Treating Mesenteric Ischemia

An overview of the many complicated facets involved in treating this presentation.

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Mesenteric ischemia has the potential for being acutely life threatening or a debilitating chronic illness. Few endovascular specialists have significant experience with the visceral or mesenteric vascular bed. Barriers to treatment can include a lack of knowledge of the anatomy, physiology, and treatment approaches. This article explores treatment approaches for mesenteric arterial insufficiency.

ANATOMY

Figure 1 demonstrates the rich arterial supply to the viscera. The three main arterial vessels arise from the aorta beginning just below the diaphragm and ending above the aortic bifurcation.

The first, the celiac artery (CA), arises perpendicularly at T12 or L1 and branches almost immediately into the left gastric (and occasionally the dorsal pancreatic), splenic, and hepatic arteries. The CA is responsible for supplying the liver, spleen, stomach, duodenum, and the pancreas.

Next, the superior mesenteric artery (SMA) arises 1 to 2 cm caudal to the CA, just above the renal arteries. It courses immediately inferiorly, supplying from the jejunum to the splenic flexure. In order, the branches are the inferior pancreaticoduodenal and then the middle, right, and ileocolic arteries.

The inferior mesenteric artery (IMA), the smallest of the three major trunks, arises obliquely 3 to 5 cm above the aortic bifurcation at the L3 level. Branches include the left colic, marginal, superior rectal, and sigmoid arteries, supplying from the splenic flexure to the rectum.

Critical stenoses may occur in all three major vessels, and the patient can remain asymptomatic due to extensive collateralization (Figure 2). Several recognized networks exist between the CA and SMA (left gastric, pancreaticoduodenal arcade), the SMA and IMA (central meandering mesenteric artery or arc of Riolan and the marginal artery of Drummond along the inner border of the colon), as well as between the internal iliac artery and IMA (rectal or hemorrhoidal arcade). Collaterals from the esophagus may also participate.

For symptoms to occur, it is classically taught that two out of three vessels need to be occluded, with the third compromised. This is certainly not the case when compromise occurs acutely and collaterals have not been established. Furthermore, there are symptomatic cases in which a single vessel (typically the SMA) is chronically stenosed. These may be evident when previous abdominal operations have disrupted the collaterals or after surgical abdominal aortic aneurysm repair.

ACUTE MESENTERIC ISCHEMIA

Acute mesenteric ischemia (AMI) accounts for less than one in 1,000 acute hospital admissions and is due to either in situ thrombosis on preexisting disease or, more commonly, from a remote arterial embolism.[1] Due to its downward course, the SMA is the most frequent recipient of the embolus, with the majority of emboli lodging distal to the middle colic artery thus sparing the first portion of small intestine and ascending colon. Less common causes of AMI are aortic dissection, recent instrumentation (atheroemboli), hypercoagulable states
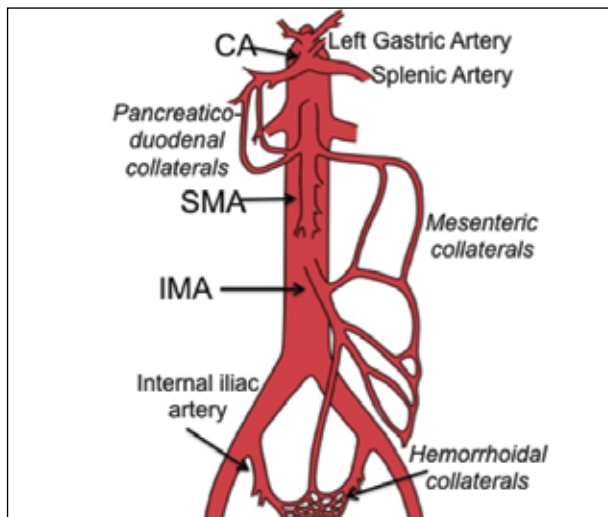
Figure 1. The CA arises just below the median arcuate ligament of the diaphragm and branches into the left gastric, splenic, and hepatic arteries. Collateral connections exist between the CA, SMA, IMA, and internal iliac arteries (italicized).

(mesenteric venous thrombosis), or low-flow states (splanchnic vasoconstriction).

