Evans, D., Dovell, G., & Smith, F. CT. (2019). Mesenteric ischaemia. *Surgery*, *37*(2), 119-127. https://doi.org/10.1016/j.mpsur.2018.12.006

VASCULAR SURGERY

Mesenteric ischaemia

David Evans BSc MBBCh, Academic Foundation Doctor North Bristol NHS Trust, UK. Conflicts of interest: None

George Dovell MBBCh MRCS, Academic Clinical Fellow Vascular Surgery North Bristol NHS Trust, Bristol Centre for Surgical Research University of Bristol, UK. Conflicts of interest: None

Frank CT Smith BSc MSc MD FRCS FEBVS, Professor of Vascular Surgery and Surgical Education at the University of Bristol and North Bristol NHS Trust, UK. Conflicts of interest: None

ABSTRACT

Mesenteric ischaemic is a life-threatening condition that occurs as a result of interrupted or reduced blood flow to the bowel. It is categorised as acute or chronic and encompasses a range of pathology and symptoms. This article looks at the presentation, diagnosis, and management of the following: acute and chronic mesenteric ischaemia, ischaemic colitis, and venous infarction.

Key Words

Mesenteric ischaemia; bowel ischaemia; bowel infarction; colonic ischaemia; mesenteric embolus; ischaemic colitis; mesenteric angina.

ACUTE MESENTERIC ISCHAEMIA

EPIDEMIOLOGY

Acute mesenteric ischaemia (AMI) is a surgical emergency with published mortality rates varying between 50% and 100% [1]. Establishing the incidence of AMI is difficult as it is usually diagnosed intra-operatively, or at autopsy (with varying post-mortem rates). 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, and the incidence increases with age, showing no gender difference.

AETIOLOGY

The principal causes of mesenteric ischaemia include:

- 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 causes are rare and include aneurysmal disease, aortic dissection, and vasculitis. Emboli are the commonest cause of AMI and account for up to 50% of cases [2]. Recent reports suggest thrombosis may be becoming more common. This is likely a reflection on

the increasing burden of atherosclerotic disease (and possibly better management of AF, or reduction in emboli due to rheumatic valve disease) [3].

PATHOLOGY

The main arterial supply to the bowel comes from 3 major branches of the abdominal aorta and is based on the embryological development of the gut:

- Coeliac Axis – supplies the foregut (distal oesophagus to 2nd part of duodenum)
- Superior Mesenteric Artery – supplies the midgut (3rd part of duodenum to mid-transverse colon)
- Inferior Mesenteric Artery – supplies the hindgut (mid-transverse colon to rectum)

The SMA is more commonly affected by emboli, due to the oblique angle of origin from the aorta compared to the coeliac axis. Five percent of all peripheral emboli lodge in the SMA. Twenty percent of patients with SMA emboli have clinical evidence of simultaneous emboli elsewhere in the body, and as many as two thirds of patients are found to have concurrent emboli at post-mortem. Splanchnic autoregulation preserves mesenteric blood flow at normal blood pressures, accounting for 10-20% of resting cardiac output, increasing to 35% following a meal. When systolic blood pressure falls below 70mmHg mesenteric blood flow also falls, showing a linear correlation and gut ischaemia occurs below 40mmHg. Changes in villi can be seen after 15 minutes of absolute ischaemia; the mucosa sloughs after 3 hours; and transmural necrosis occurs after 6 hours (Figure 3). The consequences of these ischaemic changes can result in perforation, sepsis, and ultimately, death. Collateral blood vessels may mitigate the effects of arterial occlusion, more commonly in thrombosis than

emboli, since they have time to develop as a result of the underlying stenotic disease. Bowel infarction is more severe with thrombotic occlusion, as it tends to occur more proximally than embolic occlusion. Up to 75% of patients with a thrombotic occlusion of the SMA have previous symptoms of chronic mesenteric ischaemia.

