually secondary to atrial fibrillation. Less common causes are mural thrombus following an acute myocardial infarction, paradoxical emboli through septal defects, cardiomyopathies, valvular diseases, endocarditis, and atrial myxoma. A history of previous embolic events is not uncommon and up to a third of patients with an SMA embolus have a history of antecedent embolic event (11). The aorta can be a rare source of embolism from atheromatous ulcers or more rarely from primary aortic tumour (12, 13).

### Arterial thrombosis

SMA thrombosis may occur as a result of SMA stenosis that has not been previously diagnosed or treated. Characteristically, the patient reports postprandial pain and food fear with severe weight loss. The typical

<table>
<thead>
<tr>
<th>Causes of Acute Mesenteric Ischaemia</th>
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<tr>
<td>1) Emboli (50%)</td>
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<td>2) Thrombosis (25%)</td>
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<td>Dissection</td>
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<td>Cocaine Abuse</td>
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<td>3) Non Occlusive Mesenteric Ischaemia (20%)</td>
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<tr>
<td>Cardiac Failure</td>
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<td>Cardio Pulmonary Bypass</td>
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<td>Systemic Hypotension</td>
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<td>4) Mesenteric Venous Thrombosis (5%)</td>
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<td>Primary causes</td>
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<td>Antithrombin III Deficiency</td>
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<td>Trauma</td>
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<td>5) Extravascular Causes</td>
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<tr>
<td>Bands, Strangulated Hernia, Volvulus, Intussusception</td>
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![Fig. 3](image)

Occlusion of SMA at origin (black arrowhead) with reforma-
tion of the distal segment via marginal artery of Drummond.

![Fig. 4](image)

Patient with renal artery stenosis (white arrow) and complete occlusion of IMA (black arrowhead). Note the presence of aortoiliac stents (white arrowhead).

patient is a thin female and a heavy smoker with evidence of widespread arterial disease including previous myocardial infarction or claudication.
Mesenteric venous thrombosis (MVT) is rare and accounts for 5 to 15% of all acute mesenteric ischaemia. This entity was first described by Elliot in 1895 but characterised by Warren and Eberhard in 1935 (22). It is classified as primary or secondary MVT depending on the aetiology. Primary thrombosis is most commonly due to hereditary or acquired hypercoagulation disorders. Deficiency of protein C, protein S, Antithrombin III, and factor V Leiden could be discovered on screening for thrombophilia; but these proteins could be falsely low in patients with acute thrombosis. Secondary MVT may follow hypercoagulable states, portal venous stasis and hypertension, previous sclerotherapy for varices, intra abdominal infection, inflammation or malignancy, use of oral contraceptive pills and splenectomy. Long term anticoagulation is required for the treatment of MVT because of high recurrence rates. 60% patients give previous history of deep venous thrombosis (23). The clinical presentation is usually less acute than that of arterial occlusion. Severe but vague abdominal pain that tends to be colicky and slowly progressive is usually present. Few abdominal signs are present except tenderness, distension and decreased bowel sounds. The pain again is out of proportion to the clinical findings. Faecal occult blood is present in the majority of patients. Low grade pyrexia and associated leucocytosis are frequent (24, 25). Frank peritonitis is seen only when transmural infraction or perforation has occurred. When thrombosis involves the portal or splenic veins, the initial presentation may be variceal bleeding, splenomegaly or ascites (26). Macroscopically at surgery, there is usually serosanguinous ascitic fluid and the affected bowel segment is cyanotic and oedematous with rubbery texture. Mesenteric pulsations are present but the veins contain fresh thrombus that extrude when the veins are cut. Infarction is most common in the mid bowel. Involvement of the inferior mesenteric vein and large bowel is unusual. Mortality for MVT with involvement of the Superior Mesenteric Vein or Portal vein is about 30% (11).

Nonocclusive mesenteric ischaemia (NOMI)

About 20% of patients with AMI have nonocclusive ischaemia (11). NOMI typically occurs in the intensive care setting. These patients have shock either cardiogenic or septic. The mesenteric insult is usually aggravated by the use of inotropic agents such as noradrenaline (alpha agonist causing generalised vasoconstriction). Long term dialysis patients are another group who are at high risk for NOMI. The severe and rapid fluid shift associated with the dialysis is contributory to the abdominal symptoms (27, 28). Other groups of patients who are likely to present with NOMI are patients with myocardial infarction, congestive cardiac failure, following major abdominal and cardiac surgery and cardiopulmonary bypass. Digitalis preparations tend to cause splanchnic arterial and venous contraction and this is incriminated in the cardiac patients. Watershed areas of mesenteric circulation in the region of the splenic flexure tend to be affected with NOMI.

