Techniques in
Vascular and
Interventional
Radiology

Chronic Mesenteric Ischemia

Luke R. Wilkins, MD, and James R. Stone, MD, PhD

Endovascular management and treatment of chronic mesenteric ischemia has taken on an increasing role in recent years. The safety, efficacy, and cost-effectiveness have been validated in several studies. The procedure is best performed by an operator with a complete understanding of the pertinent imaging findings; a thorough knowledge of the risks, benefits, limitations, and technical challenges of the procedure; and understanding of the importance and necessity of long-term clinical management. This article outlines a general approach to endovascular management of chronic mesenteric ischemia and discusses indications, potential complications, and technical aspects of the procedure.
Tech Vasc Interventional Rad 18:31-37

KEYWORDS Mesenteric ischmeia, stent, angioplasty

Background

Chronic mesenteric ischemia is an uncommon clinical entity owing in part to a robust collateral vascular network. Classic descriptions of chronic mesenteric ischemia require narrowing or occlusion of 2 or more main visceral arteries before the development of symptoms. However, it is increasingly being recognized that comprises of 1 or more of the celiac, superior mesenteric arteries (SMAs), or inferior mesenteric arteries (IMAs) may lead to the development of symptoms secondary to the inconsistency of collateral blood flow.

Chronic mesenteric ischemia most frequently occurs in the setting of atherosclerotic disease with resultant narrowing of 1 or more mesenteric arteries. However, in contrast to most other atherosclerotic diseases, chronic mesenteric ischemia is more frequently seen in women.[1] This is likely secondary to differences in the orientation of the mesenteric vessels to the aorta, with a more acute angle to the aorta in women when compared with men.[2] The likely result is altered flow dynamics and increased susceptibility to atherosclerotic disease. More specifically, women with a body mass index of 25-29.9 had a mean aortomesenteric angle of 49.5°. This is in contrast to male patients in the same body mass index range with a mean aortomesenteric angle of 63.8°. This difference may affect flow dynamics, predisposing women to atherosclerotic disease of the mesenteric vessels. Other etiologies of SMA stenosis or narrowing include fibromuscular dysplasia, vasculitides, and postoperative intimal hyperplasia.

The natural history and factors affecting progression of mesenteric artery stenoses are not well defined. Progression from chronic to acute mesenteric ischemia is associated with $>50\%$ mortality. However, given the risks associated with open surgical and endovascular treatments, if and when to treat asymptomatic mesenteric arterial lesions remains a controversial topic.

Clinical Evaluation

History and Physical Examination

Chronic mesenteric ischemia is often referred to "intestinal angina" and is classically manifested as recurrent episodes of abdominal pain secondary to insufficient splanchnic blood flow during periods of heightened demand. Postprandial pain will generally begin shortly after eating and last 1-2 hours and is typically described as crampy and dull. In addition, nonspecific symptoms can also be seen, including nausea, vomiting, and diarrhea. Patients typically have a history of smoking and underlying atherosclerotic disease, with approximately half of patients having known peripheral vascular disease or coronary artery disease.[3] Weight loss is present in approximately 80% of patients and is attributable to "food aversion"

Department of Radiology and Medical Imaging, University of Virginia Health System, Charlottesville, VA.

Address reprint requests to Luke R. Wilkins, MD, Department of Radiology and Medical Imaging, University of Virginia School of Medicine, Box 800170, Charlottesville, VA 22901. E-mail: lrw6n@virginia.edu

1089-2516/15/$ - see front matter
http://dx.doi.org/10.1053/j.tvir.2014.12.005

owing to anticipation of postprandial pain. Although physical findings are frequently nonspecific, an abdominal bruit may be detected in up to 50% of patients.

Imaging

Evaluation of chronic mesenteric ischemia may be performed with catheter-based angiography, computed tomographic angiography (CTA), magnetic resonance angiography (MRA), or ultrasound. Although catheter-based angiography remains the gold standard, advances in noninvasive methods have led to the use of ultrasound, CTA, or MRA as the initial tests to evaluate for mesenteric arterial disease before proceeding to angiography.

