Review

Modern treatment of acute mesenteric ischaemia

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Background: **Diagnosis of acute mesenteric ischaemia in the early stages is now possible with modern computed tomography (CT), using intravenous contrast enhancement and imaging in the arterial and/or portal venous phase. The availability of CT around the clock means that more patients with acute mesenteric ischaemia may be treated with urgent intestinal revascularization.**

Methods: **This was a review of modern treatment strategies for acute mesenteric ischaemia.**

Results: **Endovascular therapy has become an important alternative, especially in patients with acute thrombotic superior mesenteric artery (SMA) occlusion, where the occlusive lesion can be recanalized either antegradely from the femoral or brachial artery, or retrogradely from an exposed SMA after laparotomy, and stented. Aspiration embolectomy, thrombolysis and open surgical embolectomy, followed by on-table angiography, are the treatment options for embolic SMA occlusion. Endovascular therapy may be an option in the few patients with mesenteric venous thrombosis who do not respond to anticoagulation therapy. Laparotomy is needed to evaluate the extent and severity of visceral organ ischaemia, which is treated according to the principles of damage control surgery.**

Conclusion: **Modern treatment of acute mesenteric ischaemia involves a specialized approach that considers surgical and, increasingly, endovascular options for best outcomes.**

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Introduction

Recovery following resection of infarcted intestine secondary to mesenteric vessel occlusion was first described in 1895[1]. The first successful emergency superior mesenteric artery (SMA) embolectomy was undertaken in 1951[2], SMA thrombendarterectomy in 1958[3], and aortomesenteric bypass in 1973[4]. Intra-arterial thrombolysis for SMA embolus, using a combination of streptokinase and heparin, was achieved successfully in 1979[5]. The evolution and availability of high-resolution computed tomography (CT) round the clock has had an impact on the early diagnosis of mesenteric vessel occlusion[6]. Patients suspected to have mesenteric vessel occlusion need CT with intravenous contrast enhancement[7,8], without fear of contrast-induced renal failure[9], to improve the chance of survival. An increasing number of patients may therefore be diagnosed in time for intestinal revascularization. In current practice, collaboration between general and vascular surgeons is of great importance. The rapid development of endovascular techniques has made hybrid endovascular or surgical treatment feasible for acute mesenteric ischaemia (AMI). When applicable, the principles of damage control surgery should follow mesenteric revascularization[10]. This review outlines the various treatment options in the modern treatment of AMI.

Classification

The classification of AMI is shown in *Fig. 1*. Hypoperfusion syndromes belong to the same category as non-occlusive mesenteric ischaemia (NOMI).

Epidemiology

Unfortunately, contemporary population-based studies on the epidemiology of AMI are lacking owing to low autopsy rates and reporting only of patients who have surgery. The overall incidence of AMI between 1970 and 1982 in the city of Malmö, Sweden, diagnosed at either autopsy or operation, was 12·9 per 100 000 person-years. The autopsy rate in the population was 87 per cent. Among 402 patients, 270 (67.2 per cent) had thromboembolic SMA occlusion, 63 (15·7 per cent) mesenteric venous thrombosis (MVT), 62 (15·4 per cent) NOMI and seven (1·7 per cent) had indeterminate aetiology[11]. The embolus to thrombus ratio was 1·4 : 1 among the 213 patients with acute SMA occlusion diagnosed at autopsy[12]. Acute SMA

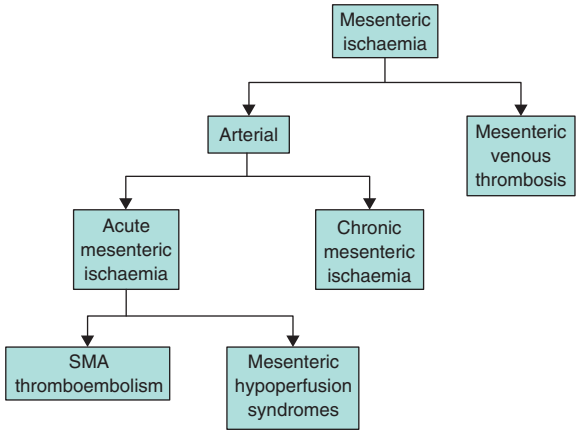


Fig. 1 Classification of mesenteric ischaemia. SMA, superior mesenteric artery

occlusion was more common than ruptured abdominal aortic aneurysm (AAA)[11].

