Mesenteric ischemia

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Purpose of review
Diagnosis of acute mesenteric ischemia 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 ischemia may be treated with urgent intestinal revascularization.

Recent findings
The establishment of a hybrid operation room is most important to be able to perform explorative laparotomy for evaluation of the extent of mesenteric ischemia and successful intestinal revascularization. Endovascular recanalization and stenting has become an important alternative, especially in patients with both acute and chronic thrombotic superior mesenteric artery (SMA) occlusion. 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. The concept of damage-control surgery is recommended after intestinal revascularization.

Summary
Intestinal revascularization in patients with arterial occlusive mesenteric ischemia reduces bowel morbidity and mortality. Observational studies report that both endovascular and open vascular therapy options are effective, but endovascular technique may be preferred in these often elderly and fragile patients.

Keywords
acute mesenteric ischemia, endovascular treatment, hybrid revascularization, peritonitis, superior mesenteric artery occlusion

INTRODUCTION

It is important to distinguish the four different entities in mesenteric ischemia, as they differ in epidemiology, risk factors, clinical presentation, operative approach and prognosis: embolic and thrombotic occlusion of the superior mesenteric artery (SMA), nonocclusive mesenteric ischemia (NOMI) and mesenteric venous thrombosis (MVT).

Classification

The classification of mesenteric ischemia is shown in Fig. 1.

Epidemiology

The overall population-based incidence of acute mesenteric ischemia (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%. Among 402 patients, 67% had thromboembolic SMA occlusion, 16% MVT, 15% NOMI and 2% had indeterminate cause [1]. The embolus to thrombus ratio was 1.4 : 1 among the 213 patients with acute SMA occlusion diagnosed at autopsy [2]. Acute SMA occlusion was more common than ruptured abdominal aortic aneurysm [1]. Incidence of chronic mesenteric ischemia (CMI) and NOMI is unknown.

The frequency of AMI among patients with acute abdomen varies from 2.1% in suspected appendicitis [3], 17.7% in emergency laparotomies [4] to 31.0% in damage-control laparotomies in nontrauma patients [5].

Pathophysiology

In AMI, whatever the cause, the infarction starts from the mucosa outward. Acute SMA occlusion,

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KEY POINTS

- CT with intravenous contrast enhancement is a prerequisite for early diagnosis of acute mesenteric ischemia and successful intestinal revascularization.
- Endovascular therapy has become an increasingly important treatment option in acute thrombotic and chronic mesenteric ischemia.
- Anticoagulation therapy alone with unfractionated heparin has become standard therapy after diagnosis of mesenteric venous thrombosis at CT venography.
- Patients with mesenteric venous thrombosis should be blood screened for inherited and acquired thrombophilic disorders.

in contrast to low flow states and left-sided colonic ischemia, causes a more significant reduction in blood flow with rapid development of extensive intestinal infarction [6].

ARTERIAL MESENTERIC ISCHEMIA

To distinguish occlusive mesenteric arterial ischemia from NOMI, or thrombotic from embolic SMA occlusion, may be very difficult or impossible in some cases at clinical presentation or even at laparotomy.

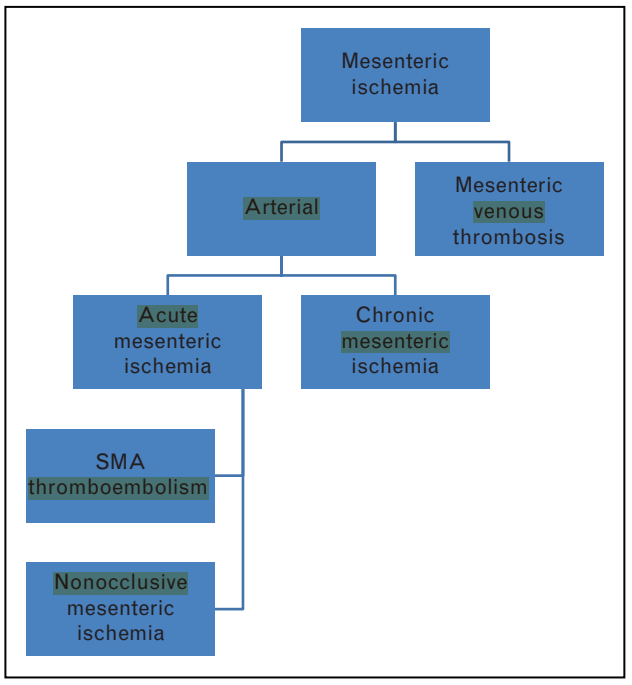

FIGURE 1. Classification of mesenteric ischemia.