Duplex ultrasound is an inexpensive and readily available imaging modality to screen for chronic mesenteric ischemia. This option provides physiological flow data and can be performed in the fasting and postprandial states to detect physiologically significant stenoses.[4] In the fasting state, the SMA possesses a high-resistance waveform characteristic of the splanchnic circulation whereas the celiac artery demonstrates higher end-diastolic velocities (EDV), with flow throughout the cardiac cycle owing to the low resistance within the liver and spleen. However, in the postprandial state, the SMA will show a marked increase in EDV with a less dramatic increase seen in the celiac circulation. A significant stenosis will be characterized by an increase in the peak systolic velocity and EDV during duplex ultrasound (US) investigation. Retrograde flow in the hepatic artery may be seen with a severe stenosis or occlusion of the celiac artery, whereas loss of diastolic flow or flow reversal may be seen with a significant stenosis of the SMA. Limitations of duplex US include presence of overlying bowel gas, normal variant arterial anatomy, patient body habitus, operator training, and lack of uniform criteria for interpretation of the duplex US findings.[4] Additionally, multivessel disease can lead to the overestimation of stenotic lesions secondary to the development of rich collateral arterial flow.

CTA has become increasingly used to diagnose chronic mesenteric ischemia. In addition to localizing sites of occlusive lesions, CTA allows for secondary findings such as an occult pancreatic malignancy and bowel ischemia or infarction. In the setting of previous endovascular or surgical treatment, CTA can allow for assessment of stent or graft patency, respectively.[5] Recent progress in the display of acquired CT data, including multiplanar reformation, maximum intensity projection, volume rendering, and surface shaded display, has been used to optimally delineate significant lesions and aid greatly in preprocedure planning (Fig. 1).[6]

MRA is another noninvasive imaging modality for the assessment of chronic mesenteric ischemia. Traditional techniques such as phase contrast and time of flight may be supplanted by the use of fast, contrast-enhanced sequences to provide superior imaging with minimization of motion and flow artifacts. Although the spatial resolution of MRA is inferior to catheter-based angiography and CTA, its sensitivity and specificity for evaluation of proximal stenotic or occlusive lesions within the renal arteries exceeds 90%.[7] Furthermore, MRA can perform functional assessment of intestinal perfusion using cine phase contrast and compare flow within the SMA and SMV following a meal. Significant postprandial increases in flow through the SMV as compared with the SMA have been found to predict the presence of mesenteric ischemia.[8]

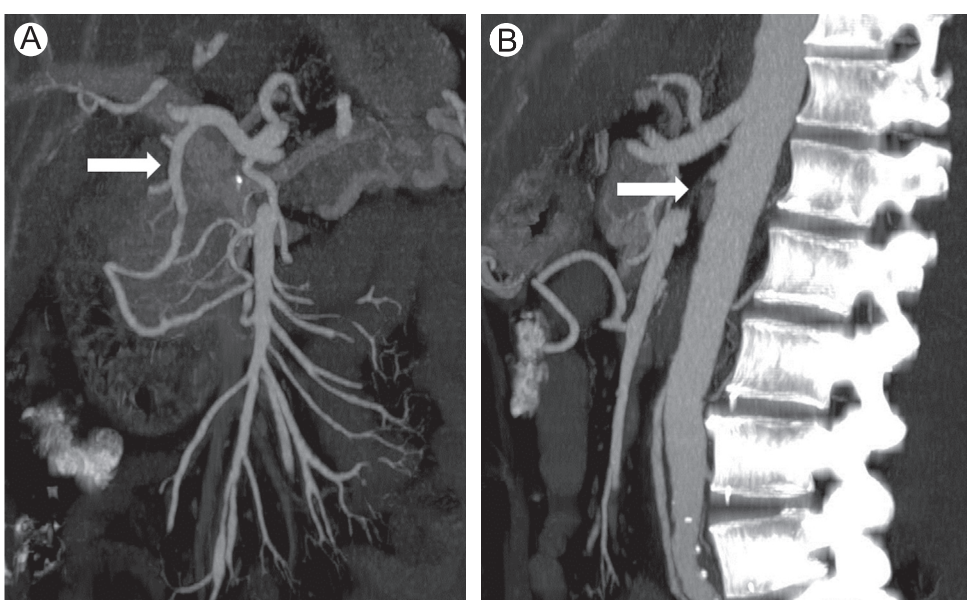

Figure 1 A 64-year-old patient with symptoms of weight loss, abdominal pain with eating, and food fear. (A) Maximum intensity projection (MIP) reconstruction of a CTA in a coronal plane shows dilation of the GDA and pancreaticoduodenal arcade (arrow), concerning for SMA ostial narrowing. (B) Sagittal plane image demonstrates short-segment occlusion of the SMA (arrow). GDA, gastroduodenal artery.