PRESENTATION

The presentation of AMI varies considerably, and therefore requires a high index of suspicion. Many patients are diagnosed too late by which point ischaemia is extensive and the bowel is unsalvageable. The classic triad of symptoms for acute embolic ischaemia include:

- Abdominal pain – especially out of proportion to clinical findings (e.g. lack of peritonitis). Pain usually starts as intermittent spasmodic discomfort in the umbilical or epigastrium region before becoming constant.
- Bowel evacuation (vomiting, diarrhoea)
- Identified source of embolus (atrial fibrillation, myocardial infarction)

Other symptoms may include:

- Per-rectal bleeding – usually a late sign indicating significant mucosal injury
- Tachycardia with hypotension
- Abdominal distension
- Peritonitis – this is a late sign associated with bowel infarction

- Bowel sounds may be present or absent

INVESTIGATIONS

Diagnosis is guided by clinical suspicion, often correlated with computed tomography (CT) scan findings, as other investigations are likely to yield non-specific abnormalities [4].

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 [5]. Lactate is a by-product of anaerobic respiration. Cells that don't receive adequate oxygenation, due to ischaemia, will switch to anaerobic respiration and release an increased amount of lactate. Lactate is very effectively metabolised in the liver, explaining why it is only elevated in the latter stages of mesenteric ischemia. Lactate only really becomes diagnostic when the bowel is necrotic/gangrenous. The patient is usually extremely unwell by this point, with hypotension, tachycardia and mounting a profound systemic inflammatory response syndrome (SIRS) response. This is why the European Society for Vascular Surgery (ESVS) guidelines have issued a strong recommendation not to use lactate to diagnose this condition in early onset [4]. A normal lactate does not exclude AMI.

Raised white cell count (e.g.>20,000) is common, found in up to 90% of cases [6], 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 [1,7]. However, a negative D-dimer can exclude AMI.

Radiology

Plain radiological films are of limited use as findings are often non-specific and may even be normal. The film may demonstrate 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 and remains the gold standard for diagnosis. Mesenteric angiography can also differentiate between the aetiology of ischaemia (embolic or thrombotic), which will help guide management. CTA is non-invasive, readily available and less operator dependent than other imaging modalities such as ultrasound, which may not be able to pick up smaller distal occlusions. A 2011 meta-analysis reported CTA to have high sensitivity (93%) and specificity (96%) in diagnosing AMI. In recent years multi-detector computed tomography (MDCT) has succeeded formal CTA in the early diagnosis of AMI in many centres. A recent study found MDCT to have equal sensitivity (93%) and superior specificity (100%) when compared to CTA. The diagnosis is based on a combination of the following signs seen on imaging: [8]

- Filling defect in the 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 considerable mortality and patients often have a poor physiological reserve with significant comorbidities. Initial management should follow established guidelines using an ABCDE approach. Administration of oxygen and rapid infusion of intravenous fluids will be essential components. 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 contra-indicated. Patients may require invasive monitoring of haemodynamic parameters by arterial line and a central venous catheter, which can be monitored in a high dependency area. A urinary catheter should be inserted to measure hourly urine output and monitor fluid balance.

After emergency resuscitation has taken place and the patient is stabilised, there is a dilemma as to the best course of action. Traditional teaching would advocate laparotomy and resection of infarcted bowel with or without revascularisation, however improved imaging and endovascular techniques mean this approach might not always be necessary and the patient could be revascularized using an endovascular technique and monitored in intensive care as opposed to going straight for a laparotomy. The choice of management will depend upon the resources and expertise available at individual centres and a combination of open surgery, endovascular surgery, and medical management may be adopted in a select number of patients.

There is some data to suggest that endovascular techniques are associated with better outcomes if the occlusion is thrombotic as opposed to embolic. However, open and endovascular surgery have similar outcomes if the occlusion is embolic [4].

Surgical management

If an open surgical approach is decided 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. Frankly ischaemic bowel appears grey or black and is foul-smelling. More subtle changes of ischaemia include loss of peristalsis and duskiness. If bowel appears salvageable, revascularisation ought to be considered as early as possible. This is preferably performed before bowel resection. However, there is no data to suggest whether revascularisation should definitely occur before or after bowel resection if it is required [4].

