Chapter 18
Techniques of Endovascular Revascularization for Acute Mesenteric Ischemia

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History for AMI

Litten gave the first description of acute mesenteric ischemia (AMI) in 1875 [1]. Since that time the condition of AMI has been viewed as one of the most serious surgical emergencies with significant morbidity and mortality. The true incidence of acute mesenteric ischemia (AMI) is unknown, as most reports quote the classic study from Stoney in 1993 [2]. The most recent report was from Malmo, Sweden, where AMI is reported to occur 12.9 per 100,000 person years over a 10-year period [3]. The etiology is most commonly attributed to in situ thrombosis in 60 % of patients, embolism from atrial fibrillation in 30 %, and nonocclusive mesenteric ischemia in 10 % [4]. The diagnosis of acute mesenteric ischemia is based on both physical examination and diagnostic imaging. The symptoms will include progressive abdominal pain, which is classically described as "pain out of proportion to physical findings." Additionally, the patients usually have a significant history of tobacco abuse and other peripheral vascular disease. Once other etiologies of abdominal pain have been excluded, most patients will have a computerized tomographic scan (CT), which will delineate the mesenteric vessel anatomy. Traditional treatment has been open surgical revascularization, which includes embolectomy, bypass, and endarterectomy [5–7]. However, the morbidity and mortality for open surgical revascularization remain unacceptably high, with rates reported as high as 70 %.

In an effort to improve on the poor results of open mesenteric revascularization for AMI, many clinicians began to explore other alternative therapies, most notably endovascular surgery [8, 9]. With improvement in training, newer devices, and less morbidity, endovascular treatment of acute mesenteric ischemia is now the first initial therapeutic choice of treatment if clinical conditions allow it.

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Endovascular Techniques

Several endovascular treatment options exist to treat acute mesenteric ischemia. The decision of which option to use depends on the patient's immediate clinical condition. The patient's symptoms can be misleading, as they usually have significant abdominal pain, but an acute abdomen only happens when there is bowel perforation. If the patient has an acute abdomen or pneumatosis intestinalis is seen on the CT scan, the traditional approach has been combined open bowel resection and mesenteric revascularization. However, contemporary treatment has evolved, and an acute abdomen no longer precludes angioplasty and stenting, as surgical intervention can be done in a hybrid operating room, where both abdominal explorations can be performed either through exploratory celiotomy or laparoscopy in addition to angioplasty and stenting.

If the patient's lactate and liver function tests are normal and there is no evidence of an acute abdominal emergency or bowel infarction, our first treatment consists of diagnostic imaging in addition to the CT scan. In the setting of acute mesenteric ischemia, the most commonly involved vessel is acute occlusion of the superior mesenteric artery (SMA). In this circumstance, there is insufficient time to develop collaterals, and acute occlusion of the SMA can lead to bowel infarction. AMI is seen less commonly with acute celiac or inferior mesenteric artery (IMA) occlusion. Most patients will have undergone a CT angiogram delineating the anatomy (Fig. 18.1). This can give a good image of both orifice lesions and the distal extent of thrombosis. We then get both an anterior-posterior (AP) and lateral angiogram to determine the patency of all vessels (Fig. 18.2a, b), and it is not uncommon to see a large meandering mesenteric artery in the AP views (Fig. 18.3).