Diagnosis

A high index of suspicion and awareness among physicians seeing patients who may have AMI is not enough to improve outcomes. The typical clinical triad in an elderly patient is severe abdominal pain but minimal findings at examination (pain out of proportion), bowel emptying and a source of embolus, although even among patients with acute embolic SMA occlusion this is not a consistent finding. Acute thrombotic SMA occlusion and MVT are difficult to diagnose at first evaluation. The clinical presentation of patients with AMI is summarized in *Table 1*. No plasma marker is yet sufficiently accurate to be an early diagnostic aid[13]. Acute on chronic mesenteric ischaemia, with an insidious clinical course, may show signs of ischaemia in the duodenum on endoscopy and in the right colon (*Fig. 2*), a fact that seems to be unknown to many physicians[14]. The major breakthrough has been the evolution of high-resolution CT[6]. Rapid reconstruction of images in the sagittal, coronal and transverse planes can be diagnostic (*Fig. 3*). Embolic occlusion often appears as an oval-shaped clot surrounded by contrast in a non-calcified arterial segment located in the middle and distal part of the main stem of the SMA. Thrombotic occlusion usually appears as a clot superimposed on a heavily calcified occlusive lesion at the ostium of the SMA (*Fig. 3*).

Treatment of acute superior mesenteric artery occlusion

Optimal treatment may include open or endovascular surgery and patients are best treated in a vascular centre with a hybrid operating room. From preoperative clinical evaluation and CT, it can be determined whether the patient has peritonitis or not, and whether the occlusion is embolic or thrombotic. Laparotomy is indicated if there is peritonitis, unless a palliative approach has been decided upon. This aims to assess the extent and severity of

Table 1 Clinical presentation at admission to hospital in patients with acute mesenteric ischaemia

	SMA embolus	SMA thrombus	Venous thrombus
History			
Age ≥ 80 years	++	+	–
Age < 50 years	–	–	+
Women > men	+	+	+/–
Atrial fibrillation	++	–	–
Previous myocardial infarction	++	+	–
Stroke	+	++	–
Previous arterial embolism/source of embolus	++	–	–
Previous symptoms of chronic mesenteric ischaemia	–	++	–
Previous deep venous thrombosis or pulmonary embolism	–	–	++
Activated protein C resistance	–	–	++
Pancreatitis or pancreatic cancer	–	–	++
Liver cirrhosis/portal hypertension	–	–	++
Symptoms			
Sudden onset	++	+/–	–
Insidious onset	–	+	+
Abdominal pain	++	+	+
Vomiting	++	++	+
Diarrhoea	+	+	+
Bloody stools	+	+	+/–
Synchronous embolism	++	–	–

++, Factor likely to be present; +, factor perhaps present; –, factor unlikely to be present.

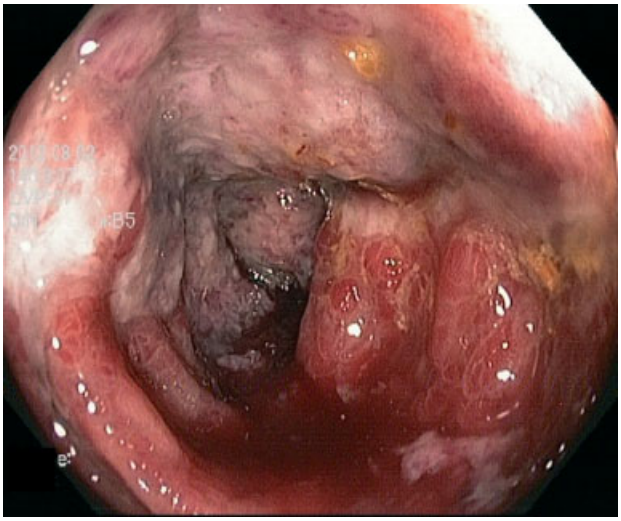


Fig. 2 Colonoscopic image from the ascending colon showing mucosal ulceration, haemorrhage, oedema and fragility consistent with colonic ischaemia