An SMA embolectomy may be performed. This involves a transverse arteriotomy across the SMA and passage of a 3-4 Fr Fogarty catheter to remove the thrombus. The arteriotomy is then closed and intra-operative Doppler used to assess SMA flow. 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 a reversed vein [9].

In order to assess intestinal availability the surgical team should wait for 20-30mins after revascularisation and cover the bowel in packs soaked in warm saline. Alternatively the bowel can be placed back into the abdominal cavity and the following factors taken into account:

- Peristalsis
- Colour
- Visible/palpable pulsations or satisfactory Doppler signals in mesenteric arcades

The extent of bowel resection required is determined by the degree of necrotic and non-viable bowel. It can be difficult to tell whether bowel is viable, but the use of Doppler and fluoroscein has been encouraged to assist with decision-making. Exteriorisation of the bowel ends as stomas may be considered following bowel resection in this situation (Figure 4a & 4b), to allow monitoring of mucosal perfusion. Occasionally it may be safest to staple to the ends of the bowel in an unstable patient and perform a relook laparotomy 24-48 hours later to determine if further bowel resection is required. Up to 50% of patients may require further resection at this stage [3]. A minimum of 70cm (ideally 1m) of small bowel is required for absorption to maintain life. Rarely the entire small bowel is affected, with only sparing of the proximal jejunum (as is frequently the case). This is likely to render the patient TPN dependent or requiring a small bowel transplant in suitable cases. In a patient with significant co-morbidities, unlikely to tolerate such a major resection, the decision to close the abdomen and palliate is usually more appropriate.

Endovascular management

Endovascular intervention should be attempted if the patient is stable and angiography highlights a suitable lesion. Endovascular techniques have been employed increasingly in the management of AMI [10]. Endovascular approaches include:

- Aspiration thrombectomy

- Thrombolysis
- Angioplasty and stenting

Endovascular treatment was associated with lower mortality than surgical revascularisation in a recent large study that analysed 23,744 patients (24.9% vs 39.3%; P=0.01). Furthermore, fewer patients in the endovascular group had bowel resections (14.4% vs 34.4%; P<0.001). A decision analysis model suggested that endovascular intervention is more cost effective for all age groups [11,12]. However, there is currently no Level 1 evidence comparing endovascular treatment with laparotomy.

Unfortunately, most patients require laparotomy as they have signs of bowel infarction and endovascular management alone cannot be performed [10]. More recently a combined approach has been advocated where both laparotomy and angiography can be performed in a hybrid theatre [9], however this is dependent on the centre's available resources.

Access to the SMA is usually via brachial or femoral approach. Aspiration thrombectomy has yielded promising results, however completion angiography should be performed and repeated aspirations may be required. If there is a distal embolisation, or incomplete aspiration, thrombolysis may be considered. Thrombolysis involves administration of recombinant tissue plasminogen activator (rtPA) at a rate of 0·5–1mg/h. An angiogram is performed every 12-24 hours to assess progress. The risk of bleeding during local thrombolysis is low and self-limiting. However, thrombolysis may be contra-indicated if there is risk of haemorrhage, or if laparotomy is required. Furthermore, in the acute situation there is not adequate time for thrombolysis to work and the risk of distal embolization exists.. Once aspiration, with or without thrombolysis, has achieved the

desired result any remaining occlusive or stenotic lesions may be treated with angioplasty and stenting [9].

Medical Management

Medical management is the most appropriate course of action in the management of NOMI, with an aim to correct the underlying cause of the ischaemia. Treatment should take place in an intensive care setting with intravenous fluid resuscitation, intravenous antibiotics and anticoagulation with heparin, if not contraindicated. Attempts should be made to reduce administration of exogenous catecholamines.

Medical management with a heparin infusion may also be suitable for those patients who are not fit enough to undergo a laparotomy, or for those who have very distal mesenteric trashing rendering open embolectomy or endovascular intervention fruitless.

