Mesenteric Ischemia

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KEYWORDS

• Mesenteric ischemia • Chronic mesenteric ischemia • Acute mesenteric ischemia
• Nonocclusive mesenteric ischemia • Median arcuate ligament syndrome

KEY POINTS

- For all forms of mesenteric ischemia, catheter-based angiography remains the diagnostic gold standard, but when not available high-quality, thin-slice computed tomographic angiography (CTA) is an acceptable alternative.
- Acute mesenteric ischemia must be diagnosed and intervened upon rapidly to prevent catastrophic outcomes. A high index of suspicion based on history and physical examination findings is essential for the proper diagnosis and expeditious treatment of this disease process.
- Endovascular treatment options, including catheter-directed thrombolysis and visceral vessel stenting, are emerging techniques in the treatment of acute mesenteric ischemia. These methods are best used when the diagnosis is made early and bowel integrity has not yet been compromised.
- Discovery of mesenteric venous thrombosis on delayed venous-phase CTA should prompt a thorough evaluation for underlying hypercoagulable states, including occult malignancy.
- Chronic mesenteric ischemia often demonstrates a significant delay in diagnosis. The triad of postprandial pain, weight loss, and food fear are most often present. Surgical bypass remains the therapy of choice, but endovascular techniques are increasingly used.
- Median arcuate ligament syndrome can be treated by open or laparoscopic release of the compressive bands followed by endovascular techniques if residual symptoms and stenosis are present after decompression.

INTRODUCTION

The recognition of mesenteric vascular insufficiency and subsequent intestinal compromise dates back to 1895, with the first description of 2 cases of bowel resection for compromised arterial inflow.[1] With these initial descriptions, the fundamental finding of "pain out of proportion to physical examination" was established. In

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addition, the recognition of the rapidity with which acute mesenteric ischemia can progress, and the gravity of missed or delayed diagnosis, was also firmly established. It was not until later that the entity of chronic visceral ischemia was borne out in the literature, initially referred to as angina abdominis.[2,3] Nearly 40 years after these initial observations, the first successful surgical intervention was reported.[4] Shaw and Maynard described the first use of thromboendarterectomy for what is now termed acute-on-chronic mesenteric ischemia. Our understanding of mesenteric ischemia continues to evolve with new recognition of compressive syndromes, including median arcuate ligament syndrome.[5]

Although visceral ischemia remains rare (2 to 3 cases per 100,000 population), the dire consequences of missed or delayed diagnosis continue to make it a focus of great attention. The need for prompt and accurate diagnosis is essential in avoiding catastrophic complications, especially in acute and acute-on-chronic mesenteric ischemia. This article focuses on each of these entities with regard to relevant history, diagnosis, and treatment options.

MESENTERIC ANATOMY

The mesenteric circulation comprises 3 main branches of the abdominal aorta: the celiac axis (CA), the superior mesenteric artery (SMA), and the inferior mesenteric artery (IMA) (**Fig. 1**).[6] Each of these is richly collateralized, such that significant disease of 2 branches is often required to result in symptoms of chronic ischemia.

The CA gives rise to branches that perfuse the liver, stomach, spleen, and pancreas. It collateralizes via the superior anterior and posterior pancreaticoduodenal branches to the SMA. The origin of the CA often arises from the ventral surface of the abdominal