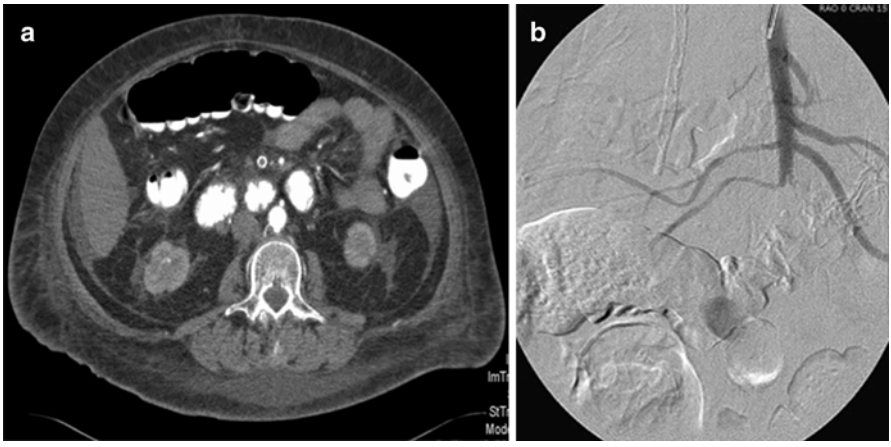

Fig. 18.1 (**a**) CT scan and (**b**) diagnostic angiogram of acute SMA occlusion

