Acute mesenteric ischaemia 2C01, 2C04

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Acute mesenteric ischaemia (AMI) is a surgical emergency, and has a high mortality. The term AMI covers arterial embolism, arterial thrombosis, non-occlusive mesenteric ischaemia and venous thrombosis which all lead to ischaemia/reperfusion syndrome of the bowel. Multi-detector row helical CT (MDCT) technology has dramatically improved the performance of CT by allowing rapid volumetric data acquisition to provide increased resolution, leading to better identification of the site, level and cause of ischaemia. CT angiography for diagnosing mesenteric ischaemia is now highly sensitive and specific, and should be used as first line when AMI is suspected. The aim of management is to restore intestinal blood flow in a timely manner. Therapeutic decisions are based on the presence of peritonitis, the presence of irreversible ischaemia or infarcted segments of the bowel, the general condition of the patient and the pathophysiological process underlying the ischaemia. AMI remains a challenging condition with high mortality. There is a need for good general surgical cover on the intensive care unit, with continuing care and clinical review by experienced senior surgeons with an interest in this condition.

Keywords: *acute mesenteric ischaemia; ischaemic bowel; non-occlusive mesenteric ischaemia; emergency laparotomy*

Pathophysiology

Acute mesenteric ischaemia (AMI) is an umbrella term that encompasses four main pathophysiological mechanisms leading to an ischaemia/reperfusion syndrome of the bowel, which is responsible for the development of multiple organ failure and death if not treated promptly.[1] These pathophysiological mechanisms are arterial embolism, arterial thrombosis, non-occlusive mesenteric ischaemia (NOMI) and venous thrombosis (**Figure 1**).[2,3]

Arterial embolus

Arterial embolus often occurs in mid to distal end of the superior mesenteric artery and usually originates from the heart.[2,3] This can often be seen following embolism of thrombus following atrial fibrillation and other cardiac dysrhythmias.

Arterial thrombosis

This usually occurs as the result of the rupture of an atherosclerotic plaque in the proximal half of the mesenteric artery and 75% of patients have pre-existing chronic mesenteric ischaemia.[4] Due to the chronic nature of this condition and the development of collateral vessels, the patient is usually able to tolerate major visceral artery occlusion before symptoms of ischaemia present.[1]

Non-occlusive mesenteric ischaemia (NOMI)

Diffuse vasospasm of the mesenteric and other visceral arteries occurs as the result of sustained hypoperfusion to the area.[5,6] Pulsatile blood flow can be seen in the large arteries, as no vascular occlusion is seen.[7] Risk factors for the development of NOMI include increased age, increased BMI, heart failure, pre-existing vascular disease, arterial hypotension, elevated sympathomemetic activity, hypovolaemia, sepsis, use of vasopressors and intra-aortic balloon pump (IABP).[6,7] Vasospasm caused by interference to arterial autoregulation from increased catecholamines can also occur.[8]

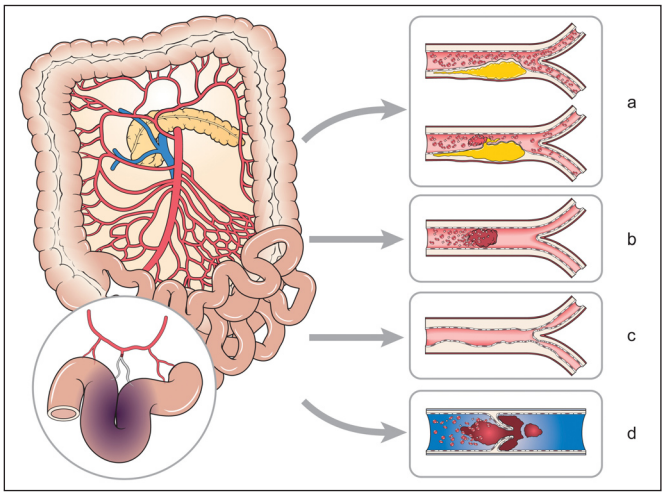

Figure 1 Pathophysiology of acute mesenteric ischaemia – (a) arterial thrombosis, (b) arterial embolism, (c) non-occlusive mesenteric ischaemia, and (d) venous thrombosis.