Characteristically, the diagnosis is missed or made late, and mortality is frequent. Pain is described in the anterior abdomen, with few physical findings until there is transmural ischemia of the bowel. Typically, there is a rapid decline in patient status due to absence of collaterals, particularly if secondary to embolism. Laboratory findings are relatively nonspecific, but a low-serum D-dimer is a promising exclusionary test.[2]

The first-choice test is a dual-phase computed tomographic scan, which will identify clot in arteries or veins,

as well as the presence of bowel ischemia/infarction. Angiography can be selectively utilized pre-, intra-, and postoperatively for both diagnostic and therapeutic purposes.

CHRONIC MESENTERIC ISCHEMIA

The primary vascular process affecting the mesenteric vessels is atherosclerosis, typically at the aorto-ostial junction. The frequency and extent of atherosclerotic narrowing are much greater than the incidence of symptomatic disease. Asymptomatic patients with multiple-vessel involvement generally are at the greatest risk of becoming symptomatic, and approximately one-third of these individuals may progress to frank intestinal infarction.[3]

Chronic mesenteric ischemia (CMI) may occur from either postprandial shunting of blood to the gastric circulation or from demand ischemia.[4] Patients are typically elderly cachectic women who have undergone a panel of tests before the diagnosis is ultimately made. Beyond the universal complaint of postprandial abdominal pain, patients may also present with complaints suggesting gastropathy or colitis.

Diagnostic testing for CMI is multimodality (Figure 3), starting with duplex ultrasound (DUS). The advantages of DUS evaluation include a high sensitivity, but it is limited by low specificity, as well as technical difficulties presented by obese patients (unlikely a priori to have CMI) and bowel gas.[5] Furthermore, established velocity criteria do not apply to stented vessels in which velocities are seen to be routinely elevated immediately postprocedure.[6]

Computed tomography angiography (CTA) or magnetic resonance angiography reconstructed in multiplanar reformats and maximum-intensity projections are utilized as confirmatory testing or when DUS is ambigu-

Figure 2. The extensive collaterals often present with chronic advanced atherosclerotic narrowing of the visceral arteries. Anteroposterior aortography reveals a wandering mesenteric artery (arc of Riolan, curved arrow) arising from a small IMA (straight arrow) (A). The reason for collaterals is a high-grade ostial SMA stenosis (dashed arrow) (B). In a different patient, selective injection into a patent CA (solid arrow) reveals filling of an occluded SMA via pancreatic-duodenal arcade (dashed arrow) (C).
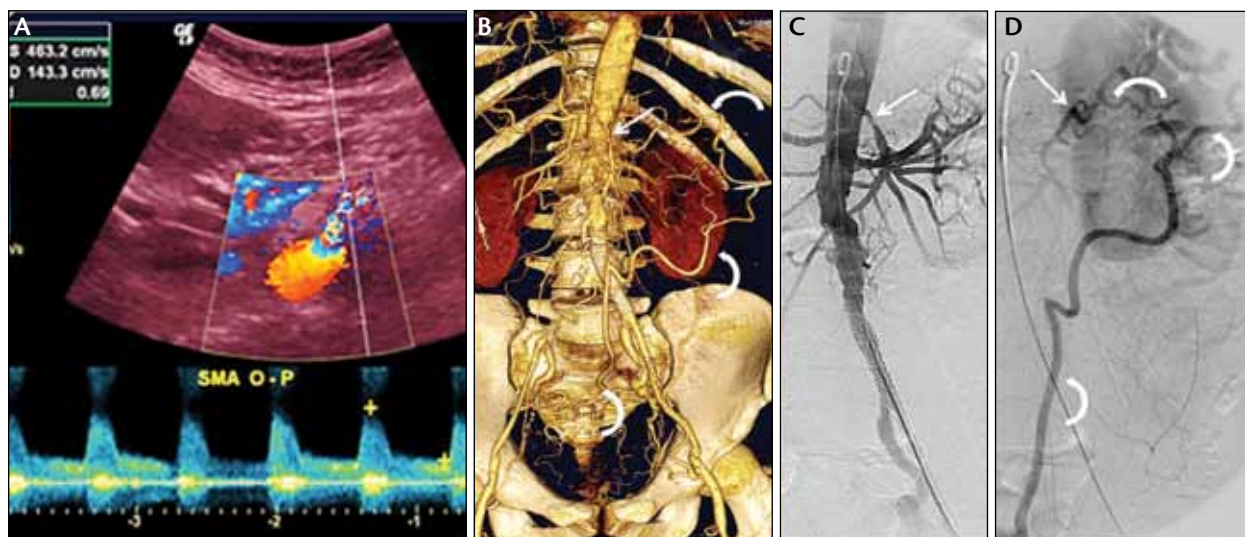
Figure 3. Multimodality imaging of a patient who presented with acute intestinal ischemia after coronary artery bypass grafting. DUS confirms occlusion of the CA (not shown), with significant SMA stenosis (systolic velocities > 275 cm/s; diastolic velocities > 45 cm/s) (A). CTA highlights the diffuse vascular disease present with occlusion of the right common iliac artery, stented left common iliac artery, and a wandering vessel (curved arrow) emanating from the left internal iliac/hemorrhoidal plexus and supplying a critically narrowed SMA (arrow) (B). Angiography confirms the presence of this collateral (curved arrow), as well as the high-grade SMA lesion that ultimately underwent stenting (C, D).