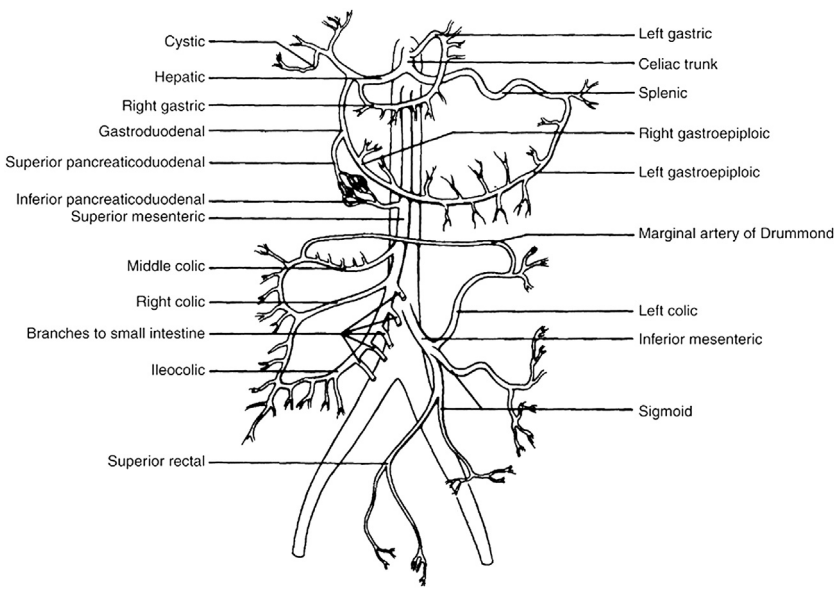

Fig. 1. The mesenteric circulation with abdominal collateral networks. (*From* Schwartz LB, Davis RD, Heinle JS, et al. The vascular system. In: Lyerly HK, Gaynor JW, editors. The handbook of surgical intensive care. 3rd edition. St Louis (MO): Mosby Year Book; 1992. p. 287; with permission.)

aorta between the T12 and L1 vertebral bodies. The median arcuate ligament runs across this origin, the implications of which are discussed later in this article. The most notable anatomic variant related to the CA is the replaced right hepatic. In approximately 12% of patients, the right hepatic artery arises from the SMA and not the proper hepatic branch of the CA.

The SMA is the next abdominal branch, arising from the ventral surface of the abdominal aorta between the L1 and L2 vertebral bodies. It collateralizes via the inferior anterior and posterior pancreaticoduodenal branches to the CA. In addition, it forms a collateral network via the marginal artery of Drummond to the IMA. The distribution of perfusion of the SMA includes partial pancreas and duodenum, the entire jejunum and ileum, and the ascending and transverse colon. Because of its relative size, long common segment, and proximal and distal collateral connection, the SMA is often a target for revascularization procedures.

The IMA arises from a slightly left anterolateral location on the abdominal aorta, usually a few centimeters above the aortic bifurcation. It collateralizes via the marginal artery of Drummond to the SMA and through the superior rectal branches to the hypogastric (internal iliac) artery. The IMA is most commonly compromised in patients with aneurysmal disease whereby mural thrombus occludes flow, or in those patients with significant tobacco abuse and subsequent severe aortoiliac occlusive disease.

ACUTE MESENTERIC ISCHEMIA

Acute mesenteric ischemia (AMI) has historically been associated with poor outcomes, with hospital mortalities rates ranging anywhere from 50% to 100%.[7–13] The essence of treatment has focused on a high clinical index of suspicion, early diagnosis, visceral revascularization, bowel resection, second-look laparotomy, and supportive care. There are 4 distinct pathophysiologies associated with acute mesenteric ischemia[9,14,15]:

- Arterial embolism (50%)
- Arterial thrombosis (20%)
- Nonocclusive pathologies (20%)
- Mesenteric venous thrombosis (10%)

Arterial Embolism

Ischemia caused by embolism is the most common form of AMI, accounting for roughly half of all patients. This condition results from embolism, usually from a cardiac source, to one of the visceral branches. The most common source of central embolism is atrial fibrillation with subsequent formation of atrial appendage thrombus.[16] Additional sources include left ventricular mural thrombus owing to hypokinesis in regions of prior myocardial infarction.[16,17] In addition, emboli may arise from cardiac valvular origins.

The branch most often affected is the SMA, because of its high basal flow rate and the anatomic angle of take-off.[18] The minority of emboli remain at the origin of the SMA (<15%), with most emboli resulting in more distal lodging, often just past the take-off of the middle colic vessel. Proximal SMA branch vessels may be preserved, as seen in **Fig. 2**.[19] Reactive vasospasm of the more distal mesenteric circulation can often compound the injury, and simultaneous embolism to other vascular beds is seen up to 15% of the time.