Venous occlusion

This leads to interstitial swelling in the bowel with subsequent arterial flow disturbance leading to the development of necrosis.[1] Causes include portal hypertension, hypercoagulability and thrombocytosis. Risk factors for AMI are summarised in **Table 1**.

Diagnosis

Mesenteric ischaemia remains a difficult condition to diagnose, with similar signs and symptoms to those seen in other

Increased age
Increased body mass index (BMI)
Heart failure
Pre-existing vascular disease
Arterial hypotension
Elevated sympathomemetic activity
Use of vasopressors
Sepsis
Use of intra aortic balloon pump (IABP)

Table 1 Risk factors for acute mesenteric ischaemia (AMI).

conditions (**Table 2**).[9] Abdominal pain which is severe and colicky in nature is usually present but other symptoms such as nausea, vomiting and diarrhoea are all fairly non-specific. Signs of peritonitis, guarding and abdominal distension are not usually present until fairly late in the disease process, often leading to delay in diagnosis and increased mortality in such patients.[1] No specific lab test is sufficiently sensitive to diagnose AMI[10] in the early stages and indeed tests such as CRP, WCC and lactate can all be normal even when ischaemia is confirmed at laparotomy.[11]

Plain abdominal radiography is not sufficiently sensitive or specific to help in the diagnosis of AMI.[12,13] MRI angiography shows promise in the diagnosis of ischaemia, however the long turnaround time and logistics of transferring critically ill patients for such tests makes this impractical for routine use.[14,15] Mesenteric duplex sonography can only detect diminished blood flow in the trunks of the mesenteric blood vessels and is very user-dependent, limiting its usefulness in the acute situation. This test is more useful in detecting chronic mesenteric ischaemia.[16,18]

CT scanning is quick and widely available and is considered the initial test of choice.[19] It has a sensitivity and specificity of 64% and 92%, respectively[11,20,21] and is useful in detecting signs of ischaemic vessel stenosis and occlusion. More recently, multi-detector row helical CT (MDCT) technology has dramatically improved the performance of CT by allowing rapid volumetric data acquisition to provide increased resolution, leading to better identification of the site, level and cause of ischaemia.[22] With these developments, the ability of CT angiography to diagnose AMI has recently been reported to have a sensitivity of around 90% to 100% and specificity of 100%.[23-26] Groups now suggest that MDCT angiography is now an accurate tool for fast diagnostic work-up of patients with suspected AMI.[23]

Angiography has long been the gold standard in the diagnosis,[27] with a sensitivity of 74-100% and specificity of 100%.[28] Angiography allows visualisation of the vessels and pre-surgery planning and targeting of the affected lesion, and is able to differentiate between embolic, thrombotic and the characteristic narrowing and multiple irregularities of NOMI. However, because the technique is invasive, time consuming and has a significant risk of nephrotoxicity, it should probably not be used in the first instance, as this may lead to delay of definitive surgical treatment. For this reason, CT angiography is now the investigation of choice and should be used promptly to identify the underlying cause and to aid surgical planning.

Abdominal abscess	Cholelithiasis
Abdominal angina	Colonic obstruction
Abdominal aortic aneurysm	Diverticulitis
Acute abdomen	Ectopic pregnancy
Acute pancreatitis	Oesophageal rupture
Acute pyelonephritis	Gastric volvulus
Aortic dissection	*Helicobacter pylori* infection
Appendicitis	Ileus
Bacterial pneumonia	Intestinal perforation
Biliary colic	Myocardial infarction
Biliary obstruction	Pneumothorax
Boerhaave syndrome	Pregnancy
Cholangitis	Sepsis
Cholecystitis	Testicular torsion
Choledocholithiasis	

Table 2 Differential diagnosis of acute mesenteric ischaemia. (Adapted from Ravipati *et al* 2011).[9]