ous. Magnetic resonance angiography has the advantage of no ionizing radiation, but 64-slice or greater CTA has better spatial resolution, making rendering of smaller vessels possible.

Angiography is reserved for procedure planning or to clarify the diagnosis. The diagnosis of CMI may be suggested during routine abdominal anteroposterior aortography when collateralization of the mesenteric vessels is observed. A lateral projection of the aorta visualizes the predominantly ostial location of the disease. This view also allows for the diagnosis of other disease entities such as CA compression syndrome (Figure 4). A steep right anterior oblique view is required to visualize the ostium and proximal course of the IMA.

ENDOVASCULAR THERAPY VERSUS SURGERY

AMI

Surgery is traditionally the treatment of choice for AMI, involving resectioning of the bowel and, frequently, relook laparotomy. In many instances, the vascular anatomy is not known prior to surgery. In situations when an imaging or vascular specialist is available, additional surgical options may include embolectomy of the SMA or surgical bypass. Hybrid endovascular techniques may be utilized in the operating room (eg, retrograde open mesenteric stent). The SMA is recanalized intraoperatively by placement of a distal sheath under direct vision followed by retrograde placement of a stent.[7]

A limitation of primary endovascular therapy (EVT) for AMI is the inability to assess bowel viability, notably, in patients with peritoneal signs. However, an early endovascular approach may be considered for subacute presentations without peritoneal signs or when there is a delay in mobilizing a surgical team. Primary treatment with standard endovascular methods, such as aspiration embolectomy, local catheter-directed lysis, mechanical thrombectomy, or direct stenting, have been shown to be safe and effective.[8-10] AMI secondary to low-flow/low-output states is associated with diffuse vasospasm of the mesenteric arteries and responds to placement of an infusion catheter to locally deliver vasodilators (papaverine, nitroglycerine).[11] EVT-treated patients may require bowel resection due to delayed necrosis or development of abdominal compartment syndrome.[12]

The 2005 combined American College of Cardiology/American Heart Association guidelines, even prior to latter-day reports, gave EVT for AMI a class IIb (level of evidence C) recommendation relative to surgery (class I, level of evidence B).[13] This is reflected in the increasing utilization of EVT for AMI. Medicare data between 2000 and 2006 show that 64.5% of patients diagnosed with AMI underwent open surgery (embolectomy, endarterectomy, bypass) versus 35.5% who were treated with endovascular techniques.[14] However, by 2006, almost as many patients were treated with EVT as with surgery and showed encouraging results.

Bowel resection occurred significantly less often in endovascularly treated patients (28% vs 37%). AMI in-hospital mortality was 16% after EVT and 39% after surgery (*P* < .001), with overall higher morbidity and longer length of hospital stay after surgery.