intestinal ischaemia: colour of the intestines, dilatation and peristaltic motion of the bowel, visible pulsations in the mesenteric arcade arteries, and bleeding from cut surfaces are most important to assess. Laparotomy, rather than laparoscopy, is usually safer and quicker to evaluate the visceral organs. Extensive intestinal paralysis with dilated bowel loops may be impossible to evaluate at laparoscopy, even by an expert. Elderly patients with complete transmural infarction of the small bowel up to the mid-transverse colon would need extensive bowel resection that would lead to short bowel syndrome and increased morbidity; this procedure may be inappropriate in some patients. In the event of perforation, the affected intestinal segment is resected. The ends may be sealed with staples, leaving anastomosis or stoma formation at second-look stage.

Acute mesenteric arterial revascularization

Acute mesenteric arterial revascularization is done preferably before any bowel surgery. If no vascular surgeon is available, it may be preferable to resect necrotic bowel, close the abdomen and transport the patient to a vascular centre for revascularization. According to the national Swedish registry of vascular procedures, Swedvasc[15], there has been a steady increase in mesenteric revascularizations for AMI since 2004. In 2009, endovascular treatment surpassed open surgery: 29 endovascular *versus* 24 open revascularizations. In contrast, this shift in treatment modality has not taken place in the North America[16]. In Swedvasc[15], the 30-day mortality rate was similar after open *versus* endovascular surgery for embolic occlusions

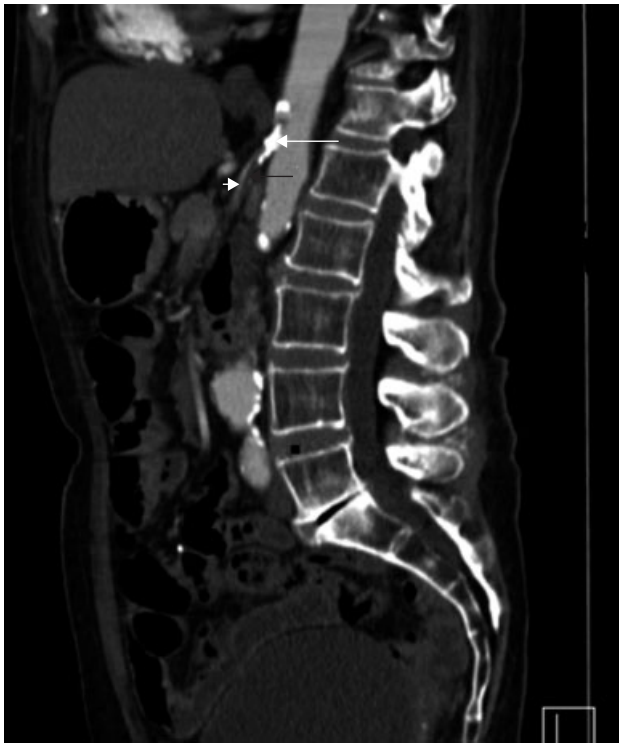


Fig. 3 Computed tomography angiography with sagittal reconstruction showing a heavily calcified occlusive lesion at the ostium of the superior mesenteric artery (white arrow), thrombotic lesion superimposed on the atherosclerosis at the ostium (black arrow), and contrast filling in a stenotic part of the superior mesenteric artery (arrowhead)

(37 *versus* 33 per cent), whereas the mortality rate was significantly higher after open than endovascular treatment for thrombotic occlusions (56 *versus* 23 per cent). Of note, no patient had completion angiography after open surgical treatment, whereas completion angiography is part of the procedure after endovascular surgery. There may have been differences in disease severity between the treatment groups, but it remains possible that the endovascular approach is better for thrombotic occlusions in elderly, fragile patients. There is rarely any indication for revascularization of both the SMA and the coeliac trunk; SMA revascularization is clearly more important. An active treatment algorithm is proposed in *Fig. 4*.