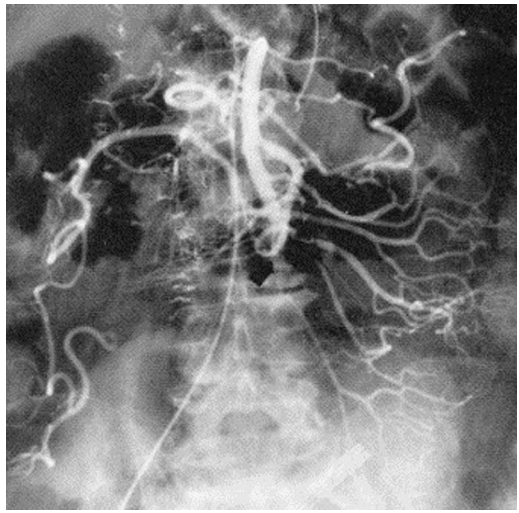

Fig. 2. Typical angiographic appearance of a superior mesenteric artery (SMA) embolism, with proximal branch sparing and mid-segment occlusion of the SMA; this is in contrast to an athro-occlusive lesion that most often results in flush occlusion at the aortic interface. (*Reproduced from* McKinsey JF, Gewertz BL. Acute mesenteric ischemia. Surg Clin North Am 1997;77:307–18.)

Arterial Thrombosis (Acute-on-Chronic)

Acute-on-chronic mesenteric ischemia implies an abrupt change overlaid on a preexisting picture of chronic mesenteric atherosclerosis. Because of the typical delay in diagnosis of chronic mesenteric ischemia, this form of acute ischemia accounts for nearly one-quarter of all AMI presentations. Careful questioning of the patient or family will most often reveal a history of weight loss, food fear, bloating, or other symptoms of chronic mesenteric ischemia, leading to acute clinical decline. Typically this form of ischemia results from slow progression of mesenteric atherosclerosis until a point of critical stenosis has developed; then, during a period of low flow, thrombosis of this critical lesion occurs. Contrary to embolic presentations, these lesions are most commonly flush to the origin of the vessel, and are often heavily calcified on computed tomography (CT) imaging. This pathophysiology has been described after myocardial infarction, acute viral illness, and even after other surgical interventions, including cardiac surgery.[20] Any clinical scenario leading to low flow and/or hypotension can result in acute-on-chronic arterial thrombosis.

Nonocclusive Mesenteric Ischemia

Nonocclusive causes of mesenteric ischemia are manyfold, but each results in a common final pathway, prolonged intestinal vasoconstriction and reduced intestinal blood flow. Typically these patients present with either shock/multisystem organ failure or after toxic pharmacologic ingestion. Common inciting pathology includes:

- Myocardial failure/cardiogenic shock[21]
- Septic shock[21]
- High-dose/prolonged vasopressor (α-agonist) infusions[21]
- Cocaine ingestion[22,23]
- Ergot poisoning[24]
- Digoxin toxicity[25,26]

This population historically has the worst in-hospital mortality rates among the various presentations of mesenteric ischemia. Presumably, this is due to the resulting mesenteric ischemia being a symptom of a more profound physiologic derangement, and not the primary disease process. This particular population is prone to significant delay in diagnosis, as they are often sedated or obtunded, which may further confound subjective reporting of symptoms. The gold standard for diagnosis is catheter-directed contrast angiography. Angiographic findings in nonocclusive mesenteric ischemia include[27]:

- SMA branch vessel origin narrowing
- Alternating dilation and narrowing "chain of lakes" or "string of sausages" sign
- Spasm of mesenteric arcades
- Impaired intramural vessel filling, "blush"

Mesenteric Venous Thrombosis

A less common form of AMI, caused by venous thrombosis, was first described in 1895 and later expounded by Warren and Eberhard.[1,28] Mesenteric vein thrombosis leads to impaired venous return, bowel-wall edema, impaired microvascular perfusion, bowel distention, and ultimately infarction of the involved segments of intestine. Mesenteric venous thrombosis is often secondary to a more global disease state. Possible predisposing pathology includes:

- Neoplastic disease processes
- Hypercoagulable syndromes
- Abdominal trauma
- Pancreatitis
- Severe dehydration
- Polycythemia vera