CMI

Despite the absence of a randomized trial, there has been a steady increase in the reported endovascular treatments of CMI. In 1988, there were < 200 surgical procedures reported to Medicare, whereas in 2006, almost 1,400 were reported, 1,000 of which were treated with EVT. The mortality associated with EVT was significantly lower than with surgery (3.7% vs 15.4%, respectively).[14] This early survival benefit for EVT is supported by large single-center reports.[15] Furthermore, because there is no standardized functional test to identify CMI, the diagnosis is retrospective and often rests on the response to mesenteric revascularization. EVT is particularly attractive when the diagnosis remains uncertain. The combination of a good technical result and the resolution of symptoms allows a confident diagnosis of CMI.

The SMA is typically the primary target for revascularization, but reports have demonstrated resolution of symptoms after isolated CA or IMA interventions if the SMA cannot be treated.[16,17] Freedom from symptomatic recurrence seems to be improved with more complete revascularization.[18,19]
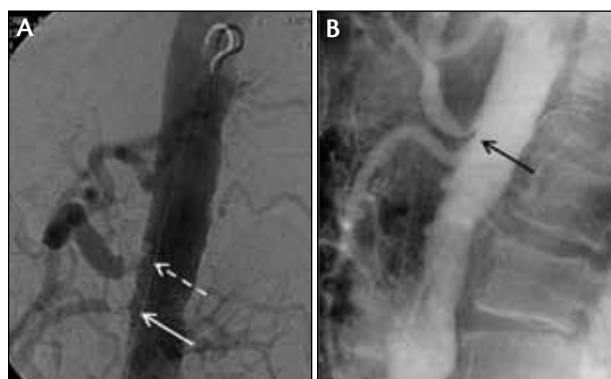

Figure 4. Lateral aortography depicts the ostial lesions involving the CA (dashed arrow) and SMA (solid arrow) (A). Selective engagement without prior knowledge of ostial disease could be hazardous or, in the case of mild disease, miss the diagnosis entirely. Characteristic appearance of CA compression with evidence of a hooked-shaped stenosis from impingement of the median arcuate ligament (B). Poststenotic dilatation is also noted. For the most part, this is an asymptomatic condition that is found in young individuals who are imaged for other reasons.
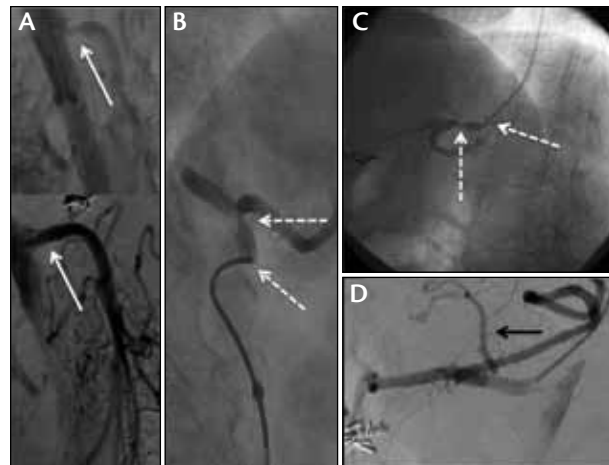

Figure 5. A patient with an SMA lesion (straight arrows) that was successfully treated from a femoral artery approach (A). Guiding catheter (8 F) support from the femoral approach was insufficient for the upgoing CA lesions (dashed arrow) (B). The procedure was successfully converted to a left brachial artery approach (C). Successful stent placement (D). Note the lesion; the left gastric artery (black arrow) that was successfully spared.

EVT is not devoid of important complications, including death, which has been reported in up to 10% of patients.[20] The restenosis rate is clearly higher than that of surgery (25%–50%).[21,22] It should be noted that restenosis after EVT can readily be treated with repeat percutaneous intervention.

The 2005 American College of Cardiology/American Heart Association guidelines give EVT and surgery equal weighting in the treatment of CMI (class I, level of evidence B).[13] Surgery is utilized for patients who are considered low risk with unfavorable anatomy for EVT, as well as for failed EVT because of flush occlusion, occluded stent, or in patients with recurrent in-stent stenosis who had multiple failed reinterventions. Nonatherosclerotic causes of CMI (vasculitis, neurofibromatosis, midaortic syndrome affecting the mesenteric arteries) may require primary surgical treatment. Open revascularization should avoid, if possible, extensive aortic or renal artery reconstruction. This typically involves a supraceliac aorta-to-CA and SMA bypass. In anatomically or physiologically compromised patients, a retrograde aortic or iliac artery bypass can be utilized. Transaortic endarterectomy may be considered in patients who are not candidates for EVT and have bacterial contamination or perforated bowel, previous abdominal irradiation, extensive abdominal wall hernias, or other hostile conditions.[23] CA compression syndrome (Dunbar syndrome) is typically treated by division of the median arcuate ligament either laparascopically or by open repair.[24]