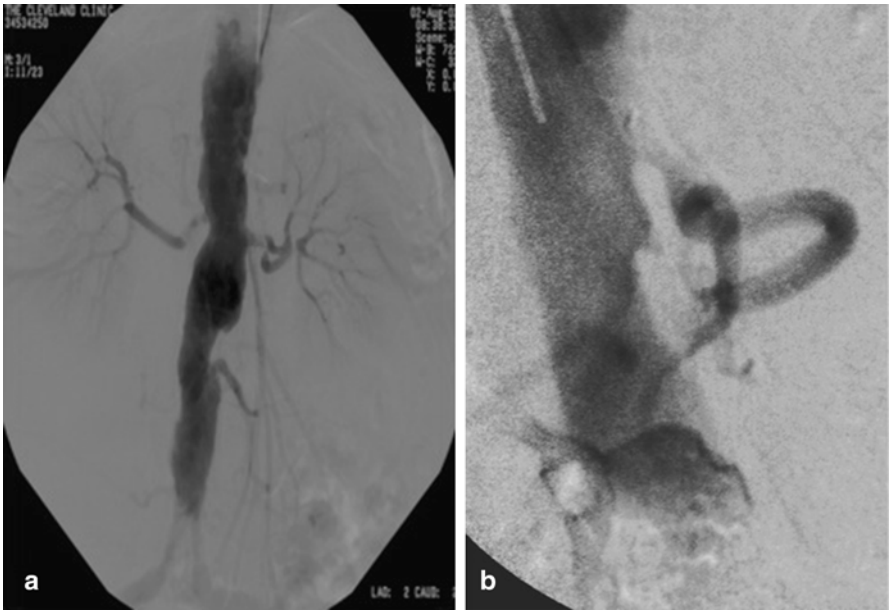

Fig. 18.2 (**a** and **b**) AP and lateral angiogram depicting visceral vessels

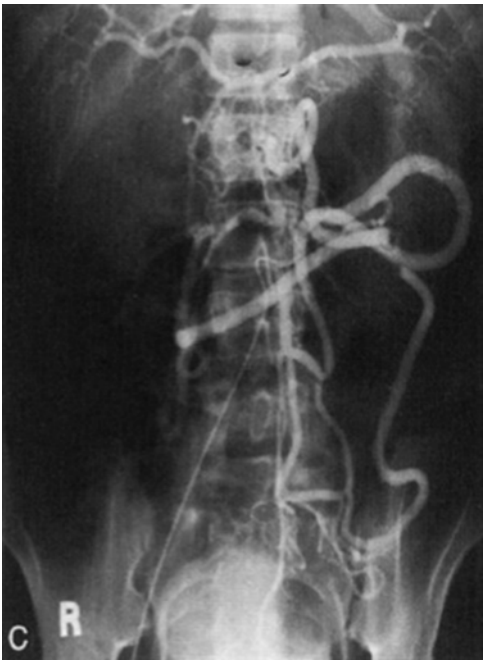

Fig. 18.3 Meandering mesenteric artery

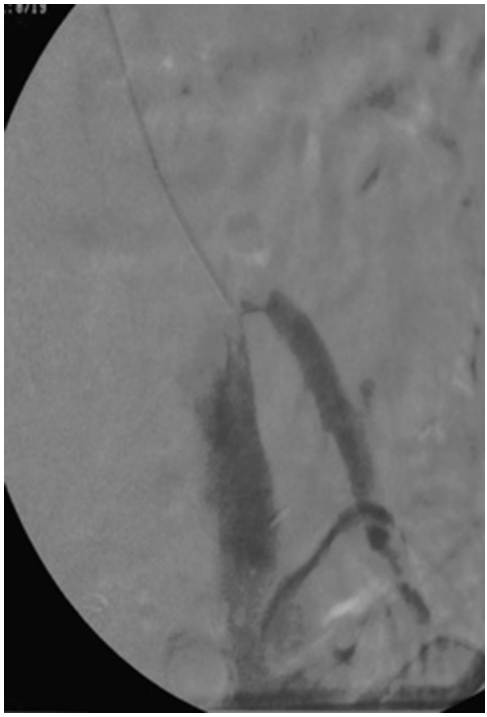

Fig. 18.4 MPA catheter with antegrade approach to the SMA

If the patient has complete occlusion of the superior mesenteric artery and their clinical condition will allow, we will attempt thrombolysis. Some contraindications to thrombolysis include severe hypertension, gastrointestinal bleeding, and embolism from atrial fibrillation. There are specific technical considerations to consider. The first step involves recanalizing and/or crossing the lesion. We usually use left brachial access as our initial approach as the angle makes it much easier to cross the lesion antegrade (Fig. 18.4). A long 5 F sheath (70–90 cm) usually is sufficient to provide adequate stability for tracking, as the final intervention will need to move to a 0.014 in wire. We usually use an MPA catheter and bury it into the stub of the occluded vessel, as it easily adapts to the curve of the aorta. Next I "drill" a 0.035 in stiff glide wire through the lesion, and then if possible pass the MPA catheter or a quick cross catheter through the lesion (Fig. 18.5). Once we have crossed the lesion, it is imperative to get an angiogram to confirm you are in the true lumen. If you have not reentered the true lumen, the catheter will need to be withdrawn and a new attempt should be made. If there is thrombus distal to the orifice and we have reentered and are not in a dissection plane, we will attempt thrombolysis with a short tip infusion catheter and/or wire [10]. The patient is then bolused with 2 mg of tissue plasminogen activator (TPA), and an infusion is started at 1 mg/h. We also keep the patient on a slightly higher dose of heparin that lower extremity ischemia, maintaining the activated partial thromboplastin time (APTT) near 40 s. If the patient's fibrinogen drops below 150 mg/dl, we then decrease the infusion rate to 0.5 mg/h.

Fig. 18.5 Sheath and wire across occluded SMA

If the fibrinogen drops to 100 mg/dl, then we will either give cryoprecipitate or suspend the infusion depending on the patient's clinical condition. If the patient demonstrates any signs of increasing abdominal discomfort, rising lactic acidosis, or clinical deterioration, we then stop and prepare for open surgery. Repeat angiograph is performed every 12 h. Once the distal clot clears, we then proceed to definitive angioplasty and stenting. If there is spasm, we will use either 30–60 mg of papaverine or 200–400 μg of nitroglycerine. If the clot has not cleared within 36–48 h, we will attempt mechanical thrombectomy with the Possis mechanical thrombectomy catheter [11]. A 0.014 in wire is needed for 5 F and 0.035 in wire for 6 F. Additionally, I prefer to use an embolic protection filter to prevent embolization, as this has been reported as high as 19 % for peripheral pharmacomechanical thrombolysis [12].