At present, neoplastic and hypercoagulable syndromes represent the most common inciting conditions. A thorough search for age-specific and gender-specific neoplasias as well as heritable hypercoagulable conditions (Leiden V, protein C and S deficiencies, antithrombin III deficiencies, prothrombin gene mutations, and antiphospholipid antibodies) should be undertaken as a secondary goal in the treatment of patients with mesenteric vein thrombosis. The most common vessels involved include the superior mesenteric vein (70%), the portal vein, and the inferior mesenteric vein.[29]

Presentation and Evaluation

Patients with acute and acute-on-chronic mesenteric ischemia will classically present with acute abdominal pain out of proportion to physical examination findings. A careful history may be all that is needed to diagnose each of these entities. A history of recent myocardial infarction or untreated atrial fibrillation combined with the acute onset of abdominal symptoms may be all that is needed to raise suspicion for central embolic sources. Furthermore, a prodrome of food fear, weight loss, and colicky abdominal pain that has acutely worsened may suggest acute-on-chronic arterial thrombosis.

It should be noted that the presence of bowel sounds does not exclude mesenteric ischemia. In fact, patients often experience increased expulsion of bowel contents with these processes. Presence of bowel sounds is a marker of early disease status, and if present should prompt aggressive movement toward therapeutic intervention in an attempt to maximize survival and reduce further complications. If diagnosis is delayed patients will precipitously decline, soon developing guarding, rebound, and other peritoneal signs. As the bowel becomes frankly necrotic, perforation ensues.

At this point, the "quiet abdomen" may be encountered, and this should be taken as an ominous sign. Beyond this, hemodynamic instability and multisystem organ dysfunction soon develop as acidosis worsens. Coagulopathy may also ensue. At this point, salvage becomes exceedingly more unrealistic, and mortality markedly increases.

Laboratory evaluation often shows evidence of hemoconcentration with marked leukocytosis. The leukocytosis often demonstrates a leftward shift with increased immature white cells. Increased base deficits along with elevated anion gap, lactate dehydrogenase, and amylase levels may also be seen.[30] These signs are all markers of late disease status, and, if present, should portend a guarded prognosis. Plain abdominal radiographs are helpful in ruling out alternative diagnoses, but have little utility in confirming the diagnosis of AMI.

Duplex ultrasonography of the mesenteric vessels is highly sensitive and specific; however, it is often technically limited by accompanying abdominal distention and overlying bowel gas.[31] The historical gold standard for the diagnosis of mesenteric ischemia was selective catheterization and mesenteric angiography. Although this continues to be a gold standard, CT, with its better image quality, expansion of availability, and ease of noninvasive image acquisition, has come to challenge catheter-based angiography.

Patients with nonocclusive mesenteric ischemia typically will show multiple areas of narrowing, consistent with a "string of beads" appearance. There will also be evidence of "pruning" of the more distal medium and small branch vessels, and loss of the submucosal blush normally seen with selective visceral angiography.

Patients with mesenteric venous thrombosis tend to present with a more protracted time course when compared with those patients with arterial-based ischemia syndromes. Often, symptoms will be present for 2 to 3 days before the diagnosis is made.[29] However, pain out of proportion to physical examination findings is still a hallmark of these patients' presentations. Rhee and colleagues[29] reviewed their 20-year experience with mesenteric venous thrombosis, and found that 75% of patients were symptomatic for more than 48 hours before diagnosis. In addition, abdominal pain (83%), anorexia (53%), and diarrhea (43%) were the most common presenting symptoms. Contrast-enhanced axial imaging with delayed venous phase is a key tool in the diagnosis of this abnormality. Failure to acquire delayed venous-phase images can hamper the diagnosis of this uncommon clinical entity. Duplex ultrasonography and magnetic resonance (MR) venography are alternative techniques for diagnosis, although they are less commonly used and not so readily available.

Treatment Options

Initial treatments should be aimed at stabilization and resuscitation of the patient, but these interventions should not delay or prolong the time to revascularization. Volume resuscitation, initiation of broad-spectrum antibiotics, and initiation of a heparin infusion are essential first steps. Secondary steps include insertion of nasogastric tubes, arterial lines, and Foley catheters, and central venous access. These interventions can be accomplished while the patient is prepared for anesthesia in the operating room, so as not to further delay revascularization.