Endovascular Techniques and Complications

The technique for mesenteric interventions is analogous to that of renal artery interventions with respect to cannulation of the vessels, choice of guiding catheter shapes, and use of 0.014-inch-based wires, balloons, and stents. Imaging of the origin of the CA and SMA, however, may need to be accomplished in steep lateral views, and prolonged procedures can be accompanied by high radiation dosages to patients and operators alike.

Specific techniques need to be highlighted. Due to the angulation of the CA and SMA, larger guiding catheters, such as 7 and even 8 F, should strongly be considered when using the femoral approach. Supportive 0.014- or 0.18-inch guidewires may be necessary for catheter passage. Femoral access can be problematic and necessitate use of an upper extremity access such as from the left brachial artery (Figure 5). A left radial artery approach requires a custom 125-cm-length multipurpose guide catheter. When working from the arm, a laterally positioned image intensifier can present logistical problems.

Occasionally, the CA and SMA can arise from a common aortic trunk, which will require additional bifurcation techniques (Figure 6). Chronic total occlusions may be addressed antegrade or retrograde with knowledge of collateral pathways.[25]

Due to the aorto-ostial nature of the disease, balloon-expandable stents (BES) can improve the acute outcome of the procedure. The size typically is in the 6- to 8-mm range. Self-expanding stents may be used for more distal

lesions or dissection poststenting. In a 140-patient case series, Dahl et al reported that the CA has a lower long-term patency rate versus the SMA after BES implantation based on duplex scanning criteria (55% vs 17% at 1 year).[26]

Based on the known durability of surgical synthetic grafts, covered BES have been utilized not only for the treatment of in-stent restenosis but as a primary treatment strategy. If they are used, it is crucial to avoid important proximal branches. The CA proximal branches are the left gastric and dorsal pancreatic, both of which can be an important source of collaterals. In the SMA, the first branch is the inferior pancreaticoduodenal artery, again, an important collateral source to the CA.

In a recent study comparing treatment strategies, there was no restenosis advantage of metal BES over plain balloon angioplasty; however, covered stent implantation appeared to have a markedly lower restenosis rate.[27] At 1 year, the primary patency rate for BES, angioplasty alone, and polytetrafluoroethylene-covered stents was 54%, 67%, and 100%, respectively. Primary patency rates based on vessel type (SMA or CA) were not different. Beyond being a nonrandomized comparison, the findings were limited by a smaller number of covered stent implantations (14 vs 77) and a shorter period of follow-up (mean, 19 vs 6.6 mo). Regardless of the limitations, the use of covered stents appears promising.