Procedural Steps

Arterial access through a femoral approach is generally preferred. Brachial artery access may be used in the setting of aorto-iliac occlusive disease or with an acute downward angle of the mesenteric arterial branches in relation to the aorta. When brachial artery access is used, care should be taken to minimize the profile of catheters and sheaths to prevent brachial artery thrombosis or formation of an arterial hematoma that may compromise the arterial supply to the extremity or result in compressive injury to the median nerve or both.

If using a 0.035 platform, it is advantageous to secure sheath access in the groin or arm with a long sheath compatible with the delivery of expected stent choice (see below). This allows the operator to minimize sheath exchanges during the course of the procedure. For example, from the groin, a 7-F, 45-cm Flexor Ansel Guiding Sheath (Cook medical, Bloomington, IN) may be used. Sheath options from a brachial access include 6- or 7-F Flexor Raabe Guiding Sheath in 55-, 70-, and 90-cm lengths (Cook medical, Bloomington, IN).

Diagnostic images include an initial aortogram in antero-posterior and lateral projections. The antero-posterior aortogram allows for the identification of distal arterial disease while providing an overview of the mesenteric vascular anatomy and dynamic flow from one territory to another. The lateral aortogram will allow for the detection of ostial disease of the celiac artery, SMA, and IMA.

A Cobra 2, SOS Omni 2, RC 1, or Simmons 1 catheter (AngioDynamics, Glen Falls, NY; Cook, Inc, Bloomington, IN; Boston Scientific, Natick, MA; Cordis Endovascular, Warrenton, NJ) may be used for catheterization of the celiac artery or SMA. Catheterization of the IMA may be achieved using a SOS Omni 2 or RIM catheter (AngioDynamics and Cook, Inc, respectively). In the setting of arterial occlusion or critical stenosis, a hydrophilic steerable guidewire may be used to cross the lesion. This guidewire may be exchanged for a stiff exchange wire once the lesion is traversed. In the celiac or SMA branches, a 0.035 in system is preferred for the treatment of heavily calcified lesions, given the increased durability of balloons and stents on a 0.035 in platform as compared with those available for use with 0.018 or 0.014 in wires. Treatment of IMA lesions may require 0.018 or 0.014 in wires given the relatively small caliber of this vessel.

Lesions with $>60\%$ diameter narrowing or with 70%-80% reduction in cross-sectional area, in combination with at least 20-mm Hg systolic translesion pressure gradients at rest, without provocative maneuvers, are generally considered significant. These criteria have largely been adapted from the renovascular hypertension literature.[9-11] A systolic peak measurement of <10 mm Hg is considered not significant, whereas 10-20 mm Hg is considered borderline with treatment guided by presence and nature of clinical symptoms.

When stent placement is required, the choice of stent is generally dictated by the location and type of lesion. For ostial lesions, balloon-expandable stents are generally preferred. Options include Express LD and Herculink (Boston Scientific, Natick, MA and Abbott Vascular, Menlo Park, CA). Lesions within the trunk of the mesenteric arteries may be treated with either balloon-expandable or self-expanding stents (Figs. 2 and 3). Although bare metal stents are often used, covered stents (CS) may be employed in the setting of soft plaque, in arteries