The treatment of choice for arterial embolization is open surgical embolectomy and evaluation of bowel viability.[21] This procedure is performed via a generous midline laparotomy, and allows for rapid clearing of clot, both antegrade and retrograde, using transverse arteriotomy and embolectomy catheters. The transverse colon is retracted cephalad, and the duodenum mobilized to the ligament of Treitz. From here, the SMA can be palpated in the root of the mesentery. A transverse arteriotomy is made, and balloon-tipped embolectomy catheters are advanced proximally and

distally. Clearance of clot should be confirmed with multiple clot-free passes of the catheter, after which heparin flushing and primary repair of the arteriotomy is completed. At this point the bowel can be assessed, clearly nonviable portions resected, and the abdomen packed for second-look laparotomy in 24 to 48 hours.

Others have advocated for a primary percutaneous approach, especially in the setting of early intervention where peritoneal signs have not yet ensued.[32–36] This approach remains somewhat controversial, as direct visualization of the bowel is not an option during percutaneous interventions. If endoluminally directed transcatheter therapy is selected there should be a low threshold for surgical exploration and bowel evaluation, based on patient data and clinical examination.

For the management of acute-on-chronic mesenteric ischemia, open thrombectomy with either endarterectomy or distal bypass are the treatments of choice. Visceral bypass can be performed either antegrade, using the supraceliac aorta for inflow, or retrograde, using the common iliac artery for inflow.[37] Choice of conduit is important, as bowel viability and bacterial translocation may very readily occur. Native venous conduit is ideal and if not available, cryopreserved alternatives may be used. Prosthetic reconstructions are discouraged because of the high risk of graft infection if bowel failure occurs. Others have advocated the use of classic open abdominal embolectomy with retrograde stenting of the primary lesion.[38] This approach is particularly useful in the setting of compromised bowel when native conduit for bypass is not available, or when inflow vessels are severely diseased, precluding safe antegrade or retrograde bypass techniques.

Nonocclusive intestinal ischemia remains a difficult entity to treat. The mainstay of therapy involves correcting the primary issue, leading to compromised intestinal perfusion. Observation and resuscitation in an intensive care environment are essential.[21] Transcatheter infusion of vasodilators has been described, especially in the setting of illicit drug overdose or accidental therapeutic drug overdoses.[39,40] Despite these interventions, this population has one of the poorest survival rates, primarily because of the severity of comorbid conditions leading to this condition.

The essential treatment of mesenteric venous thrombosis is systemic anticoagulation. Initiation of an unfractionated heparin bolus, followed by continuous infusion, is critical. Secondary therapies include bowel rest, nasogastric decompression, and the intravenous administration of broad-spectrum antibiotics to guard against sequelae of bacterial translocation. Patients who develop peritoneal signs will require exploration and bowel resection. There have been conflicting reports about the utility of percutaneous, transhepatic catheter-directed thrombolysis in these patients. Some groups have advocated for its use,[41–43] whereas others have found a significantly increased mortality rate when used.[44] Most patients can be treated conservatively with anticoagulation, bowel rest, and observation. Those patients requiring more aggressive intervention after failure to respond to conservative measures have been shown to have a worse prognosis.[44]

CHRONIC MESENTERIC ISCHEMIA

Presentation

Contrary to most cardiovascular conditions, chronic mesenteric ischemia is represented by a preponderance of female patients, up to 60% in most reported series. The average age at presentation is 50 to 60 years. More than 75% of patients have current or former tobacco exposure, and most have concurrent hypertension, coronary artery disease, prior cerebrovascular accident, and renal insufficiency. Despite the relatively common finding of significant celiac and SMA stenosis rates

(50% and 30%, respectively) during autopsy series, true chronic mesenteric ischemia remains relatively rare,[45,46] owing in part to the rich collateral networks described earlier. It has long been thought that significant disease of at least 2 of the 3 major visceral branches is needed to compromise overall mesenteric flow.