Open superior mesenteric artery embolectomy

Open SMA embolectomy remains a good treatment option[17]. When laparotomy has been performed in a patient with peritonitis, exposure of the SMA and balloon embolectomy, with a 3- or 4-Fr Fogarty catheter, through

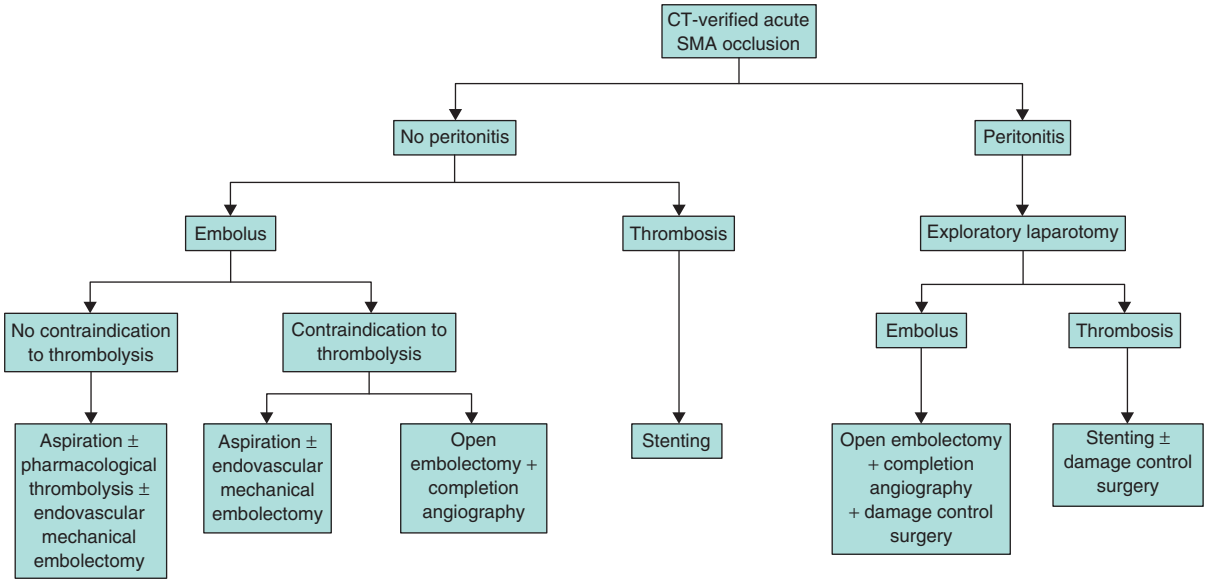


Fig. 4 Surgical algorithm for management of patients with acute superior mesenteric artery (SMA) occlusion. CT, computed tomography

a transverse arteriotomy is indicated. The result should be checked by ultrosonic transit-time flow measurement, but completion angiography of the SMA with anteroposterior and lateral views is optimal. Stenosis and dissection at the arteriotomy closure site, residual peripheral embolus in arterial branches not cleared, and venous return to the portal vein can all be assessed formally.