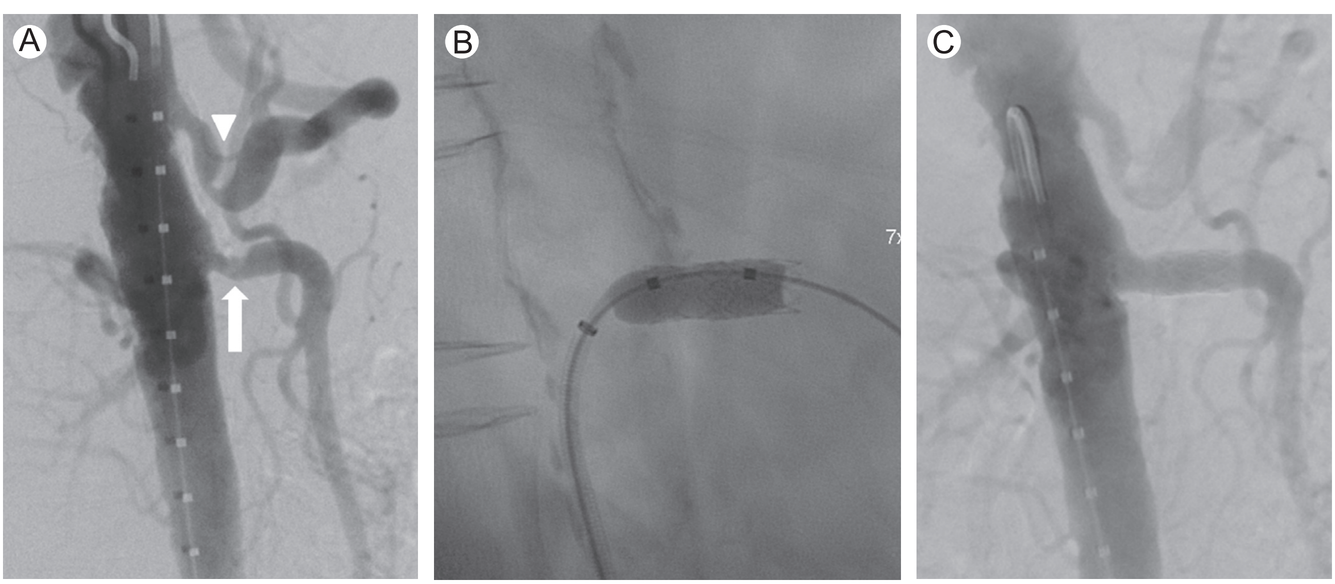

Figure 2 A 58-year-old patient with symptoms of fullness and weight loss. (A) Nonselective abdominal aortogram in the lateral projection demonstrates narrowing at the origin of the SMA (arrow). Also noted is narrowing of the celiac artery with appearance consistent with median arcuate ligament compression (arrowhead). Pressure measurements across the SMA lesion demonstrated a gradient of 23 mm Hg. (B) Following predilation, a balloon-expandable bare metal stent of 7 × 18 mm was positioned within the proximal SMA. (C) After stent deployment, the SMA is patent, with good flow, as seen on the lateral view.