Acute thrombotic occlusion of the superior mesenteric artery

There exists a collateral arterial network between the celiac trunk, SMA, inferior mesenteric artery and internal iliac arteries, which may become increasingly important and enlarged to compensate for the decreased visceral perfusion after gradual development of atherosclerotic occlusion or stenosis of the celiac trunk and/or SMA. Narrowing of the SMA has the greatest impact on the development of mesenteric ischemia. Thrombosis occurs at areas of severe atherosclerotic narrowing, most often at the origin of the SMA [2]. Dehydration, low cardiac output and hypercoagulable state are major contributing factors to thrombosis. In case of thrombotic occlusion at the origin of the SMA, ischemia usually develops from the proximal jejunum to the mid transverse colon.

Embolic occlusion of the superior mesenteric artery

Like most peripheral arterial emboli, mesenteric emboli usually originate from the heart. Cardiac thrombi may be associated with valvular disease, dilated left atrium, recent myocardial infarction, atrial arrhythmias and ventricular dilatation with mural thrombus. History of prior arterial embolism is common [7▪]. In an autopsy series, 19% had an acute myocardial infarction, 48% had remnant cardiac thrombus and 68% had synchronous embolus [2]. The embolus may occlude the arterial lumen completely or partially. Emboli tend to lodge at points of normal anatomical narrowing, usually immediately distal to the origin of a major branch. Typically, the embolus lodges a few centimeters distal to the origin of the SMA, sparing the proximal jejunal branches, and allowing preservation of the proximal jejunum.

Diagnosis

The nature of mesenteric vessel occlusion becomes clear after examination with CT with intravenous contrast enhancement and imaging in the arterial and portal phase.

Clinical presentation

The clinical triad, although inconsistent, for an acute embolic SMA occlusion in an elderly patient is severe abdominal pain but minimal findings at examination (pain out of proportion), bowel emptying and a source of embolus, most often atrial fibrillation. The often sudden onset of abdominal pain (phase I) may decrease in intensity (phase II),

followed by an increase in abdominal pain associated with clinical deterioration and progression toward peritonitis (phase III). In retrospect, a high proportion of the often misunderstood patients with acute thrombotic SMA occlusion may have had long-standing preexisting symptoms of CMI, including postprandial abdominal pain (abdominal angina), food fear, diarrhea and weight loss. Indeed, 80% of patients were inappropriately medically treated with proton pump inhibitors, cortisone or antibiotics in the diagnostic phase in a recent series [7▪]. The majority of these patients at diagnosis does not seem to be cachectic nowadays, maybe because of earlier diagnosis and/or a relatively high proportion of patients with over-weight at onset of symptoms [7▪].

Laboratory markers

D-dimer has been found to be the most consistent highly sensitive early marker, but specificity was low [8]. Most importantly, clinicians should be aware of diagnostic pitfalls that may be encountered in patients with acute SMA occlusion such as elevated troponin I and elevated pancreas amylase and normal plasma lactate, which may lead the clinician away from the diagnosis [9]. Hence, no plasma marker is yet sufficiently accurate to be of an early diagnostic aid in the acute setting [8]. In a recent report, intestinal fatty acid binding protein (I-FABP) was found to have the best diagnostic performance for the 24 patients with AMI among the tested plasma biomarkers among 208 patients with clinical suspicion of AMI [10▪].

Endoscopy

Acute or chronic mesenteric ischemia, with an insidious clinical course, may show signs of ischemia in the duodenum and in the right colon on endoscopy. *Helicobacter pylori* testing seems to be negative. Capsule endoscopy may be helpful to detect ischemic lesions in the small bowel [7▪].