Endovascular therapeutic options

Access to the superior mesenteric artery

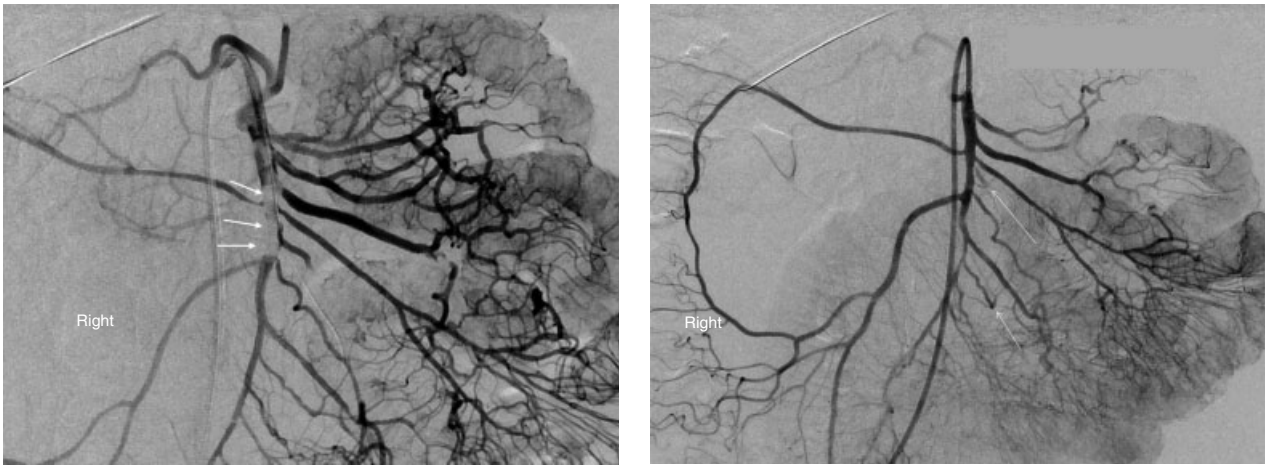
The SMA can be reached via the femoral and brachial routes, although sometimes local exposure of the SMA in the abdomen is also needed. Brachial access may be preferable if there is a sharp downward angle between the aorta and the SMA, or if the ostium of the SMA is calcified. Passage of wires, catheters and introducers may cause dissection of the SMA. If an antegrade approach from the femoral or brachial artery fails, a retrograde approach through the exposed SMA after laparotomy is possible[18].

Aspiration superior mesenteric artery embolectomy

Endovascular aspiration embolectomy is a treatment option in patients without peritonitis[19–21]. The SMA is cannulated as above and catheterized using a reverse-curve catheter and a hydrophilic 0·035-inch guidewire, which is passed into the ileocolic branch of the SMA. The wire is then replaced with a stiffer, Jindo or Rosen, wire to achieve stability. With the wire in place, typically a 7-Fr, 45-cm introducer with a removable hub (Destination®; Terumo, Tokyo, Japan) is placed proximal to the embolus in the SMA. Inside this, a 6-Fr guiding catheter is introduced into the clot, which is aspirated with a 20-ml syringe as the introducer is withdrawn. The hub of the introducer is removed to allow clearance of residual clots. Angiography is performed, usually followed by repeated aspirations (*Fig. 5*). An alternative is to use an over-the-wire double-lumen aspiration catheter such as the Export® (Medtronic, Minneapolis, Minnesota, USA), which may allow removal of smaller peripheral clots.

Local superior mesenteric artery thrombolysis

In cases of incomplete aspiration embolectomy or distal embolization, local thrombolysis is a viable treatment alternative in patients without peritonitis[19–21]. With the introducer placed in the proximal SMA, a multiple side-hole catheter delivering drugs over 10 cm, or a 4-Fr end-hole catheter, is advanced within or to the embolus, respectively. Local thrombolysis is achieved by administration of recombinant tissue plasminogen activator at a rate of 0·5–1 mg/h, checking progress angiographically once or twice per day. Bleeding complications during local thrombolysis are few and self-limiting; in a recent report[21], bleeding from the gastrointestinal tract occurred in only one of 34 procedures. Small peripheral residual emboli



a Embolus in SMA **b** After local aspiration and nitroglycerine injection

Fig. 5 Successful superior mesenteric artery (SMA) aspiration embolectomy from the common femoral artery. **a** Embolus in the main trunk of the SMA (arrows). **b** Embolus cleared after repeated aspirations. Residual embolic occlusive fragments are left in the proximal part of a few arterial branches to the ileum (arrows) with good peripheral collateral flow in the mesenteric arterial arcades

can be treated conservatively with heparin anticoagulation as the marginal arteries in the mesentery may provide sufficient collateral circulation to the affected intestinal segment[19]. Only 38 per cent of patients needed to undergo check laparotomy after local thrombolysis[21].