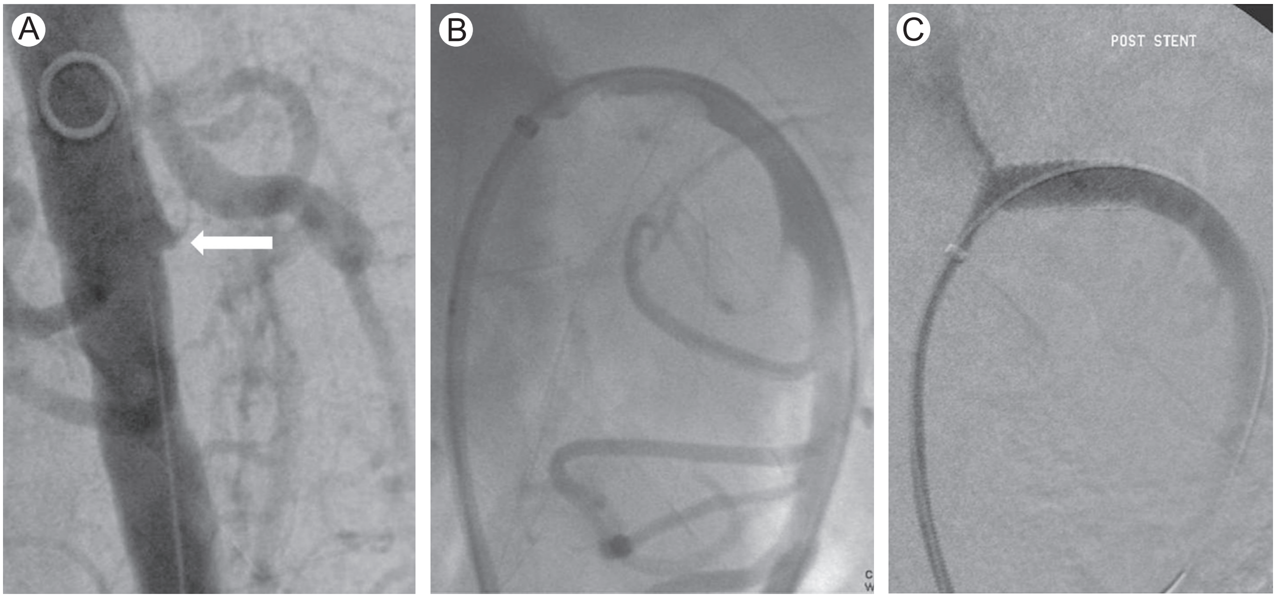

Figure 3 A 78-year-old patient with 2-week history of worsening abdominal pain and occluded SMA on CTA presented for evaluation. (A) Nonselective lateral aortogram demonstrates occlusion of the SMA near the ostium (arrow). (B) A hydrophilic guidewire and reverse-curve catheter was used to cross the occlusion. A sheath was unable to cross the lesion. Following predilation with a balloon of 4×20 mm, a sheath was able to be placed. The sheath was withdrawn, and a contrast injection demonstrated focal narrowing (arrow), which is still seen at the proximal aspect of this vessel, likely secondary to atherosclerotic disease. (C) A balloon-expandable bare metal stent of 6×18 mm is positioned within the proximal SMA. After stent deployment, the SMA is patent, with good flow, as seen on the lateral view.

<6 mm in diameter (to reduce the risk for in-stent restenosis), or for the treatment of in-stent restenosis secondary to intimal hyperplasia (Fig. 4). Options include the iCast covered stent (Atrium, Hudson, NH). Additionally, in a recent comparison of CS vs bare metal stent, CS were associated with improved freedom from restenosis (92% vs 53%), symptom recurrence (92% vs 50%), and primary patency at 3 years (92% vs 52%).[12] Furthermore, improved rates of restenosis, symptom recurrence, and patency were also seen in patients undergoing reintervention for chronic mesenteric ischemia.

Following diagnostic arteriograms, pressure measurements, and stent selection, the sheath should be advanced beyond the lesion. This may be accomplished with predilation with a 4-mm balloon. Following inflation of the balloon, the balloon may be deflated and the sheath advanced over the balloon as it is slowly deflated. The stent is then placed through the sheath and uncovered when in optimal location based on previous lateral arteriogram. When the stent is uncovered, the position may be confirmed before final deployment through an injection through the side arm of the sheath.

Balloon or stent advancement may be hindered by tortuosity of the iliac vessels, acute angle of the artery to be treated, and the presence of a tight or occlusive lesion. In these settings, an 8-F Morph catheter (Biocardia, San Carlos, CA) may be of some use given the ability to steer the tip of the catheter and provide additional support. Treatment of tight or occlusive lesions is frequently aided by predilation of the lesion with a low-profile balloon.

After stent deployment, dilation with an angioplasty balloon may be necessary to ensure stability of the stent. In addition, the proximal end of the stent may be overdilated and flared to maximize hemodynamic flow through the stent. For example, if a 7-mm CS is placed, the proximal end of the stent may be dilated with an 8-mm balloon and flared into the aorta (Fig. 4G).

Pharmacologic adjuncts used in the treatment of chronic mesenteric ischemia include intravenous heparin of 3000-5000 IU with a target activated clotting time of >220 seconds. Intra-arterial nitroglycerin in boluses of 100-200 μg may be administered to prevent or minimize spasm. When a stent is deployed, 325 mg of aspirin and 300 mg of clopidogrel may be administered in the recovery room. Aspirin 81-325 mg should then be continued for life and 75 mg of clopidogrel should be administered for at least 30 days.

Potential Complications

Complications associated with stent placement in the SMA include dissection, embolization, thrombosis, perforation, and stent dislodgement. Although complication rates are frequently low (less than 10%), the occurrence of anatomical complications within the mesenteric artery significantly increases risk of death and morbidity, up to 18% and 54%, respectively.[13] Most common causes of death following mesenteric artery stenting include myocardial infarction and multiorgan system failure caused by bowel ischemia from distal embolization, dissection, or stent thrombosis. Despite