PROGNOSIS

AMI carries significant mortality, showing a 30-day mortality rate of 32-81%, with most being over 60%. A study from the Mayo Clinic reporting a peri-operative mortality of 32%, showed long-term survival still remained very poor (32% at 3 years). An analysis of 827 patients from the American College of Surgeons Quality Improvement Database of patients undergoing bowel resection for acute mesenteric ischaemia (small intestine and colonic) showed the 30-day mortality rate was 28%. This lower mortality rate may be because the analysis didn't include patients who did not undergo bowel resection. The following factors are associated with an increased mortality risk:

- Pre-operative coma (85% 30-day mortality)

- Open Wound
- Pre-operative sepsis
- Dirty versus clean-contaminated case
- Age
- Low albumin
- Reduced preoperative functional status (i.e. dependent, in residential or nursing home)
- Pre-operative sepsis/septic shock
- Acute renal failure (30 day mortality 32%)
- ASA grade
- Recent (<6 months) myocardial infarction (30 day mortality 53%)

Less than 1% of patients received concomitant revascularisation. Morbidity was high and 50% of patients developed post-operative pulmonary complications. Following this the authors developed a risk calculator, available on-line, to predict peri-operative mortality and morbidity, (http://www.surgicalriskcalculator.com/ami-risk-calculator). Further markers for increased mortality risk include peritonitis, bowel necrosis, and TPN dependence.

CHRONIC MESENTERIC ISCHAEMIA

EPIDEMIOLOGY

Significant stenotic disease of the mesenteric arteries occurs in 6-17.5% of the population, with 1 in 6 patients over the age of 65 years being affected [3]. However, due to the extensive collateral blood supply between the coeliac axis, SMA, and IMA, only a few of these patients develop symptoms of mesenteric ischaemia (Figure 5). While asymptomatic disease has equal gender prevalence, the majority (70%) of patients with symptomatic chronic mesenteric ischaemia (CMI) are women [10].

AETIOLOGY AND PATHOLOGY

The good collateral blood supply between the coeliac axis, SMA, and IMV means that at least two of these visceral arteries need to be significantly narrowed or occluded to cause symptoms. Atherosclerosis accounts for the majority (95%) of causes of CMI (Figure 6a &b). Other causes include: Buerger's disease, fibromuscular dysplasia, aortic dissection, Behçet's disease, thromboangiitis obliterans, Takayasu's disease, Crohns and external compression of the coeliac axis. The latter is known as Median Arcuate Ligament Syndrome and is thought to be due to pressure exerted by the diaphragmatic crura on the coeliac axis.

PRESENTATION

CMI is characterised by an insidious onset and classically presents with postprandial pain, weight loss, and concurrent vascular disease. The postprandial abdominal pain is often referred to as "mesenteric angina" and starts 30 minutes to 3 hours after eating. Other

symptoms may include ‘food avoidance’, nausea, vomiting, diarrhoea, and/or constipation. The patient is often cachectic on examination and abdominal bruits may be heard.

It is important to identify and treat CMI early as approximately 20% of patients with symptoms of CMI go on to develop AMI. Malignancy and neoplastic lesions should be excluded before confirming a diagnosis of CMI [10].

INVESTIGATION

Duplex ultrasonography is a useful tool in the diagnosis of CMI as it is safe, fast and efficient. It is particularly good in CMI as this group of patients are thinner which makes visualisation of the visceral vessels easier. Peak systolic velocities of >200cm/sec in the SMA and >275cm/sec in the coeliac axis indicate >70% stenosis.

CT angiography is normally utilised alongside duplex scanning. Sensitivity and specificity of CMI diagnosis on CTA is >95%. CTA has the advantage that other pathology (e.g. malignancy) may be detected, which could account for the patient’s symptoms. Furthermore, as many patients present with unexplained weight loss most patients should also have an upper and lower gastrointestinal tract endoscopy to exclude malignancy or other pathology. Other experimental functional techniques have been employed to measure visceral mucosal perfusion (gastric tonometry, spectroscopic oximetry, MR flow) but are not in routine clinical use.