Management

The aim of management is to restore intestinal blood flow in a timely manner. Aggressive haemodynamic optimisation, correction of metabolic acidosis, broad spectrum antibiotics and nasogastric tube placement should be undertaken promptly while definitive diagnosis and treatment are sought. The use of splanchnic vasodilators had been described in the management, as has the use of anticoagulation; however it has been shown that the management of AMI with revascularisation and open surgical techniques may result in improved survival rates.[29] Although percutaneous angioplasty may be successful in AMI[30], it is not usually performed due to the associated high risk of thromboembolus.[9]

There is no role for heparin in the acute management of bowel ischaemia, however heparin anticoagulation is the main treatment of mesenteric venous thrombosis.[9]

Therapeutic decisions are based on the presence of peritonitis, the presence of irreversible ischaemia or infarcted segments of the bowel, the general condition of the patient and the pathophysiological process underlying the ischaemia.[1] If surgical intervention is required, the use of laparoscopy can be useful in the first instance to assess bowel viability and allow prompt visualisation of the bowel and vasculature.

In patients with embolic disease, urgent surgical embolectomy and anticoagulation should be undertaken, along with an assessment of the bowel for possible resection.[31] Infarcted bowel should be removed surgically. If bowel resection is not required or there is marginally perfused bowel after revascularisation, there should be a low threshold for