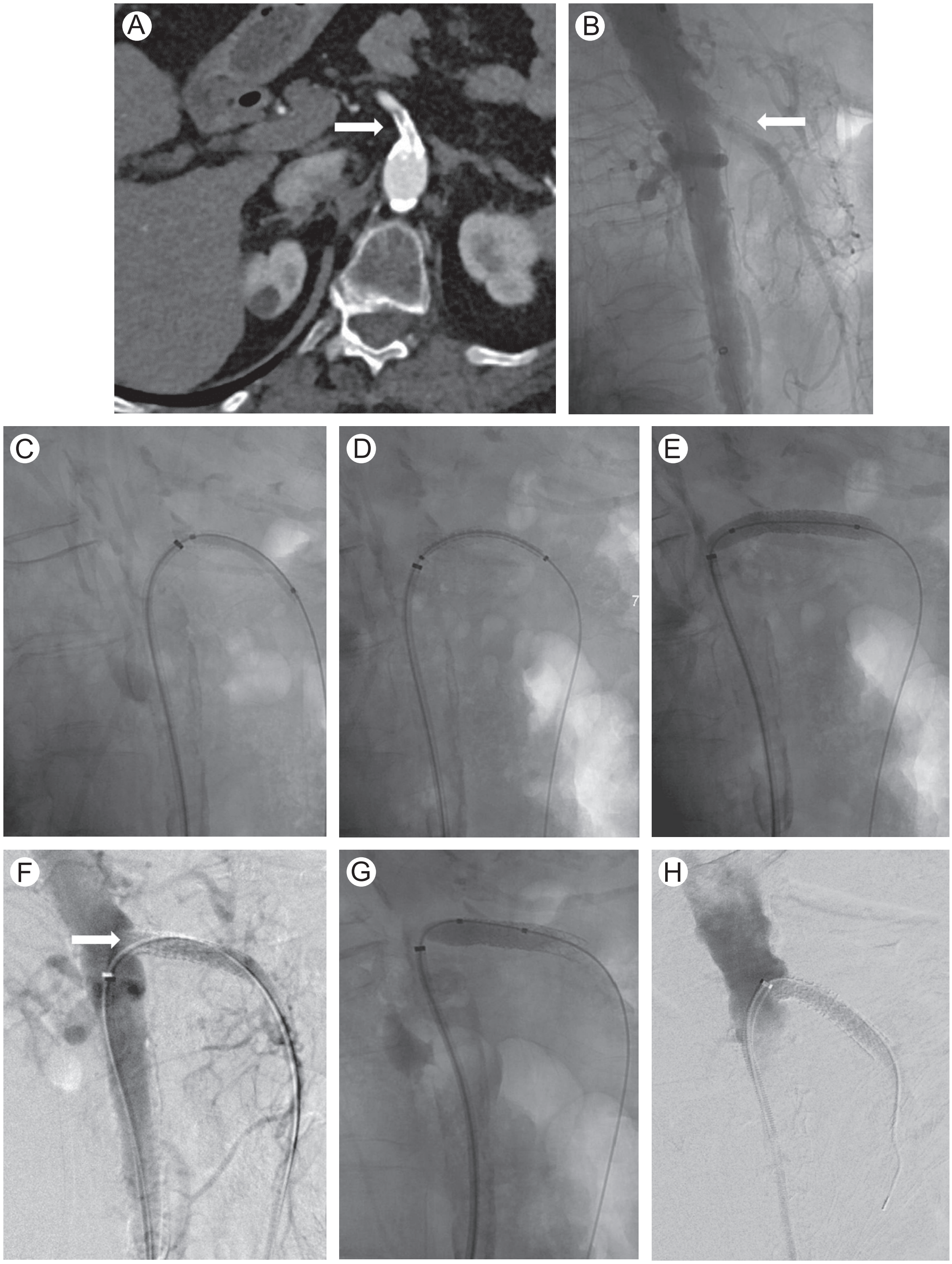

Figure 4 A 63-year-old woman with previously placed balloon explandable stent presented with recent worsening of abdominal pain and food intolerance. (A) Axial image from a CTA shows occlusion of the SMA stent (arrow). (B) Lateral aortogram shows marked narrowing in the previously placed stent, consistent with in-stent restenosis (arrow). (C) Predilation was performed with a 4-mm angioplasty balloon. (D) An iCast of 7 × 38 mm was deployed. (E) Postdilation was performed with a 7-mm balloon. (F) After stent placement, lateral aortogram demonstrates residual narrowing at the proximal margin on the stent. (G) A balloon of 8 × 20 mm was used to flair the proximal edge of the stent. (H) Lateral aortogram shows improved appearance of stent with no significant pressure gradient detected.

the morbidity and mortality associated with embolic events, distal embolic protection remains a controversial topic in this setting. Although this tool may be considered in treatment of chronic mesenteric ischemia, it may be argued that many embolic events may be prevented with proper angiographic technique and adequate heparinization.