Another group which is vulnerable to NOMI is the surgical post-operative or polytrauma patient receiving enteral feeding in the intensive care unit. The reported incidence of AMI in this group is 0.3 to 8.5% (11). The increased demand from enteric feeding fails to be met by the systemic hypoperfusion and the mesenteric vasoconstriction. The diagnosis is difficult to make since the patients are often unable to alert the attention to the abdomen. Before this entity was recognised, the mortality was nearly 100% in cardiac patients following use of vasopressors, but the mortality associated with NOMI has decreased with increasing awareness and increasing use of vasodilators and afterload – reducing agents.

Iatrogenic intestinal ischaemia

Intra-aortic manipulations related to interventional radiological procedures or intra-aortic balloon pumps following cardiac surgery can lead to visceral malperfusion (29). Embolisation that results is usually global, involving the kidneys, pelvis and lower limbs as well as the viscera and hence the high mortality. The ideal treatment is not known but intuitively full intravenous anticoagulation unless specifically contraindicated, appropriate fluid resuscitation and infusion of prostacyclin or its analogues appear to be justified. Cholesterol emboli may be suspected by an eosinophilia in the blood film. This may occur in patients undergoing systemic
thrombolysis or anticoagulation and with aortic manipulation. Anticoagulation is relatively contraindicated and the use of statins might be beneficial to stabilize the plaque.

Left colonic Ischaemia can occur following aortic reconstruction (Fig. 5-6) (30). It is more common following aneurysm repair after ligation of a patent IMA. The quoted incidence is 2% (ranging from 0.2% to 10%). Mortality in this condition is 40 to 50% and approaching 90% if there is full thickness bowel wall ischaemia (31). To reduce the risk of colonic ischaemia following aortic surgery, it is imperative that the surgeon is satisfied with the colour and perfusion of the left colon prior to closure of the laparotomy. If the IMA had not been back bleeding on opening the infrarenal aorta, consideration should be given to reimplanting the IMA back on to the aortic graft. Diagnosis of ischaemic colitis can be difficult in the postoperative period and depends on high index of suspicion. Diarrhoea especially if bloody should prompt urgent bedside sigmoidoscopy. A leucocytosis greater than 20000, fever and shock and severe metabolic acidosis should alert the surgeon to the possibility of severe colonic ischaemia.

Clinical Presentation

Severe abdominal pain is the cardinal presentation of AMI. This can be sudden and dramatic in a previously well and asymptomatic patient (embolism), recurrent abdominal pain precipitating into unrelenting pain (thrombosis) or vague colicky abdominal pain which is progressive over 1 to 2 weeks (MVT). This is often accompanied by copious vomiting and explosive diarrhoea and intense urge to defaecate. In the unconscious patient, abdominal distension, gastrointestinal bleeding, occult sepsis (leucocytosis or fever) and worsening metabolic acidosis may be the subtle presenting features. Physical examination may reveal a soft abdomen with non-specific abdominal distension. Peritonism or blood in the stool or vomitus indicates advanced gastrointestinal ischaemia with likely intestinal gangrene and is generally a late clinical feature. The paucity of clinical signs initially to alert the surgeon remains the main reason for the missed or delayed diagnosis.

Diagnosis

In a patient with a known embolic source, evidence of widespread atherosclerosis and hypercoagulable state, the combination of severe abdominal pain out of proportion to the clinical findings should heighten the suspicion of mesenteric ischaemia. Delay in diagnosis contributes directly to ischaemic damage. Biochemical markers include lactic acidosis, leucocytosis, and raised CRP (10, 32-34).

Elevation of serum inorganic phosphate levels have been proposed as a marker of mesenteric Ischaemia, but this only occurs in a third of patients with AMI (32, 34).
However, in those patients who did have elevated phosphate levels, it predicted extensive injury and poor prognosis (35). The fibrinolytic marker D-dimer is elevated in thrombo-embolic occlusion of the SMA, although levels are also raised in other conditions of acute bowel ischaemia such as strangulation (36). Normal levels may serve to exclude the AMI (37).