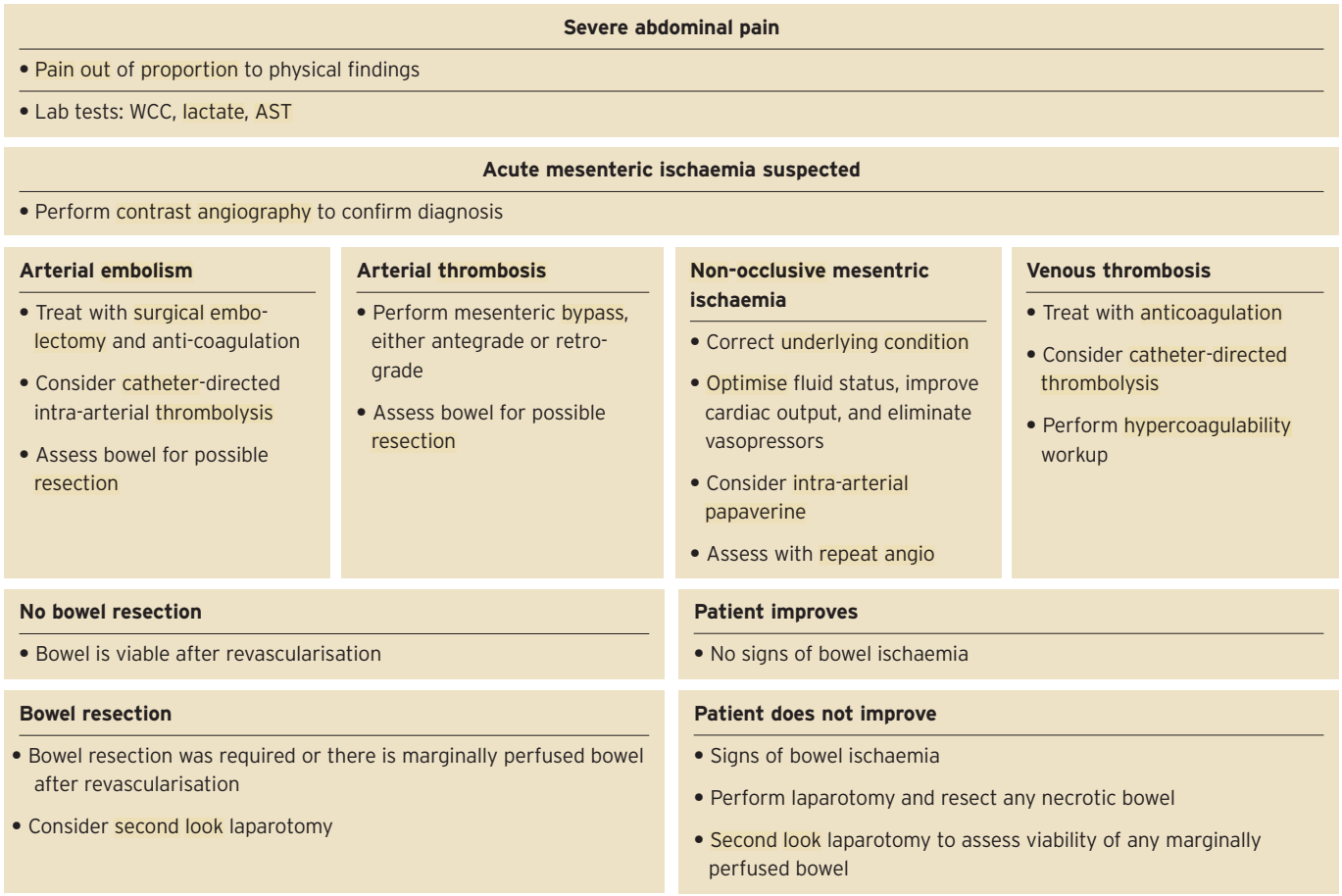

Figure 2 Flow chart of diagnostic and therapeutic options (adapted from Kibbe and Hassoun.[44]

second-look laparotomy.[31] Endovascular treatment may be an effective alternative to surgical revascularisation in appropriately selected patients.[32] The use of thrombolytic agents is still largely experimental[33] and generally confined to patients with a contraindication to surgery,[9] however, they can be effective[1] especially if clots are distally placed and treated within 12 hours after onset of symptoms.[34] If these patients develop signs of peritonitis, they should undergo surgical exploration without delay, when the bowel can be assessed for viability and infarcted bowel removed.

In patients with arterial thrombosis, urgent mesenteric bypass, either antegrade (from supraceliac aorta to superior mesenteric artery (SMA)) or retrograde (from infrarenal aorta or iliac artery to SMA) should be performed at laparotomy.[31] As with embolic disease, the revascularised bowel should be assessed for viability, with non-viable bowel resected. Again, second-look laparotomy should be undertaken if there is any concern over further ischaemia.

In patients with NOMI, the aims of management are to optimise the fluid status,[31] improve cardiac output and eliminate the use of vasopressors, in the first instance. Therapeutic options during angiography include the administration of intra-arterial vasodilators or thrombolytic agents, angioplasty, placement of a vascular stent and embolectomy, depending upon the cause of ischaemia and the anatomy of the obstruction. Intra-arterial papaverine[42] or tolazoline with heparin[35] are both effective treatments. Haemodynamic stabilisation should be achieved prior to the study, since angiography will demonstrate mesenteric vasoconstriction even in the absence of mesenteric ischaemia in the setting of hypotension or hypovolemia. Repeat angiography should be used to assess therapy. If the patient does not improve or shows signs of bowel ischaemia, a laparotomy should be performed promptly, with any necrotic bowel resected.[31]

Patients with mesenteric venous thrombosis and no signs of bowel infarction should be anticoagulated and catheter-directed thrombolysis should be considered. Thrombolytics can be administered either intravenously or directly into the mesenteric venous circulation, through a transjugular[36] or percutaneous approach. Thrombolysis should be followed by anticoagulation for 3-6 months. Again, if the patient does not improve or shows signs of bowel ischaemia, a laparotomy should be undertaken urgently.

Surgery

Whereas the treatment for AMI of the inferior mesenteric artery (IMA) territory is often surgical and consists of a single stage Hartmann's resection, the treatment options for SMA ischaemia are different. Treatment may include open surgery or an endovascular approach, and patients are best treated in a vascular centre with a hybrid endovascular operating room.

Morbidity and mortality following open versus endovascular AMI interventions are similar, but no randomised clinical trial data exist making it difficult to compare outcomes, as there are many potential confounders.[37,38]

Laparotomy or laparoscopy after mesenteric revascularisation is necessary to evaluate possible damage to the visceral organs. Bowel and organ resection as a result of clear transmural necrosis are performed according to damage control principles, with resection, stapling of bowel ends and re-look in 24-48 hours.[39]

Patients with complete transmural infarction of the small bowel to the mid-transverse colon need extensive bowel resection that will lead to short bowel syndrome and increased morbidity; this procedure may be inappropriate in some patients. In the event of perforation, bowel resections are performed with staples, leaving the creation of anastomoses or stomas until the second- or third-look laparotomy.