The typical culprit lesion from in situ thrombosis usually occurs from extension of an orifice lesion. However, it is not uncommon for lesions of the superior mesenteric artery to extend further. If there is not distal thrombus, immediate revascularization with balloon angioplasty and stenting is warranted. For orifice lesions of the celiac, SMA, and IMA, we use a balloon expandable stent (Fig. 18.6), and for more distal lesions we will use a self-expanding stent to adapt to curves (Fig. 18.7). Occasionally, we will have to use a combination of a self-expanding stent distally and of a balloon expandable stent proximally. Recent data from the Mayo Clinic [13] now supports the use of an ePTFE-covered stent graft for chronic mesenteric ischemia, and we have extended this to AMI for thrombosis in situ. For patients with AMI from embolism, we will attempt percutaneous mechanical thrombolysis with the Possis thrombectomy catheter. If their clinical condition will allow it, we will get a transesophageal echocardiogram, and if there is no clot in the atrium, we will also offer them thrombolysis. If this is unsuccessful or there is suspected persistent atrial thrombus, we will either attempt stenting with a covered stent or proceed with open embolectomy.

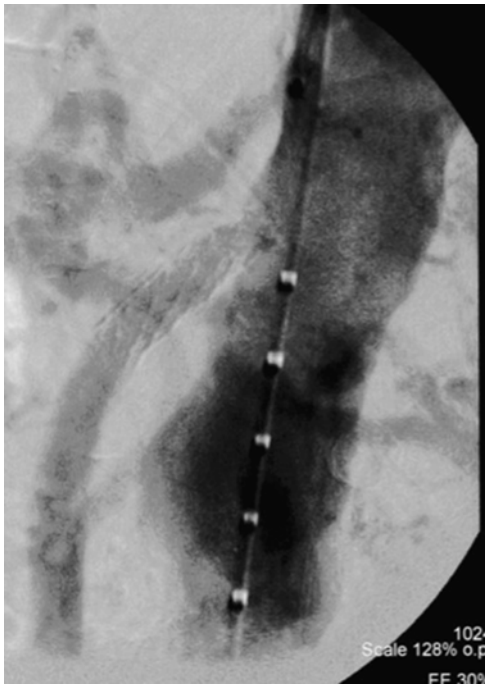

Fig. 18.6 Balloon expandable stent in orifice of the SMA

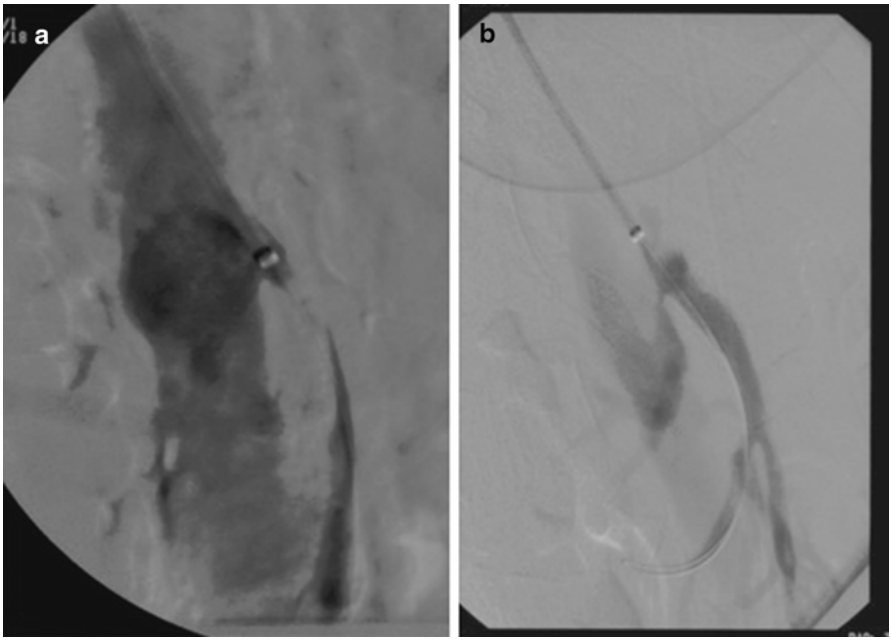

Fig. 18.7 (**a**) Selective SMA angiogram with disease beyond the orifice. (**b**) Self-expanding stent adapting to the curve