TREATMENT

Patients without explicit clinical manifestations can be managed conservatively, with a focus on secondary prevention therapy for atherosclerosis (antiplatelet and lipid lowering

therapy). These patients may also require nutritional assessment and support. However when surgical intervention is required, the two main treatment options for CMI involve either endovascular or open surgical techniques. Endovascular treatment involves percutaneous angioplasty and stenting of the affected vessel (Figure 7a & b). This is a good option in patients who are high risk for surgical revascularisation. Endovascular intervention for CMI has a high technical success rate, up to 97% in a study of 166 patients (over a 28 year period). Stenting had a higher success rate of (99.4%) when compared with angioplasty (86%, $P<0.01$). Immediate clinical improvement was seen in 88.2% of patients. Another recent study looking at endovascular intervention in 65 patients showed immediate symptomatic relief in 85% of patients. Primary patency rates at 1 year varied across both studies from 65% to 88%. Both groups of authors advocated using angioplasty with stenting rather than on its own. Peri-procedural heparin is given and patients are maintained on an antiplatelet agent following the procedure.

Surgical revascularisation involves midline laparotomy and exposure of the affected vessels at their origins from the aorta, followed by subsequent bypass construction. Aorto-coeliac and aorto-mesenteric bypass conduits usually extend from the suprarenal aorta, infrarenal aorta, or iliac artery to the relevant diseased visceral vessel (Figure 8 a & b). As vein is prone to kinking, prosthetic material is usually used to construct the bypass. However, a reversed vein should be used if there is a significant risk of graft infection. SMA revascularisation is associated with a mortality risk of 5-10%. Endarterectomy and re-implantation of the SMA are alternatives to bypass surgery.

There have been no randomised controlled trials comparing open surgery with endovascular surgery. In a UK study, bypass surgery was associated with a better patency rate than

endovascular intervention (81% vs. 54% 1-year primary patency) but increased morbidity (32% vs. 6%) and mortality (13% vs. 4%) [13]. A further recent study has found that while endovascular revascularisation is equally as safe as open surgery, patency is superior at mid-term follow-up with open surgical revascularisation [14].

ISCHAEMIC COLITIS

EPIDEMIOLOGY

The incidence of ischaemic colitis (IC) is 4.5-44/100,000 per year, but this figure may be lower that the true incidence due to underdiagnosis. The incidence has increased nearly 4-fold over the past 40 years [15] and increases with age. It is also diagnosed more frequently in females.

AETIOLOGY

Ischaemic colitis occurs when there is a transient, acute interruption in mesenteric blood flow. Risk factors for primary ischaemic colitis include chronic obstructive pulmonary disease (COPD), inflammatory bowel disease, and rheumatoid diseases. In these patients ischaemic colitis typically occurs at the splenic flexure in the “watershed” area between the inferior and superior mesenteric arteries.

Ischaemic colitis is also a well-recognised complication of abdominal aortic aneurysm (AAA) repair. The inferior mesenteric artery (IMA) is often chronically occluded during open AAA repair, and ligation is considered standard operative technique. Intraoperatively the IMA is observed for back-bleeding, brisk-back bleeding indicates good collateralisation and ligation

is usually of no consequence. Similarly, an occluded IMA does not need revascularisation. Poor collateralisation of the descending and sigmoid colon may be indicated by poor or trickle flow back-bleeding, and reimplantation of the IMA should then be considered at the time of aneurysm repair (Figure 9) as there is concern regarding the perfusion of the hindgut. Overall incidence of ischaemic colitis following aneurysm repair varies from 3-35%, with the highest incidence occurring in the emergency setting.

PRESENTATION

Ischaemic colitis has a variable presentation, which is dependent on the severity of ischaemia involved. Symptoms range from vague lower abdominal pain, with or without bloody diarrhoea, to an acute abdomen. “Failure to thrive” following aneurysm repair surgery should also raise clinical suspicion of colonic ischaemia.