Computed tomography angiography

The major breakthrough for diagnosis of acute (or chronic) SMA occlusion has been the evolution and availability of high-resolution CT around the clock [11,12▪]. Rapid reconstruction of images in the sagittal (Fig. 2a), coronal and transverse planes can be diagnostic. The presence of vascular disease precedes the intestinal disease [13]. In the absence of intestinal findings at CT or peritonitis, patients have likely been diagnosed in time for intestinal revascularization. Patients with impaired renal function should undergo CT angiography if there is a suspicion of acute SMA occlusion, without fear of contrast-induced renal failure [14], to improve the chance of survival.

Diagnosis of chronic mesenteric ischemia

Diagnosis of CMI is first based upon medical history, CT angiography or MRI and endoscopy findings. If available, gastric exercise tonometry or 24 h of gastrointestinal tonometry may be performed in the diagnostic workup [15]. Patients with significant or unclear grade of stenosis of the SMA on CT angiography or color doppler ultrasound are referred for angiography with pressure measurements across the stenotic lesion, and immediately stented if the pressure gradient in mean arterial pressure is 10 mmHg or higher. The presence of postprandial abdominal pain before treatment should disappear after successful stenting of the SMA.

Treatment of acute superior mesenteric artery occlusion

Optimal treatment may include surgery or endovascular techniques and patients are best treated at a vascular center with a hybrid operating room. Laparotomy is indicated if there is peritonitis. This aims to assess the extent and severity of intestinal ischemia: 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. In the event of bowel perforation, the affected intestinal segment is resected with staples, leaving the reconstruction of the intestines until second look laparotomy according to the principles of damage control surgery [16].

Acute mesenteric arterial revascularization

Acute mesenteric arterial revascularization is done preferably before any bowel surgery. According to the national Swedish registry of vascular procedures, Swedvasc [17], 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 North America [18]. The 30-day mortality rate in Swedvasc was similar after open versus endovascular surgery for embolic occlusions (37 versus 33%), whereas the mortality rate was significantly higher after open than endovascular treatment for thrombotic