Antegrade recanalization and stenting of the superior mesenteric artery

Treatment of underlying stenotic or occlusive lesions is most often achieved during the same procedure, after removal of a thrombotic clot by aspiration or thrombolysis[22]. The sequence of endovascular intervention *versus* exploratory laparotomy depends on the clinical state of the patient (*Fig. 4*). If femoral access fails, an attempt to cross the occlusive lesion using a brachial approach with a 4-Fr catheter (Headhunter®; Terumo) may be successful. Occasionally, a 0·014-inch coronary guidewire is needed, followed by dilatation with a 1·5-mm balloon. When eventually a stable 0·035-inch wire has been placed in the ileocolic artery, an introducer is advanced past the atherosclerotic lesion. A balloon-expandable stent is chosen as these stents have better properties than self-expanding stents to maintain lumen diameter after stent deployment. The balloon-expandable stent is placed at the treatment site, followed by retraction of the protective introducer sheath, thus exposing the stent. The hard, calcified ostial lesion is then treated with a 7–8-mm diameter stent. Unfavourable artery angulation or a potential risk of arterial dissection at the distal end of the stent is treated by extension with a self-expanding stent into the middle SMA.

Results after stenting are checked by angiography, as well as pressure measurement. If there is residual pressure gradient across the stent exceeding 10 mmHg, additional angioplasty and/or stenting is performed. One of the most feared complications is dissection from the SMA ostium into the peripheral branches of the SMA, worsening the intestinal circulation further[19]. This complication can be managed at laparotomy by exposure of a peripheral branch in the arterial mesentery, with puncture into the true arterial lumen. The guidewire then has a better chance of following the true lumen into the aorta, establishing through-and-through access with the femoral artery. The dissection is then stented, sometimes into peripheral branches beyond the main trunk of the SMA.

Retrograde recanalization and stenting of the superior mesenteric artery

If brachial access fails, laparotomy and exposure of the SMA is performed for retrograde SMA recanalization and stenting[23,24]. This approach offers the opportunity to inspect the abdominal viscera, to have distal control of the SMA, and to avoid bypass surgery in the setting of necrotic bowel. The SMA is exposed at the junction of the mesocolon and the small bowel mesentery. A puncture is made in the vessel in its main trunk with a micropuncture needle; the occlusion is often recanalized easily with a 0·018-mm guidewire into the aorta. The SMA is clamped distally to avoid distal embolization if there is fresh thrombus at the occlusion site. The proximal SMA lesion is then crossed with a stiff, braided 4-Fr catheter, exchanging

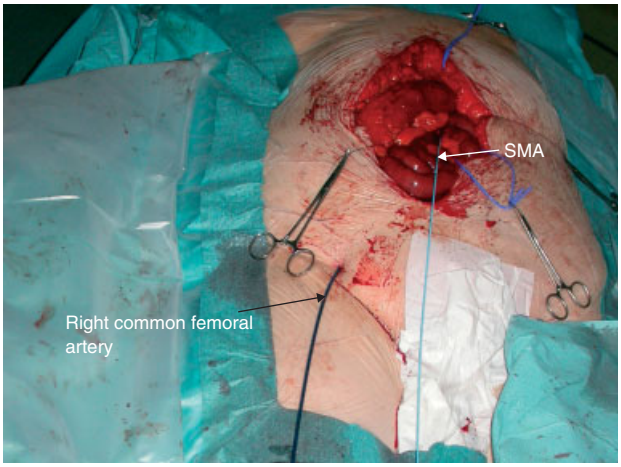


Fig. 6 Through-and-through access. A guidewire runs inside a catheter (white arrow) from the distal superior mesenteric artery (SMA) through the inside of the introducer (black arrow) via the right common femoral artery

for a 260-cm long 0·035-inch hydrophilic guidewire. The wire is snared in the aorta using a snare passed through a brachial or femoral access and then brought out, creating through-and-through access (*Fig. 6*). A small transverse arteriotomy is then done at the level of the puncture and an over-the-wire Fogarty balloon is passed into the aorta if thrombectomy is necessary. Thrombectomy is performed over the wire and the SMA inflow evaluated. If thrombectomy is not necessary, no arteriotomy is required. With slight traction on the wire, a 6–7-Fr introducer, Flexor® (Cook Medical, Bloomington, Indiana, USA) or Destination®, is then placed antegradely in the SMA over the through-and-through wire. An appropriate stent can then be placed across the lesion. The access puncture from the 4-Fr catheter in the SMA is treated by manual compression or suture. Antegrade stenting is better than retrograde stenting, because the procedure can be performed in a familiar manner with standard devices without exposing the operators to a higher dose of radiation.