INVESTIGATION

Endoscopy (flexible sigmoidoscopy / colonoscopy) combined with biopsy is the gold standard for the diagnosis of IC. Endoscopic examination is not usually possible in the acute setting and the diagnosis is largely made on clinical and radiological findings. In this situation contrast enhanced CT is the diagnostic method of choice. CT can pick up positive findings in 98% of cases [16]. CT appearances may be similar to infections colitis so this should be considered in the differential diagnosis. Inflammatory markers (white cell count and CRP) and lactate may also be raised, but do not have high specificity.

MANAGEMENT

The majority of cases of IC are managed medically. Laparotomy and bowel resection would be indicated with evidence of bowel necrosis, peritonitis and/or perforation. A Hartmann's procedure to resect the ischaemic bowel with formation of an end colostomy is usually required. Medical management consists of oxygen therapy, bowel rest (including a naso-gastric tube), fasting, parenteral nutrition, intravenous antibiotics, intravenous fluids, and correction of electrolyte imbalances. The National Institute for Health and Care Excellence (NICE) recommends prophylactic dose low molecular weight heparin in patients with ischaemic colitis, but do not advocate therapeutic anticoagulation [17]. Conservative management may be associated with the development of a late ischaemic stricture, which can cause obstructive symptoms. This can be dilated endoscopically or surgically resected where appropriate.

VENOUS INFARCTION

EPIDEMIOLOGY

Venous infarction due to superior mesenteric vein thrombosis (SMVT) was until recently thought to account for approximately 15% of cases of acute mesenteric ischaemia [10]. However, with improved recognition and diagnosis, this proportion has decreased to 10% [18]. The highest incidence occurs in the 70-79 years old age group accounting for 11.3/100,000 population years. Case series suggest that a younger population than this is affected (45-60 years), dependent on the aetiology.

AETIOLOGY

SMVT can be divided into primary and secondary thrombosis. No cause is found in up to 10% of cases. There is a predisposition to thrombosis in the remainder of cases. Thrombophilia exists in at least 50% of cases of venous thrombosis. Inherited conditions predisposing to SMVT include Factor V Leiden, Protein C or Protein S deficiency, Antithrombin III deficiency and Prothrombin gene sequence variation. Acquired causes include haematologic conditions (polycythaemia vera, myelofibrosis, thrombocythemia, antiphospholipid antibodies) and non-haematologic causes (malignancy, oral contraceptive pill) (4-5% of cases), pregnancy, nephrotic syndrome, hyperhomocysteinemia). Other predisposing factors include pathologies located in the abdomen (local factors) and include pancreatitis, inflammatory bowel disease, diverticulitis, peritonitis, appendicitis, intra-abdominal surgery, and abdominal trauma. Venous stasis also increases the risk of developing SMVT. Conditions that result in venous stasis include congestive splenomegaly, cirrhosis and congestive cardiac failure. Thrombosis of the IMV is uncommon, the reasons for which are unclear.

PRESENTATION

The presentation of MVT can be categorised as acute (sudden onset), sub-acute (over days to weeks) or chronic. The chronicity of symptoms can often make this condition more difficult to diagnose. Abdominal pain is the principal symptom, and often occurs with other similar features of AMI such as nausea, vomiting, diarrhoea or per-rectal bleeding. A large proportion of patients with acute mesenteric vein thrombosis are pyrexial (25-50%) whilst tachycardia is present in one in five of patients. Peritonitis will be evident with bowel

infarction with perforation. There may be a history of previous venous thrombo-embolic events in 20-40% patients.

INVESTIGATION

Routine laboratory tests are often not of benefit as they are often non-specific to venous infarction. Lactic acidosis is likely to be present in severe cases. As with AMI biochemical markers (including transaminases and amylase) may be elevated and an abdominal plain film is unlikely to be of benefit and can even be normal in 25% of cases. At least 50% of patients have a positive faecal occult blood test. CT in the portal venous phase is the mainstay of diagnosis with an accuracy of up to 95%. In a proportion of patients the diagnosis is made at laparotomy.