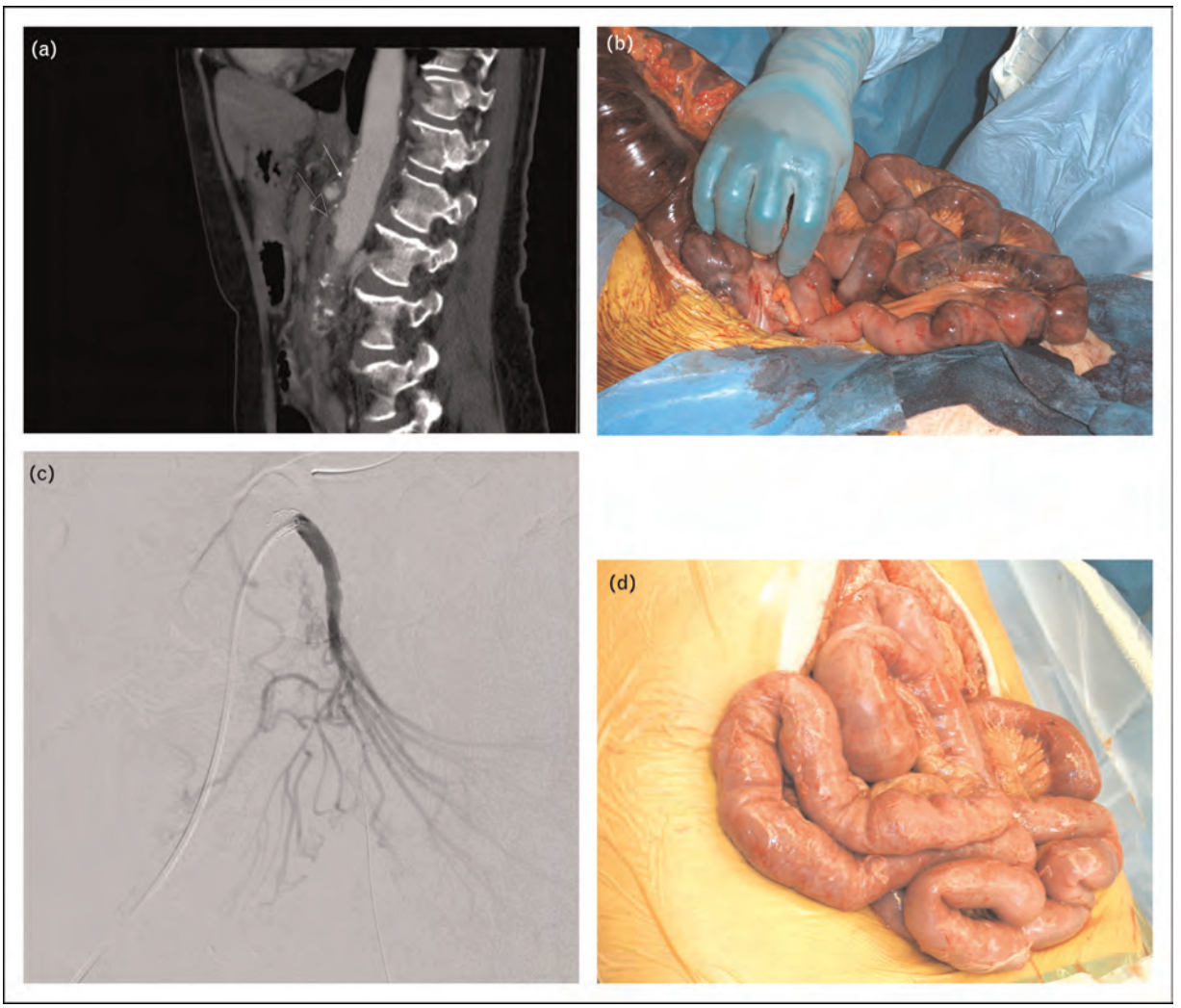

FIGURE 2. Case of a patient with an acute on chronic mesenteric ischemia: 76-year-old female patient with a history of smoking and chronic obstructive pulmonary disease. She fell ill for 6 months with abdominal pain and 13 kg of weight loss. Colonoscopy and gastroscopy were negative. She was admitted with 1 day of worsening of abdominal pain, vomiting and developed generalized peritonitis during her stay. (a) Computed tomography angiography with reconstructed images in the sagittal view showed a long thrombotic occlusion of the proximal SMA (large arrow head) and a short occlusion of the celiac trunk (small arrow head). Explorative laparotomy was performed after 48 h of acute symptoms. (b) Extensive intestinal ischemia is shown. Full bowel wall gangrene of the right and transverse colon, and variable depths of ischemia in the ileum are shown. (c) Completion frontal angiography after retrograde recanalization through the exposed SMA in the open abdomen and antegrade stenting shows good flow within the SMA and the branches to the small bowel. Fifteen minutes afterwards, another two meters of proximal small bowel were normalized in color. Resection of the right and transverse colon and two meters of small intestine was performed. d) At second look 40 h after the first procedure, three meters of viable small bowel were found and no additional bowel resections were required, and an ileocolic anastomosis was reconstructed.

occlusions (56 versus 23%). 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 celiac trunk and SMA revascularization seems to be more important.

Open vascular surgery for acute and chronic thrombotic superior mesenteric artery occlusion

If available, the preoperative CT angiography scan can be very useful to determine the type of occlusion

and suitable source of inflow artery and avoidable sites with extensive atherosclerotic lesions. Open acute SMA embolectomy remains a good treatment option [19], wherein the result should be checked by completion angiography. Division of the SMA distal to the occlusive lesion and re-implantation into the infrarenal aorta, thrombendarterectomy and patch, and bypass distal to the occlusive lesion are the surgical options. Autologous reversed saphenous vein may be the preferred conduit, especially in elective supra-celiac–SMA bypass. Polytetrafluoroethylene grafts reinforced with rings from the infrarenal aorta or the common iliac artery may be a good bypass conduit alternative, especially in the emergency setting in a noncontaminated peritoneal cavity, to withstand kinking of the graft.

Endovascular therapeutic options in acute or chronic mesenteric ischemia

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. If an antegrade approach from the femoral or brachial artery fails, a retrograde approach through the exposed SMA after laparotomy is performed [20]. The therapeutic options are aspiration SMA embolectomy, local SMA thrombolysis, antegrade or retrograde recanalization and stenting of the SMA (Fig. 2a–d).