In circumstances where the patient's condition warrants emergent or urgent laparotomy, recent efforts have focused on a hybrid open surgery/stenting procedure [14, 15]. The technical details of this consist of performing the procedure in a hybrid operating suite and immediate exploration for control of sepsis and enteric spillage. We have been more inclined to use the brachial approach rather than the direct SMA cutdown if possible, as it allows more rapid revascularization. Nevertheless, there are advantages of the direct SMA cutdown as it will allow embolectomy. The procedure begins with dissecting the SMA out at the inferior margin of the pancreas or base of the transverse mesocolon. The artery is cannulated with a micropuncture needle, wire, and catheter, and then retrograde angiogram is performed. A 0.035 in wire is then used to exchange out for a 5 F sheath, and diagnostic imaging is performed. The lesion is crossed and the wire and a longer sheath are advanced into the aorta. Balloon angioplasty is frequently necessary before passing the sheath, and this usually requires exchanging out to a 0.014 in system. Retrograde stenting is done in the same fashion as antegrade which was previously described. It is not uncommon to have to perform a concomitant embolectomy, which can also be done with an over-the-wire balloon embolectomy catheter. On completion, the vessel is closed with either a vein or bovine pericardial patch angioplasty.

Less common etiologies of acute mesenteric ischemia include thoracoabdominal aortic dissection (TAAD), mesenteric venous thrombosis, and nonocclusive mesenteric ischemia. Acute mesenteric ischemia occurs in between 15 and 42 % of patients who suffer from TAAD [16]. Patient's symptoms include not only those severe of mid-thoracic back and chest pain but also abdominal pain. The diagnosis of AMI is entertained based on symptoms but also CT scan evidence of obstruction of the visceral vessels from the dissection flap (Fig. 18.8). Additionally, it is not uncommon in these circumstances for multiple visceral branch vessels to be involved.