The abdominal wall can be left unsutured when repeat laparotomy is planned. In this situation, simple skin closure or temporary abdominal closure with a 'bogota bag' or an abdominal VAC dressing (KCL, V.A.C® Therapy, ABThera™) may be applied.

Laparoscopy does not offer significant advantages in AMI but maintains a potential role as a bedside/intensive care unit (ICU) and second-look procedure. There are limited studies with comparative data to help establish benefit of the diagnostic role of laparoscopy.[40]

Following surgical repair of abdominal aortic aneurysm, CT angiography is the best diagnostic approach in a patient with the clinical suspicion of AMI, (sensitivity 93.3% and specificity 95.9%)[41] and the use of early laparoscopy following endovascular abdominal aneurysm reconstruction (EVAR) is a promising option, but again limited data are available.

Depending on the degree of ischaemia and necrosis, a number of surgical outcomes are possible. In cases where there is extensive necrosis that is too extensive for surgical resection, the patient should have palliative care measures instituted. Patients with less extensive necrosis can have either primary end-to-end anastomoses or resection with stoma formation. Where necrosis is extensive but deemed resectable, issues which can arise are short bowel syndrome, the requirement for long-term parenteral nutrition and in certain cases, the possibility of bowel transplantation. Diagnostic and therapeutic options are summarised in **Figure 2**.

Emergency laparotomy network

The Emergency Laparotomy Network was launched in January 2010. The network's aims are to bring together clinicians from relevant specialties in order to improve outcomes.[42] The network has recently published results of a prospective audit of 1,853 patients.[43] The results show unadjusted 30-day mortality of 14.9% for all patients and 24.4% in patients aged 80 or over. There was wide variation between units in terms of the proportion of cases subject to key interventions which may affect outcomes and was reflected in the wide differences in mortality rates, from 3.6% to 41.7%. Given the heterogeneity of the sample, it is difficult to make any meaningful conclusions from the data. The variation in clinical management and outcomes indicates the need for a national quality improvement programme. The National Emergency Laparotomy Audit (NELA) aims to develop a continuous prospective registry to capture all patients undergoing emergency laparotomy. The registry will capture data including patient demographics, organisation of care and process and outcome measures peri-operatively.[43] The NELA started to collect data in Jan 2014. All centres dealing with patients undergoing emergency laparotomy should participate.

Conclusion

Although relatively uncommon, AMI remains a disease process associated with high mortality. Improved diagnostic accuracy of multi-detector row helical CT angiography looks promising in establishing prompt diagnosis.

Efficient patient management relies on multi-disciplinary team collaboration. The most important prognostic factor that can be influenced by the intensivist is the time interval between onset of symptoms and definitive treatment. CT angiography allows prompt diagnosis, lesion identification and surgical planning, and should be performed urgently in any patient in whom there is a suspicion of bowel ischaemia. The benefits of new surgical approaches such as embolectomy and mesenteric bypass to improve bowel perfusion and reduce the need for surgical resection, are dependent on having a high index of suspicion for AMI. In any patient with symptoms or signs, an urgent CT angiogram should be performed. This can be performed rapidly and doing this early is the only way to catch the pathological process before the onset of bowel necrosis and associated high mortality.

Management priorities in NOMI are haemodynamic and cardiac output optimisation, reduction in vasopressor administration and consideration of interventional techniques. However patients with peritonitis or signs of late disease still require urgent laparotomy and may need bowel resection.

By improving awareness and early recognition of AMI, coupled with multi-disciplinary diagnostic and treatment pathways, there is a chance that diagnosis and treatment in the early stages of ischaemia will lead to improved survival in this condition. Work by the National Emergency Laparotomy Network (NELN) goes some way to identifying the wide variation in outcomes across the country, and should lead to better treatment of this condition in the future.

AMI remains a challenging condition with a high mortality. There is a need for good general surgical cover on the ICU, with continuing care and clinical review by experienced senior surgeons with an interest in this condition. There is also a requirement for the provision of interventional radiological services and agreed diagnostic and therapeutic pathways to allow for prompt diagnosis and treatment of this condition.

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