The utility of embolic protection remains undefined. As in the renal arteries, early branching limits the efficacy (Figure 7). However, placement of one of these devices is reasonable to consider in certain anatomical situations
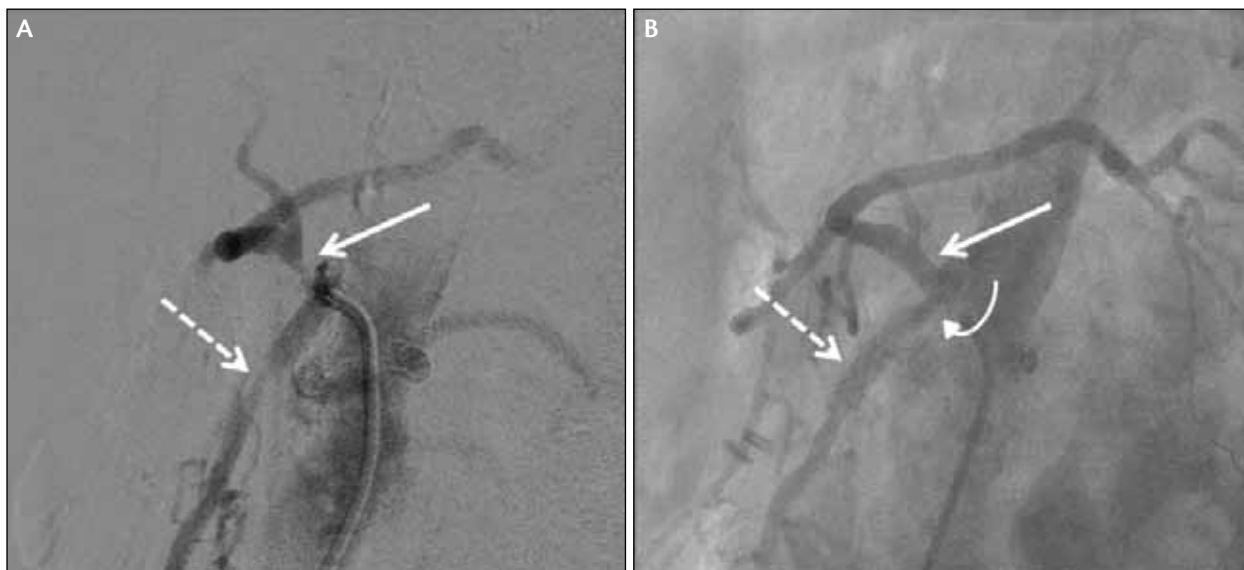
Figure 6. Patient with a common origin of both the CA and SMA. The origin stenosis of the CA (solid arrow) is evident, as well as a proximal SMA lesion (dashed arrow) (A). Stent placement in both locations with mild nonflow-limiting narrowing of the origin of the SMA (curved arrow) (B).
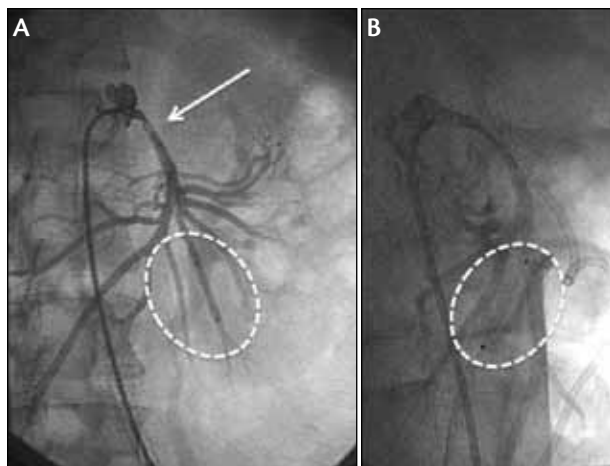
Figure 7. Patient with a high-grade proximal SMA stenosis (arrow) and occlusions of both the CA and IMA. An embolic protection device had to be positioned (A) and deployed distally (B) enough to lend support to and not interfere with stent delivery. The efficacy of protection was diminished by its position beyond important proximal branches.

(long-segment occlusions, severe calcification, or acute/subacute ischemia) in which there may be an increased risk of distal embolization.

The most serious procedural complication is the development of acute intestinal ischemia. "Trashing" the distal mesenteric bed with atheroemboli is usually not a rectifiable situation. Aggressive guide catheter manipulations in diseased aortas can also easily "trash" the kidneys or feet. Dissection or thrombosis can be rescued by additional stents or thromboaspiration. If required, mesenteric bypass with prior full anticoagulation must be undertaken promptly to prevent propagation of clot into the distal jejunal and ileal arcades in which surgical embolectomy is not practical. Isolated splenic infarction has also been reported after stenting of the CA.[28] Intestinal reperfusion hemorrhage is rare but has reportedly been successfully treated by endovascular embolization.[29]

SUMMARY

Although any one endovascular specialist is unlikely to regularly see either AMI or CMI, its timely identification is imperative. Early detection requires a high degree of clinical suspicion and, in particular, an appreciation of the variable syndromes and imaging protocols associated with the disease. EVT has emerged as a viable treatment option for both AMI and CMI. Endovascular techniques can be accomplished with less morbidity and acceptable clinical results with straightforward options for retreatment without compromising the subsequent ability to proceed with surgery. ■

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