Animal studies have suggested intestinal fatty acid binding protein (I-FABP) as a serum marker reflecting bowel ischaemia. Early human studies show promise, as patients with ischemic bowel disease demonstrate significantly higher I-FABP levels than either healthy subjects or patients with acute abdominal pain. Patients with mesenteric infarction had the highest serum I-FABP levels (38).

Radiology

Plain abdominal X-ray may reveal distended bowel loops with thumb printing suggestive of mucosal oedema and gas in the bowel wall or portal vein which is a late and ominous finding in neglected cases. Unfortunately plain films are not helpful in most cases. However they are useful to exclude other causes of acute abdomen like perforation and obstruction.

Abdominal Computerised Tomogram (CT) is increasingly used due to its universal availability and noninvasive nature. Contrast enhancement allows direct visualisation of the thrombus or embolus with multislice techniques (39). Ancillary evidence of mesenteric ischaemia includes intramural or portal venous gas, segmental bowel thickening with lack of bowel wall enhancement and liver or splenic infarcts. Conspicuous prominence of peripheral mesenteric vessels arranged in a palisade or comb fashion is seen in mesenteric ischaemia due to vasculitis. This may be associated with genito-urinary and splenic involvement which would be evident in CT. Perivascular oedema and inflammatory cell infiltration associated with vascular stasis or pooling results in the “comb-sign” described in vasculitis (18). The main disadvantage of CT angiography is its need for a large contrast load in patients who may have associated renovascular disease.

MVT can be diagnosed on contrast enhanced CT scanning by demonstration of thrombus within the superior mesenteric vein and is superior to angiography in this respect (40). Superior Mesenteric Vein may be visualised as enlarged vein with central lucency and contrast enhanced surrounding vessel wall- the so called “halo” sign in MVT (12). Mesenteric angiography is traditionally considered to be the gold standard for the diagnosis of arterial occlusion but at the cost of delay in treatment. If there are convincing abdominal signs of peritonitis urgent laparotomy is the best course of action. In the remainder of patients where AMI is suspected biplanar angiography is indicated. A lateral flush aortogram will help in assessing the origins of the mesenteric vessels and the antero-posterior views will demonstrate the distal arcades. Selective angiograms are performed when intervention is considered appropriate. In acute SMA
thrombosis, there is usually non-visualization of this artery because of the ostial nature of the disease (Fig. 3), although delayed views may show slow filling of the distal vessel. In SMA emboli, the proximal artery to just beyond the origin of the middle colic artery can be demonstrated angiographically. Delayed arterial filling and lack of venous phase on angiograms suggest MVT. Selective angiography of mesenteric arteries in NOMI will show vasospasm and is useful to exclude a significant arterial lesion (41). Narrowing of the origin of SMA with alternating dilatation and narrowing of the enteric branches gives rise to the radiological appearance of “string of sausage” sign in NOMI. Once the diagnosis is made on selective angiogram, the angiocatheter should not be removed until all therapeutic possibilities are discussed with the surgeon.

Magnetic resonance angiography (MRA) with three dimensional gadolinium enhanced reconstruction does not require ionising radiation and is particularly useful in chronic mesenteric ischaemia since it is noninvasive. However its role in acute setting remains controversial. The distal mesenteric vessels cannot be well visualised on MRA (42) and it is unhelpful in cases of NOMI.

Duplex scanning is limited by the increased intestinal gas that is frequently present in association; it is again non-invasive and in good hands gives comparable results (43).

**Treatment**

 Expedient revascularisation to prevent intestinal gangrene is the main aim of treatment. In all cases, the patient should be aggressively resuscitated, broad-spectrum intravenous antibiotics instituted and fully heparinised once the diagnosis is confirmed. As yet, the twin goals of mesenteric revascularization and resection of nonviable bowel can only be achieved by surgical means. Treatment pathways is summarised in Figure 7.

**Surgical**

Surgery is indicated in all patients with peritonitis after rapid resuscitation. At laparotomy the surgeon should establish the diagnosis, consider appropriate revascularisation and resect the already damaged bowel. The decision for relook laparotomy should be made at the initial
surgery itself and is independent of the clinical status of the patient between the two procedures (5).

The first step is to identify the underlying pathology. The presence of proximal artery pulsation and jejunal sparing are intraoperative clues for an embolus. Conversely, absence of proximal mesenteric pulsation and jejunal involvement indicate thrombosis.