Outcomes after open *versus* endovascular revascularization for AMI

No randomized clinical trials exist, making it difficult to compare outcomes, as there are many potential confounders. Four non-randomized studies[15,16,22,25] have compared open *versus* endovascular revascularization for AMI. One retrospective single-centre experience[16] showed no difference in mortality between the two treatments modalities, whereas the other single-centre study[22] showed lower bowel morbidity and lower mortality after endovascular therapy for acute thrombotic occlusions compared with open surgery. The other two multicentre studies are nationwide reports[15,25]. These studies showed a lower frequency of bowel resection, and lower short-term[15,25] and long-term[15] death rates after endovascular compared with open surgical therapy for acute thrombotic occlusion.

Treatment of mesenteric venous thrombosis

Patients with MVT are diagnosed on CT with intravenous contrast enhancement and imaging in the portal phase, or at exploratory laparotomy. There is a clear trend towards a higher proportion being diagnosed at CT[8]. Therapeutic anticoagulation with a continuous infusion of unfractionated heparin is used for patients treated without surgery as well those diagnosed at operation, using standard monitoring protocols. If necessary, the infusion can be stopped or protamine given to reverse the anticoagulation if urgent repeat laparotomy or second-look laparotomy is indicated.

In one series[8], CT in the portal venous phase was diagnostic in all 20 patients investigated, and conservative management was possible in 19. Hence, there is seldom need for invasive vascular treatment if the diagnosis is made on CT. An overall 30-day survival rate of 80 per cent has been reported[26]. The prognosis after hospital discharge depends on the underlying cause of MVT, and patients with cancer have the poorest survival[26].

Endovascular treatment

There are no studies with comparative data, to help establish the indication for endovascular treatment of MVT. Few patients deteriorate during medical treatment[26]; endovascular treatment might be an option for them. A number of endovascular procedures for the treatment of MVT have been developed in recent years, including percutaneous transjugular intrahepatic portosystemic shunting (TIPS) with mechanical aspiration thrombectomy[27] and direct thrombolysis[28,29], percutaneous transhepatic mechanical thrombectomy[30] (for case report see *Appendix S1*, supporting information), percutaneous transhepatic thrombolysis[31,32], thrombolysis via the SMA[33], and thrombolysis via a surgically placed mesenteric vein catheter[34]. Rapid thrombus removal or dissolution can be achieved by these techniques, especially after TIPS and stent placement to create a low-pressure run-off[35]. Mechanical thrombectomy is performed using a variety of thrombectomy devices and is most effective in cases of acute rather than chronic thrombus. Indirect thrombolytic therapy via the SMA is less effective and more time-consuming, may require longer infusion times and higher doses of

thrombolytic agent, and is also associated with an increased risk of bleeding. Balloon angioplasty is an alternative technique for clot fragmentation in cases of refractory thrombus and fixed venous stenosis. Aspiration thrombectomy is performed with a stiff, large-diameter (at least 8 Fr), angled catheter connected to a Luer-Lok™ syringe (Bluebird Medical, Göteborg, Sweden) to create a vacuum effect[28].

Endovascular techniques improve survival, increase patency of the portomesenteric veins, with lower rates of portal hypertension[32], and have low complication rates, avoiding bowel resection in selected patients[32]. In a series[33] of 16 patients with MVT treated by local thrombolysis, one had complete lysis, 11 had partial lysis and four had no lysis. Local thrombolysis was associated with bleeding complications in 60 per cent of patients, including intra-abdominal bleeding, bleeding from the access site, perihepatic haematoma, nosebleed and haematuria[33]. Accumulation of blood from the portal vein in the right pleural space, causing right-sided haemothorax, has also been reported during percutaneous transhepatic thrombectomy and thrombolysis[36], as have deaths from gastrointestinal haemorrhage and sepsis[33,36].