Clinical Follow-Up

Patients should be followed in an outpatient clinic at 30 days after procedure with a history, physical, and duplex US. A full evaluation for assessment of symptoms, weight gain, and food tolerance should be performed to assess for clinical success. If the procedure was technically and clinically successful, the patient may return to clinic at 6 months after procedure for repeat duplex and clinical assessment. The patient may then be followed yearly thereafter.

Expected Outcomes

The primary goal of treatment of chronic mesenteric ischemia is to provide adequate revascularization of the mesenteric arterial bed. Surgery has classically been performed for the treatment of chronic mesenteric ischemia and has typically involved either transaortic endarterectomy or mesenteric artery bypass grafting.[14]

Endovascular management has taken on an increasing role in the treatment of chronic mesenteric ischemia caused by atherosclerotic occlusive lesions or fibromuscular dysplasia.[15] Median arcuate ligament compression of either the celiac artery or SMA is generally better managed with surgical decompression of the affected arteries as surgical decompression also alleviates compression of the celiac ganglion and the neural plexus.[16-18] Endovascular management will frequently result in stent fracture by extrinsic forces associated with diaphragmatic compression.

A marked increase in the number of procedures performed for mesenteric revascularization has occurred over past 20 years with much of the increase secondary to an increased use of endovascular techniques.[15] Many case series have been published evaluating the efficacy of endovascular therapy in chronic mesenteric ischemia. Razavi et al evaluated 70 symptomatic patients that underwent stent placement for treatment of chronic multisymptom illness. Technical success was seen in 97%. The recurrence rate was 10.5% with a mean follow-up of 3 years. Risks for recurrence included occlusions, lesions >3 cm in length, and stent diameter <5 mm[19] (Fig. 4). A separate study showed a clinical success rate of 87.9% in a cohort of 33 patients with a mean period of clinical follow-up of 38 months. There was a 17% rate of recurrent symptoms with a primary assisted long-term clinical success rate of 96.6%.[20]

In the comparison of open surgical vs endovascular techniques, Oderich et al[2] performed a 14-year retrospective review of outcomes in 229 patients undergoing open surgical vs percutaneous transluminal angioplasty or stent placement for treatment of chronic mesenteric ischemia. Morbidity was 36% in the open surgical group vs 18% in the endovascular group ($P < 0.001$). However, 5-year recurrence-free survival was 55% in the endovascular vs 89% in the open surgical group ($P < 0.05$). Additionally, open surgical repair was associated with improved primary (88% vs 41%, $P < 0.05$) and secondary rates (97% vs 88%, $P < 0.05$) when compared with endovascular repair. However, a recent meta-analysis evaluating the comparative effectiveness and cost-effectiveness of endovascular repair vs operative repair found that endovascular repair is favored over operative repair in all age groups.[21] Although endovascular repair is associated with more expected reinterventions, it is overall more cost-effective than operative repair.

Summary

Chronic mesenteric ischemia is a relatively uncommon clinical phenomenon owing to the rich collateral circulation of the gastrointestinal tract. When strong clinical suspicion for this disease process is present, assessments can be performed with US, CTA, or MRA to confirm a diagnosis. Catheter-directed angiography can be performed in patients with equivocal findings on noninvasive imaging or when intervention is anticipated. The primary goal of treatment is to improve blood flow to the mesenteric vascular bed. Endovascular therapy has taken on an increasing role in the primary management of chronic mesenteric ischemia in recent years. Although prospective comparisons of endovascular and open surgical approaches remain lacking, retrospective studies comparing these 2 approaches suggest surgical bypass is associated with a more durable result. Open surgery, however, is associated with increased periprocedural morbidity and mortality when compared with endovascular approaches.[2,22,23] As such, current recommendations for treatment involve endovascular management with stent placement as a first-line approach, particularly in patients who are poor surgical candidates owing to nutrition status or comorbidities or those who have undergone previous abdominal surgery. In patients who are surgical candidates and fail endovascular therapy, bypass generally remains an option.