The crucial and most taxing step is to assess the degree and extent of bowel viability. Ischaemic bowel has a characteristic appearance with loss of its normal sheen. It is dull, grey in colour and flabby in tone without any peristalsis. Infarcted bowel is purplish black in colour, often friable and perforated. Free, foul smelling peritoneal fluid is a sign of advanced necrosis even if perforation has not occurred. In many cases the bowel ischaemia will be so extensive and advanced that it would be prudent to offer palliation alone. Where there is hope of sufficient bowel viability, revascularization should be first performed before any bowel resection is considered. After successful revascularization, previously precarious segments of intestine may recover and resection of clearly ischaemic bowel can then take place.

**SMA embolectomy**

The proximal portion of the SMA is dissected free from the paraaortic fat and lymphatic tissue just as it emerges from the pancreatic neck into the base of the mesentry. The artery is medial to the vein and approximately 3 to 4 centimetres of artery is cleared, with care taken not to cause any collateral damage. Heparin 80 iu/kg is given intravenously and the vessel is controlled between slings. A transverse arteriotomy is made in the SMA proximal to the middle colic artery take off unless reconstruction is anticipated and a 3F or 4F embolectomy catheter is passed proximally and distally to clear the embolus and re-establish vigorous pulsatile flow. If proximal flow cannot be established, SMA thrombosis is likely and reconstructive surgery will be required. The use of intraoperative thrombolysis after embolectomy is likely to help in removing the thrombus from distal branches which cannot be cleared completely by catheter trawl alone (44). Anticoagulation is continued in the postoperative period with heparin at 18 iu/kg to maintain APTT ratio of over twice normal.

**SMA reconstruction**

Revascularization can be performed either by re-implantation of the healthy portion of the SMA after thromboendarterectomy into the aorta or using bypass grafting from the aorta to the patent SMA. The clear advantage of autologus saphenous vein over prosthetic graft observed in other vascular beds is not so evident in the visceral circulation (45). In presence of a potentially or de facto infected field, prosthetic grafts should be avoided. Reversed saphenous vein aortomesenteric grafting or direct SMA re-implantation is the procedure of choice in this situation. Bypass with vein or prosthesis is prone to kinking due to its configuration, and great care must be taken to align the grafts to avoid this complication. Antegrade bypass grafting from relatively disease free supracoeliac aorta resists kinking better than retrograde bypass grafts (45). Single vessel revascularization is usually adequate in emergency (46) and probably also in the non emergency situation, but opinion is varied as to whether SMA or celiac axis should be revascularised in preference (5, 47).

After successful revascularization, previously precarious segments of intestine may recover and resection of clearly ischaemic bowel can then take place. However, determination of viability by eyeballing could be deceptive. The decision about how much to resect can be crucial to the long-term outcome. Clinical assessment by detecting pulsation in the arcades, colour of the bowel, peristalsis and bleeding from cut edges are most commonly used. This is often complemented by the use of the intraoperative Doppler probe to detect flow in the intestinal wall in addition to flow in the arcade vessels. Other techniques include the use of fluorescein and inspection under Wood’s lamp, pulse oximetry and laser Doppler flowmetry. Since most of the ischaemic damage is at the mucosal level, the gross appearance of the serosal aspect is often misleading. For the same reason, it may be advisable to avoid anastomosis after resection. Some even advocate stapling off both ends of the remaining gut so that the surgeon would be forced to undertake relook laparotomy prior to establishing intestinal continuity (48).

In cases of MVT, resection of infarcted bowel with liberal margins should be performed with a primary anastomosis considered only if perfusion is inadequate. Venous thrombectomy has poor results with a high recurrence rate and is rarely indicated. Laparoscopy is suspected MVT is appealing and can be useful in the management algorithm to reduce the surgical stress (49).

Post aortic aneurysm repair colon ischaemia requiring surgery usually necessitates a Hartmann’s procedure, a primary anastomosis being contraindicated. Consideration must be made to protect the aortic graft and its limbs from contamination by using antiseptic soaked swabs.

Abdominal compartment syndrome is more commonly recognised now and in cases of massive fluid shifts associated with infarcted gut, closure of the abdomen with Bogota bag or its variation may be appropriate (50, 51).