Medical treatment after acute mesenteric vessel occlusion

Patients who survive after acute mesenteric vascular occlusion need long-term medical treatment. After thrombotic arterial occlusion, patients should have best medical therapy against atherosclerosis, including an antiplatelet agent and a statin[14]. In case of embolic arterial occlusion, lifelong warfarin or an alternative is indicated. Patients with MVT also receive anticoagulation for at least 6 months, or lifelong, depending on the underlying cause[26].

Radiological follow-up

Patients who have a stent inserted in the SMA need to be followed regularly by either duplex imaging or CT angiography owing to the risk of restenosis and the need for reintervention to prevent the serious consequences of stent occlusion[14].

Hypoperfusion syndromes

Occlusive arterial disease is a more common cause of bowel gangrene than non-occlusive hypoperfusion, yet these syndromes are important to recognize. NOMI is a well established clinical condition affecting patients with low cardiac output, associated with the use of digitalis and/or other vasoactive drugs. Modern pharmacotherapy may have decreased the frequency of this condition, but a new clinical situation has occurred with the use of cardiac pump devices, resulting in better survival among patients with low cardiac output, but paradoxically a higher risk of NOMI. There are no modern epidemiological data on this condition, but NOMI was as fatal as MVT and about one-fifth as deadly as SMA occlusion in a reported cohort from Malmö[11,37]. An interesting finding was that 40 per cent of the patients with NOMI had an SMA stenosis, a lesion potentially treatable with stenting. Patients with NOMI are sometimes difficult to transport to radiology or hybrid suites, in particular if they are connected to cardiac pump devices. If at all possible, however, angiography should be performed to rule out this treatable condition. Screening in critical care can be done with duplex ultrasonography in some patients[38].

Aortic dissection of Stanford type A or B can be complicated by mesenteric hypoperfusion, either by compression of the true lumen creating a general hypoperfusion of the lower body, which is the most common situation, or by dissection propagating into the SMA[39]. The primary treatment is to perform an open repair with prosthetic graft of the ascending aorta (type A) or a thoracic endovascular aortic repair covering the entry tear (type B), followed by stenting of the SMA and exploratory laparotomy, when required.

A third hypoperfusion syndrome that can complicate trauma, burn injury, pancreatitis and aortic aneurysm repair, among others, is the intra-abdominal hypertension (IAH) that can deteriorate into abdominal compartment syndrome (ACS). The condition is common after ruptured AAA repair; in this clinical situation a relationship between IAH and colonic ischaemia has been established[40]. In a recent multicentre study[41], open abdomen treatment was used after approximately 3 per cent of ruptured AAA repairs, compared with less than 1 per cent after elective AAA repair. Definitions and treatment guidelines[42] regarding this condition were updated recently using the evidence-based Grades of Recommendation, Assessment, Development and Evaluation (GRADE) methodology. Treatment of IAH consists of proactive early non-surgical treatment and, whenever necessary, open abdomen treatment[43].

In the aftermath of arterial or venous occlusive disease, when the patient is hypotensive and has lost part of their bowel, an active treatment of even lesser degrees of IAH may save life and maximize perfusion of the remaining intestinal tract. Thus, open abdomen treatment should be considered after treatment of AMI. Although it has been shown that intestinal ischaemia is a major risk factor for development of enteroatmospheric fistula during open abdomen treatment[44], closing a tense abdomen is not a good alternative.

Damage control surgery

Laparotomy after mesenteric revascularization serves to evaluate the possible damage to the visceral organs. Bowel resection and organ removal as a result of clear transmural and gallbladder necrosis respectively are carried out according to the principles of damage control surgery[45–47]. Bowel resections are performed with staples, leaving the creation of anastomoses or stomas until the second- or third-look laparotomy (for case report see *Appendix S1*, supporting information). The abdominal wall can be left unsutured when repeat laparotomy is planned. In this situation, skin-only closure or temporary abdominal closure with an abdominal VAC® dressing (Kinetic Concepts, San Antonio, Texas, USA) may be applied.

Disclosure

The authors declare no conflict of interest.

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Supporting information

Additional supporting information may be found in the online version of this article:

Appendix S1 Case reports (Word document)