MANAGEMENT

As SMVT is a rare condition there haven't been any robust trials comparing different treatment modalities. Management can be divided into medical, surgical, and endovascular approaches and is based on treatment of venous thromboembolism elsewhere.

Medical Management

Medical management involves optimising analgesia, bowel rest, replacing intravascular volume, prophylactic antibiotics, and anticoagulation. Initially low molecular weight (LMWH) or UFH heparin is used with switch to an oral anticoagulant (e.g. warfarin, rivaroxaban) once the patient has improved and there is no indication for further invasive intervention. Anticoagulation is associated with gastro-intestinal bleeding (risk of <10%) [19]. However, anticoagulation should not be delayed unless there is an obvious contraindication.

Anticoagulation is effective in recanalization of splanchnic vein thrombosis and is achieved up to 45% of patients. This compares to a recanalization rate of 18% in those not treated with anticoagulation in a recent series of 121 patients. The duration of anticoagulation is decided on an individual basis but usually a six-month course is suggested, with long-term therapy to be decided at the end of that period.

Surgical Management

If the patient has an acute abdomen with peritonitis or CT reveals signs of transmural infarction, surgical intervention is indicated. SMV thrombectomy may be possible during laparotomy however, poor outcomes are reported. Infarcted bowel should be resected to healthy margins and re-anastomosed with a plan for a relook laparotomy in 24-48hours.

Endovascular Management

Endovascular options normally reserved for patients with a low risk of bowel infarction who deteriorate despite two to three days of optimal medical management. Endovascular techniques are often employed in conjunction which anticoagulation. Thrombolytic agents can be infused systemically or locally. Local infusion involves employing trans-hepatic or trans-jugular approaches to place a catheter in the thrombus for local administration of the thrombolytic agent. Thrombolysis into the SMA and direct aspiration thrombectomy have also been performed.

PROGNOSIS

The 30-day mortality has been reported at 20% in recent series with intestinal infarction being the predominant cause of death followed by advanced cancer and pulmonary

embolism. Thrombolysis has comparable 30-day mortality rates to surgery, but thrombolysed patients had a much shorter hospital stay (43 days vs 20 days) [20].

REFERENCES

1. Cudnik MT, Darbha S, Jones J, Macedo J, Stockton SW, Hiestand BC. The Diagnosis of Acute Mesenteric Ischemia: A Systematic Review and Meta-analysis. Academic Emergency Medicine 2013; 20 (11): 1087–1100.

2. Clair D and Beach J. Mesenteric Ischemia. NEJM. 2016;374:959-968.

3. Wyers M. Acute Mesenteric Ischemia: Diagnostic Approach and Surgical Treatment. Semin Vasc Surg 2010;23:9-20.

4. Bjorck M, et al. Editor's Choice – Management of the Diseases of Mesenteric Arteries and Veins. European Journal of Vascular and Endovascular Surgery. 2017;53(4):460-510.

5. Studer P, Vaucher A, Candinas D, Schnüriger B. The Value of Serial Serum Lactate Measurements in Predicting the Extent of Ischemic Bowel and Outcome of Patients Suffering Acute Mesenteric Ischemia. J Gastrointest Surg. 2015;19(4):751-755.

6. Bala M, Kashuk J, Moore EE, et al. Acute mesenteric ischaemia: guidelines of the World Society of Emergency Surgery. World Journal of Emergency Surgery. 2017;12:38.

7. Yukaya T, Saeki H, Taketani K, et al. Clinical outcomes and prognostic factors after surgery for non-occlusive mesenteric ischemia: a multicenter study. J Gastrointest Surg. 2014 Sep;18(9):1642-7.

8. Barrett T, Upponi S, Benaglia T, Tasker AD. Multidetector CT findings in patients with mesenteric ischaemia following cardiopulmonary bypass surgery. Br J Radiol 2013;86(1030):20130277.

9. Acosta S, Björck M. Modern treatment of acute mesenteric ischaemia. Br J Surg. 2014 Jan;101(1):e100-8.

10. Upponi S, Harvey JJ, Uberoi R, Ganeshan A. The role of radiology in the diagnosis and treatment of mesenteric ischaemia. Postgrad Med J. 2013 Mar;89(1049):165-72.