References

1. White CJ: Chronic mesenteric ischemia: Diagnosis and management. Prog Cardiovasc Dis 54:36-40, 2011
2. Oderich GS, Bower TC, Sullivan TM, et al: Open versus endovascular revascularization for chronic mesenteric ischemia: Risk-stratified outcomes. J Vasc Surg 49:1472-1479.e1473, 2009
3. Moawad J, Gewertz BL: Chronic mesenteric ischemia: Clinical presentation and diagnosis. Surg Clin North Am 77:357-370, 1997
4. Armstrong PA: Visceral duplex scanning: Evaluation before and after artery intervention for chronic mesenteric ischemia. Perspect Vasc Surg Endovasc Ther 19:386-392, 2007 [discussion 393-384]
5. Shih MC, Angle JF, Leung DA, et al: CTA and MRA in mesenteric ischemia: Part 2, normal findings and complications after surgical and endovascular treatment. Am J Roentgenol 188:462-471, 2007
6. Calhoun PS, Kuszyk BS, Heath DG, et al: Three-dimensional volume rendering of spiral CT data: Theory and method. Radiographics 19:745-764, 1999
7. Bhatti AA, Chugtai A, Haslam P, et al: Prospective study comparing three-dimensional computed tomography and magnetic resonance imaging for evaluating the renal vascular anatomy in potential living renal donors. BJU Int 96:1105-1108, 2005
8. Burkart DJ, Johnson CD, Ehman RL: Correlation of arterial and venous blood flow in the mesenteric system based on MR findings. 1993 ARRS Executive Council Award. Am J Roentgenol 161:1279-1282, 1993

9. Mangiacapra F, Trana C, Sarno G, et al: Translesional pressure gradients to predict blood pressure response after renal artery stenting in patients with renovascular hypertension. Circ Cardiovasc Interv 3:537-542, 2010
10. Corriere MA, Pearce JD, Edwards MS, et al: Endovascular management of atherosclerotic renovascular disease: Early results following primary intervention. J Vasc Surg 48:580-587, 2008 [discussion 587-588]
11. Drieghe B, Madaric J, Sarno G, et al: Assessment of renal artery stenosis: Side-by-side comparison of angiography and duplex ultrasound with pressure gradient measurements. Eur Heart J 29:517-524, 2008
12. Oderich GS, Erdoes LS, Lesar C, et al: Comparison of covered stents versus bare metal stents for treatment of chronic atherosclerotic mesenteric arterial disease. J Vasc Surg 58:1316-1323, 2013
13. Oderich GS, Tallarita T, Gloviczki P, et al: Mesenteric artery complications during angioplasty and stent placement for atherosclerotic chronic mesenteric ischemia. J Vasc Surg 55:1063-1071, 2012
14. Shanley CJ, Ozaki CK, Zelenock GB: Bypass grafting for chronic mesenteric ischemia. Surg Clin North Am 77:381-395, 1997
15. Schermerhorn ML, Giles KA, Hamdan AD, et al: Mesenteric revascularization: Management and outcomes in the United States, 1988-2006. J Vasc Surg 50:341-348.e341, 2009
16. Kohn GP, Bitar RS, Farber MA, et al: Treatment options and outcomes for celiac artery compression syndrome. Surg Innov 18:338-343, 2011
17. Roseborough GS: Laparoscopic management of celiac artery compression syndrome. J Vasc Surg 50:124-133, 2009
18. Grotemeyer D, Duran M, Iskandar F, et al: Median arcuate ligament syndrome: Vascular surgical therapy and follow-up of 18 patients. Langenbecks Arch Surg 394:1085-1092, 2009
19. Razavi M, Chung HH: Endovascular management of chronic mesenteric ischemia. Tech Vasc Interv Radiol 7:155-159, 2004
20. Matsumoto AH, Angle JF, Spinosa DJ, et al: Percutaneous transluminal angioplasty and stenting in the treatment of chronic mesenteric ischemia: Results and longterm followup. J Am Coll Surg 194:S22-S31, 2002
21. Hogendoorn W, Hunink MGM, Schlosser FJV, et al: A comparison of open and endovascular revascularization for chronic mesenteric ischemia in a clinical decision model. J Vasc Surg 60:715-725, 2014
22. Atkins MD, Kwolek CJ, LaMuraglia GM, et al: Surgical revascularization versus endovascular therapy for chronic mesenteric ischemia: A comparative experience. J Vasc Surg 45:1162-1171, 2007
23. Davies RS, Wall ML, Silverman SH, et al: Surgical versus endovascular reconstruction for chronic mesenteric ischemia: A contemporary UK series. Vasc Endovascular Surg 43:157-164, 2009