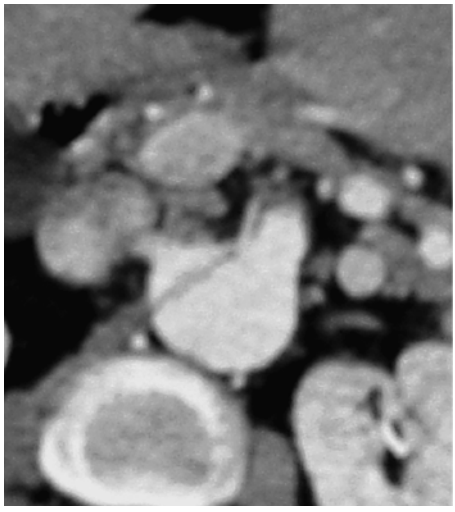

Fig. 18.8 CT scan depicting dissection flap in SMA

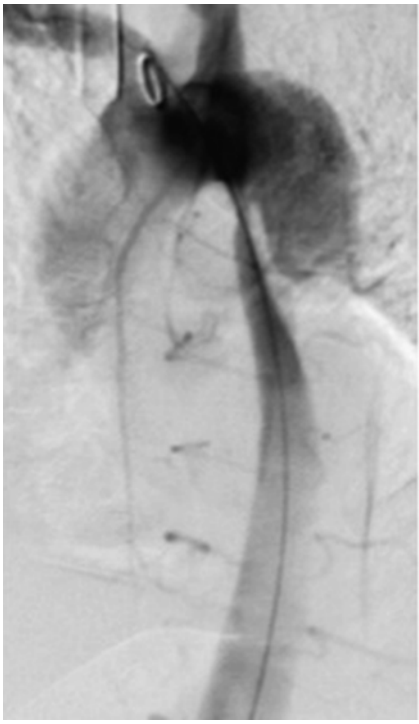

Fig. 18.9 Arch aortogram showing dissection at left subclavian artery

Symptoms can also be classified as subacute with waxing and waning of symptoms due to dynamic movement of the dissection flap. The morbidity and mortality with open surgical therapy have been previously described and remain as high as 50 % [16]. In 1999, Dake first described treatment for TAAD with a thoracic aortic stent graft [17]. Since that time there have been numerous studies documenting the success of endovascular therapy in treating malperfusion from TAAD [18–20]. Treatment in these circumstances consists of expansion of the true lumen with a thoracic stent graft, and occasionally an adjunctive self-expanding stent will need to be placed in the individual branch vessel. There are a number of technical details necessary to accomplish this, starting with preparation for bilateral femoral artery access, and be prepared to do an iliac conduit. Additionally, it is essential to have left brachial artery access, so the arterial line for blood pressure monitoring should be placed on the patient's right side. Intravascular ultrasound is a necessary adjunct. We usually perform a right femoral artery cutdown and obtain left brachial artery access from the start. The first step is to confirm you are in the true lumen, and here intravascular ultrasound is essential (Fig. 18.8). We then obtain an arch aortogram (Fig. 18.9) and exchange out for a 0.035 in stiff Lunderquist wire. A pigtail catheter is then placed from the left brachial artery into the aortic arch, which not only takes angiograms but also marks the subclavian artery. Often in an emergent setting, it is necessary to cover the origin of the left subclavian artery to cover the entry tear.

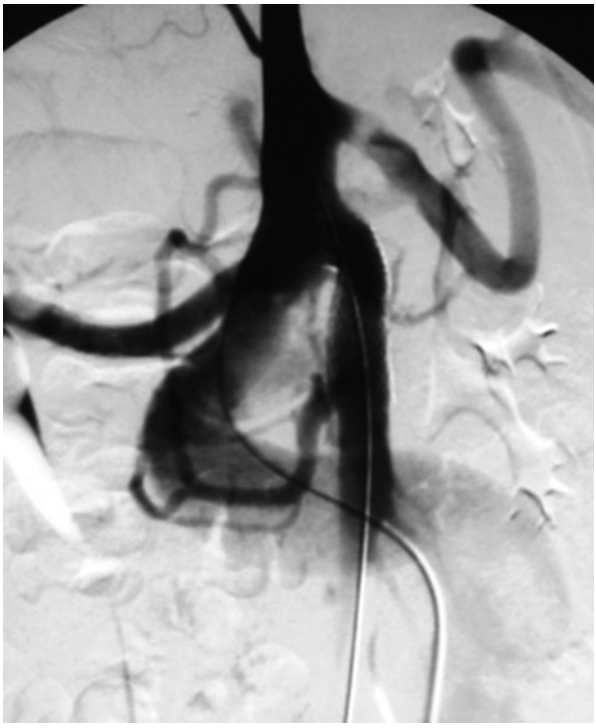

Fig. 18.10 Self-expanding stent in s = dissection

A subsequent emergent carotid subclavian bypass is necessary if there is not a normal size contralateral vertebral artery or if the patient is known to have an incomplete circle of Willis. Once the stent graft is placed, a distal angiogram is taken to confirm expansion of the aortic true lumen and evaluate the patency and flow of the visceral vessels. Occasionally, the dissection flap will necessitate the need to place a self-expanding stent into the branch vessel if flow remains compromised (Fig. 18.10).

Mesenteric venous thrombosis is another unusual cause of acute mesenteric ischemia, and the etiology usually is from a hypercoagulable state [21]. The mainstay of treatment usually consists of conservative management with systemic heparinization and conversion to oral anticoagulation, as long as there are no signs of an acute abdomen. However, if the patient's clinical condition deteriorates, several authors have reported success in improving symptoms by using intra-arterial thrombolysis through the SMA, and others have reported transhepatic venous success similar to TIPS [22]. Finally, nonocclusive mesenteric ischemia typically occurs from "low-flow states," such as cardiogenic or septic shock. The initial treatment involves supportive care for correcting the underlying condition. However, if the patient has persistent lactic acidosis, selective intra-arterial SMA perfusion with papaverine can correct the vasospasm (Fig. 18.11a and b).