11. Beaulieu RJ, Arnaoutakis KD, Abularrage CJ, Efron DT, Schneider E, Black JH 3rd. Comparison of open and endovascular treatment of acute mesenteric ischemia. J Vasc Surg. 2014 Jan;59(1):159-64.

12. Hogendoorn W, Hunink MG, Schlösser FJ, Moll FL, Muhs BE, Sumpio BE. A comparison of open and endovascular revascularization for chronic mesenteric ischemia in a clinical decision model. J Vasc Surg. 2014 Sep;60(3):715-25.

13. Rawat N, Gibbons C, on behalf of the JVRG. Surgical or Endovascular Treatment for Chronic Mesenteric Ischaemia: A Multicenter Study. Ann Vasc Surg. 2010;24:935-45.

14. Arya S, Kingman S, Knepper JP, Eliason JL, Henke PK, Rectenwald JE. Open Mesenteric Interventions Are Equally Safe as Endovascular Interventions and Offer Better Midterm Patency for Chronic Mesenteric Ischemia. Ann Vasc Surg. 2016;30:219-226.

15. Yadav S, Dave M, Edakkanambeth Varayil J, Harmsen WS, Tremaine WJ, Zinsmeister AR, Sweetser SR, Melton LJ 3rd, Sandborn WJ, Loftus EV Jr. A population-based study of incidence, risk factors, clinical spectrum, and outcomes of ischemic colitis. Clin Gastroenterol Hepatol. 2015;13(4):731-738.

16. Cruz C, Abujudeh HH, Nazarian RM, Thrall JH. Ischemic colitis: spectrum of CT findings, sites of involvement and severity. Emergency Radiology. 2015;22(4):357-365.

17. National Institute for Health and Care Excellence. Venous thromboembolism: reducing the risk of venous thromboembolism (deep vein thrombosis and pulmonary embolism) in patients admitted to hospital. (Clinical guideline 92.) 2010. www.nice.org.uk/guidance/CG92.

18. Tilsed JVT, Casamassima A, Kurihara H, et al. ESTES guidelines:acute mesenteric ischaemia. European Journal of Trauma and Emergency Surgery. 2016;42(2):253-270.

19. Ageno W, Riva N, Schulman S, Beyer-Westendorf J, Bang SM, Senzolo M, Grandone E, Pasca S, Di Minno MND, Duce R, Malato A, Santoro R, Poli D, Verhamme P, Martinelli I, Kamphuisen P, Oh D, D'Amico E, Becattini C, De Stefano V, Vidili G, Vaccarino A, Nardo B, Di Nisio M, Dentali F. Long-term Clinical Outcomes of Splanchnic Vein Thrombosis. JAMA Intern Med. 2015;175(9):1474-1480.

20. Yang SF, Liu BC, Ding WW, He CS, Wu XJ, Li JS. Initial transcatheter thrombolysis for acute superior mesenteric venous thrombosis. World J Gastroenterol. 2014;20(18):5483-5492.

LEGENDS TO FIGURES

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

Figure 3. Small bowel ischaemia and necrosis

Figure 4. (a) Resected specimen of ischaemic bowel involving distal ileum and proximal colon; (b) Defunctioning transverse colostomy and terminal ileostomy

Figure 5. CTA reconstruction showing severe atherosclerosis at the origins of the SMA and coeliac axis (arrowed). There is good collateral supply between the SMA and IMA via the "wandering artery" of Drummond (arrowed)

Figure 6a & b. CTA with reconstruction, demonstrating severe disease at origin of coeliac axis and occlusion of proximal SMA

Figures 7a & b. Selective mesenteric angiography demonstrating SMA stenosis pre-dilation (a) and post-angioplasty and stent placement (b)

Figure 8a & b. Aorto-SMA bypass with prosthetic (PTFE) graft, employed to reduce risk of kinking

Figure 9. Reimplantation of IMA into aortic graft following aneurysm repair