Outcomes after open versus endovascular revascularization for acute mesenteric ischemia

Five nonrandomized studies [17,18,21,22,23▪] have compared open versus endovascular revascularization for AMI. One retrospective single-center experience [18] showed no difference in mortality, whereas the other single-center study showed lower bowel morbidity and lower mortality after endovascular therapy for acute thrombotic occlusions compared with open surgery [22]. The other three multicenter studies are nationwide reports [17,21,23▪]. These studies showed a lower frequency of bowel resection, and lower short-term [17,21,23▪] and long-term [17] death rates after endovascular therapy for acute thrombotic occlusion. The long-term survival at 5 years after endovascular treatment and open vascular surgery was 40 and 30%, respectively [17]. Independent risk factors for decreased long-term survival were short bowel syndrome and age.

Treatment of chronic mesenteric ischemia

Open surgical [24▪] and endovascular [25] revascularization are the treatment options for CMI. Endovascular therapy seems to be the best treatment option in vascular centers with expertise in these often elderly and fragile patients because of the minimally invasive nature of the treatment, shorter in-hospital stay, no requirement for ICU, lower costs and better cost-effectiveness [26].

Radiological follow-up

Patients who have a stent inserted in the SMA after treatment for an acute or CMI need to be followed regularly by either duplex imaging or CT angiography owing to the risk of restenosis and the need for reintervention [25] to prevent the serious consequences of stent occlusion [7▪].

Nonocclusive mesenteric ischemia

NOMI is a well established clinical condition affecting patients with low cardiac output, associated with the use of digitalis and/or other vasoactive drugs. In an autopsy series, 40% of the patients with NOMI had an SMA stenosis [27], a lesion potentially treatable with stenting. Angiography, if possible, should be performed to confirm or rule out this treatable condition [28], followed by immediate local administration of continuous vasodilatation therapy.

MESENTERIC VENOUS THROMBOSIS

MVT has a more insidious onset of symptoms and has a less severe and more limited extent of disease than arterial mesenteric ischemia. The high percentage of patients that can be managed by a limited small bowel resection, compared with patients with arterial cause, even at doctors' delay and late laparotomies, contributes to the better prognosis.

Pathogenesis

Several conditions are associated with MVT as follows:

(1) Direct injury
  (a) Abdominal trauma (blunt and penetrating)
  (b) Postsurgical (particularly postsplenectomy)
  (c) Intra-abdominal inflammatory states (pancreatitis, inflammatory bowel)
  (d) Peritonitis and abdominal abscess
(2) Local venous congestion or stasis
  (a) Portal hypertension; cirrhosis of the liver
  (b) Congestive heart failure

(c) Hypersplenism
(d) Obesity
(e) Increased abdominal pressure, abdominal compartment syndrome
(f) Pregnancy

(3) Thrombophilia
(a) Protein C and protein S deficiency
(b) Antithrombin III deficiency
(c) Activated protein C resistance (factor V Leiden gene mutation)
(d) Presence of 20 210 A allele of prothrombin gene
(e) Methylenetetrahydrofolate reductase mutations
(f) JAK2 V617F gene mutation
(g) Neoplasms (particularly pancreatic and colonic)
(h) Oral contraceptive use
(i) Polycythemia vera
(j) Essential thrombocytosis
(k) Heparin-induced thrombocytopenia
(l) Lupus anticoagulant-antiphospholipid syndrome
(m) Cytomegalovirus infection
(n) Extramesenteric venous thromboembolism

Obesity, systemic venous thrombus, pulmonary embolism, but not liver cirrhosis or abdominal cancer, were found to be the risk factors in a case–control study [29]. Inherited factor V Leiden mutation has been reported in up to 45% of patients with MVT [30].

The degree of intestinal ischemia that develops depends on the extent of venous thrombosis within the splanchnic venous circulation and whether there is occlusion and collateral flow. Patients with isolated PVT without peripheral propagation to the superior mesenteric vein are asymptomatic in the majority of cases, 61%, and almost never experience intestinal infarction [31]. In 270 patients with porto-MVT found at autopsy, 29 of 31 (94%) patients with MVT had intestinal infarction and 0 of 239 (0%) with isolated PVT had intestinal infarction [32].