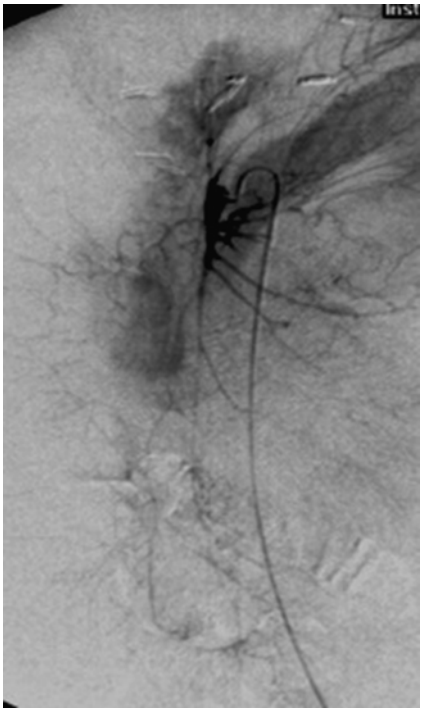

Fig. 18.11 Catheter in SMA infusing papaverine into a vessel with spasm

Results of Endovascular Therapy for Acute Mesenteric Ischemia

When analyzing new therapies, it is important to fully evaluate and compare the results to the traditional approach, here being open surgical revascularization for acute mesenteric ischemia. One recent series reporting the results of open surgical intervention came from Kougias et al. They evaluated 72 patients and found that the perioperative morbidity and mortality were 39 and 31 %, respectively [23]. As endovascular interventions became more popular, several authors reported their results. Arthurs et al. reported the Cleveland Clinic experience from 70 patients over 9 years [24]. Similar to almost all other studies, 65 % of the occlusions were thrombotic and 35 % embolic occlusions. Successful endovascular treatment resulted in a mortality rate of 36 % compared with 50 % with traditional open surgical therapy. Soon after Ryer et al. reported the Mayo Clinic experience over two decades [25]. They reported on 93 patients with AMI, 45 of who were treated during the 1990s and 48 during the 2000s. The majority of patients were treated with open revascularization. Endovascular therapy alone or as a hybrid procedure was used in 11 total patients, eight of which were treated in the last 10 years. Thirty-day mortality was 27 % in the 1990s and 17 % during the 2000s. Major adverse events occurred in

47 % of patients with no difference between decades and no significant difference in outcomes between open and endovascular revascularization.

Two separate reports evaluated and compared open to endovascular therapy using the National Inpatient Data Sampling during similar periods, with similar results. Beaulieu et al. [26] evaluated 23,744 patients presenting with AMI, 4665 underwent interventional treatment from 2005 through 2009, 679 patients underwent vascular intervention, 75.7 % underwent open surgery, and 24.3 % underwent endovascular treatment overall during the study period. The proportion of patients undergoing endovascular repair increased from 11.9 % of patients in 2005 to 30.0 % in 2009. Mortality was significantly more commonly associated with open revascularization compared with endovascular intervention (39.3 % vs 24.9 %); length of stay was also significantly longer in the patient group undergoing open revascularization (12.9 vs 17.1 days). They identified 6683 patients of which majority had an endovascular procedure (62.7 % vs 37.3) with an overall in-hospital mortality rate (IHM) of 17.4 %. Despite endovascular therapy having significantly lower in-hospital mortality (IHM) rates (15.3 % vs 21.2 %), over the 8-year period of study, there was no difference between open surgery and endovascular therapy.

Conclusions

Acute mesenteric ischemia has high mortality rates. However, with the incorporation of minimally invasive endovascular therapies, the rates have decreased. Prompt diagnosis and early treatment remains the mainstay of therapy.

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