Second look laparotomy to assess marginally viable bowel left behind at the original surgery should take place as a planned procedure (48). Laparoscopy under
local anaesthesia is also in vogue now to reduce the trauma of repeated laparotomies in these sick patients (49, 52). The concept of bedside laparoscopy in a critically ill patient to avoid the trauma of a relook laparotomy, with laparoscopic trocar inserted and secured at the initial surgery appears very appealing (53).

Continued intensive care is required in the postoperative period with optimization of cardiac and respiratory status. There is an associated deterioration in hepatic function with transaminases rising 90- to 100-fold (8). The hepatic impairment and associated coagulopathy is usually transient, returning to normal within seven to ten days. Total parenteral nutrition may be started early in the postoperative period, and may need to be continued for months in cases with short gut syndromes.

In cases of MVT, long-term anticoagulation is required because of the high recurrence rates. Complications such as ascites and portal hypertension may require specific treatment (25).

**Nonsurgical/conservative treatment**

Non surgical treatment is contemplated only in absence of clinical evidence of peritonitis. In NOMI, treatment is nonoperative and depends on treating the underlying conditions such as sepsis and optimizing cardiac output with judicious use of inotropes and after load reducing agents. Noradrenalin is best avoided. If the diagnosis of AMI is made without angiography, intravenous glucagon infused at 1µg/kg per minute and titrated up to 10 µg/kg/minute as tolerated may help to reduce the associated vasospasm. If the diagnosis is made on angiography, the catheter is left in-situ and intra arterial infusion of papaverine at a dose of 30 to 60 mg/ h may be attempted (11). Papaverine, a phosphodiesterase inhibitor increases the mesenteric flow to improve bowel salvage. Up to 65% of patients who underwent cardiac surgery have had symptomatic improvement within hours when AMI was diagnosed early (54).

If MVT is diagnosed at angiography, intra-arterial thrombolytic therapy can be given successfully (55, 56). However, thrombolysis is contraindicated when bowel infarction is suspected (11). Nonoperative management by full anticoagulation for acute MVT is feasible when the initial diagnosis is certain and when the bowel infarction has not led to transmural necrosis and bowel perforation. The morbidity, mortality, and survival rates are similar in cases of surgical and nonoperative management (56).

Other reported endovascular procedures for acute intestinal ischaemia include fenestration and stent placement in aortic dissection (57, 58), angioplasty and stenting in acute occlusion in patients with chronic mesenteric insufficiency (59, 60) and angioplasty alone (61). This contrasts with an increasing use of angioplasty for chronic mesenteric ischaemia (62). The role of thrombolysis in AMI is not well defined and remains anecdotal. The body of experience with endovascular therapy is emerging (7, 60, 63) but it is likely that it would be favoured only in those with unacceptable surgical risk. There may be a role for endovascular treatment followed by laparoscopy to assess bowel viability but currently the evidence is not sufficient to support this strategy (64).

The use of allopurinol, angiotensin converting enzyme inhibitors and other free oxygen scavengers may help to reduce the reperfusion syndrome (11).

**Prognosis**

Most studies tend to report dismal outcome in acute mesenteric ischaemia in comparison to chronic ischaemia. Perioperative mortality for arterial thrombosis remains high, in part because of delayed diagnosis, the extensive nature of the bowel ischaemia and the need for complex surgical revascularisation (11).

A retrospective multicenter study by the French associations of surgical research comparing the periods from 1980 to 1985 and 1990 to 1995, appear to hold promise (65). Mortality rates decreased from 83% to 63% in thrombotic acute mesenteric ischaemia and from 51% to 19% in MVT. There were no significant increases in frequencies of angiography, vascular or second look procedures to account for the change. Patients with NOMI have fewer treatment options since the offending stimulus often results from treatment for another disorder. Unless the original insult is reversed, the outcome is grave.

**Conclusion**

Earlier in the last century, it was felt that in AMI “the diagnosis is impossible, the prognosis hopeless and the treatment useless” (66). The same pessimistic attitude is shared by many and is reflected in Taylor’s words “mortality rate for patients with AMI will probably always remain high” (67). With high index of suspicion for the diagnosis, aggressive approach to early restoration of perfusion, second-look laparotomy and supportive care, patient outcome has improved over the years. Though the results of surgical treatment remain unsatisfactory, improved imaging and thrombolytic therapies hold much promise (68). The contemporary management of AMI with revascularisation with open surgical techniques, resection of non-viable bowel and liberal use of second look laparotomy results in the early survival of two thirds of patients with embolism and thrombosis (5).

**References**


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