Diagnosis

The availability and development of high-resolution CT scanners, and injection of intravenous contrast and imaging in the portal phase, have had a great impact on the more timely diagnosis of MVT and the possibility to manage most patients with a non-surgical, medical approach.

Clinical diagnosis

Patients with symptoms of less than 4 weeks of duration are classified as having acute MVT. The majority of patients, above 70%, in two large clinical series [30,33], have acute MVT. The onset of acute MVT is often insidious, and diffuse abdominal pain may be present for days or weeks before progression toward peritonitis [34]. The second most common symptom is nausea/vomiting, whereas diarrhea and lower gastrointestinal bleeding are rare. Typically, a middle-aged patient with a personal or a family history of deep venous thrombosis presents with abdominal pain of a few days' duration, vomiting and abdominal distention, as well as a clearly raised C-reactive protein level.

Computed tomography venography

CT of the abdomen, with intravenous contrast injection and imaging in the portal venous phase, is the most important and accurate diagnostic tool. MVT is seldom suspected by the clinician before ordering CT [35]. CT often demonstrates extensive thrombosis of the portomesenteric system, with extension of thrombosis to at least the extrahepatic portal and splenic veins. Intestinal findings are less common and more subtle.

Treatment of mesenteric venous thrombosis

Anticoagulation alone has become standard therapy after diagnosis at CT [34,35]. Therapeutic anticoagulation with a continuous infusion of unfractionated heparin is used for patients treated without surgery as well those diagnosed at operation. If necessary, the infusion can be stopped or protamine given to reverse the anticoagulation if urgent repeat laparotomy or second-look laparotomy is indicated.

A few patients deteriorate during medical treatment in whom endovascular treatment might be an option. A number of endovascular procedures for the treatment of MVT have been developed [36,37] in recent years, including percutaneous transjugular intrahepatic portosystemic shunting (TIPS) with mechanical aspiration thrombectomy and direct thrombolysis, percutaneous transhepatic mechanical thrombectomy, percutaneous transhepatic thrombolysis, thrombolysis via the SMA and thrombolysis via a surgically placed mesenteric vein catheter.

At operation, laparoscopy or laparotomy, MVT is characterized by a limited jejunal or ileal segment of intestinal ischemia, with edema, swelling and reddish discoloration of the affected small bowel and its adjacent mesentery and a palpable pulse in the SMA and its branches. MVT can be confirmed during surgery if an infarcted bowel segment is removed. Division of a small part of the adjacent mesentery, without previous vessel ligation, reveals

thrombosis within the veins, whereas a pulsatile hemorrhage arises from the arteries.

Blood screening

Patients with MVT should be screened for the following inherited disorders: factor V Leiden mutation, prothrombin gene mutation, protein C deficiency, protein S deficiency, JAK2 V617F mutation and antithrombin deficiency. Simultaneously, the patient should be checked for acquired disorders such as lupus anticoagulant and cardiolipin antibodies [30].

Prognosis in patients with mesenteric venous thrombosis

In a recent series, none of the 12 patients who underwent bowel resection developed short bowel syndrome [30]. The median length of the resected intestinal segment in that series was 0.6 m. The relatively high frequency (23%) of short bowel syndrome in patients with acute MVT in an older study might be attributable to unnecessarily extensive bowel resections or suboptimal pharmacologic therapy [33]. The survival rate after bowel resection is around 80% [33,38]. Long-term survival in patients with MVT depends largely on the underlying disease, especially the presence of cancer [30].

Medical treatment in mesenteric ischemia

After thrombotic arterial occlusion, patients should have antiatherosclerotic therapy, including an antiplatelet agent and a statin. After embolic arterial occlusion, lifelong vitamin K antagonist is indicated. Patients with MVT also receive anticoagulation for at least 6 months, or lifelong, depending on the underlying cause.

CONCLUSION

Early intestinal revascularization is the key to success in arterial occlusive mesenteric ischemia, using either endovascular or open vascular technique. Conservative anticoagulation therapy is very often successful in patients with a CT-verified MVT.

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Conflicts of interest

There are no conflicts of interest.

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- ■ of special interest
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