First described by Councilman nearly 120 years ago, the cardinal symptom of chronic mesenteric ischemia remains postprandial abdominal pain. This pain usually sets in 1 to 2 hours after a meal and results in dull, crampy, or spasmodic waves of pain in the epigastrium or periumbilical abdomen. The pain is often in disproportion to other clinical examination findings. Over time, patients will learn to equate food consumption with pain elicitation, and the classic "food fear" or avoidance will develop. Along with this, a history of unintentional weight loss is often present. Alternatively, patients may report a history of continual low-volume grazing habits, another strategy to avoid bolus feeding, which increases visceral blood flow in response to digestive needs and precipitates abdominal angina. Patients may also present with increasing complaints of nausea, vomiting, diarrhea, and/or bloating after meals.

Diagnosis

Visceral duplex scanning plays a significant role in the evaluation of chronic mesenteric ischemia, contrary to its modest role in AMI. Evaluation of mesenteric vessel waveforms and peak systolic velocity (PSV) calculation has been shown to be highly sensitive and specific (**Table 1**).[47] Others have added preprandial and postprandial scanning techniques, which slightly improve specificity.[48] PSVs greater than 275 cm/s in the SMA and greater than 200 cm/s in the celiac artery have been demonstrated to correlate to stenoses of greater than 70% in each vessel.[47] As with arterial stenosis in general, poststenotic dilation and the presence of a turbulent jet are also supportive findings on duplex evaluation. In addition, retrograde flow in the common hepatic artery can be seen if severe celiac stenosis is present.

Because of the high negative predictive value and sensitivity of duplex ultrasonography, it makes an excellent screening examination for chronic mesenteric ischemia. Confirmatory evaluation with thin-slice CT angiography (CTA) or MR angiography is recommended if surgical or transcatheter interventions are being entertained. In addition, selective catheter-based angiography remains the gold-standard diagnostic modality.

Treatment Options

Both endovascular and open revascularization options exist for the treatment of chronic mesenteric ischemia. However, open surgical revascularization remains the treatment of choice when the patient's condition allows, either by bypass or endarterectomy. Visceral bypass can be performed either antegrade, using the supraceliac aorta for inflow, or retrograde, using the common iliac artery for inflow.[37] Native venous conduit is ideal when suitable segments can be obtained. If native vein is not available, prosthetic or cryopreserved alternatives may be used.

Antegrade bypass is completed via a midline incision with exposure of the distal thoracic aorta.[49] The triangular ligaments of the liver are divided, and the left lobe of the liver protected and retracted laterally out of the operative field. The gastrohepatic ligaments are divided carefully with electrocautery to gain entry into the lesser sac and periesophageal space. The esophagus is then retracted to the left lateral position, and the diaphragmatic crus and median arcuate ligament are divided. Care must be taken to protect the esophagus from injury during these steps. Once the crus is divided, the distal thoracic aorta can be identified and cleared to allow clamping and anastomosis. The CA is often immediately visible during this dissection; it can be traced to the right,

Table 1
Moneta visceral duplex criterion

Specific Mesenteric Artery	Peak Systolic Velocity (cm/s)	Corresponding Percent Stenosis	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Overall Accuracy (%)
SMA	>275	≥70	92	96	80	99	96
Celiac	>200	≥70	87	80	63	94	82

Abbreviations: NPV, negative predictive value; PPV, positive predictive value.
Data from Moneta GL, Lee RW, Yeager RA, et al. Mesenteric duplex scanning: a blinded prospective study. J Vasc Surg 1993;17:79–84 [discussion: 85–6].

allowing anastomosis to the hepatic or directly onto the bifurcation of the splenic and hepatic branches. If SMA revascularization is needed, blunt tunneling retrograde to the pancreas can be achieved to the left of the aorta (**Fig. 3**).[50]

Retrograde bypass can use the distal aorta or either iliac system as inflow. This region tends to be more prone to atherosclerotic change, but in many patients can be a suitable donor vessel. Care must be taken to avoid acute angulation or kinking of the bypass graft when this approach is used. Some have advocated the use of this approach only when:

- Emergency revascularization is needed
- Patient comorbidities preclude supraceliac clamping
- Prior foregut surgery prevents supraceliac dissection

The abdominal contents are packed to the right side of the abdomen, and the retroperitoneum overlying the distal aorta is incised in the usual fashion. A suitable portion of the distal aorta or iliac vessel is dissected free and prepared for anastomosis. The SMA is exposed as discussed previously, and if celiac revascularization is needed, exposure of the common hepatic artery can be accomplished with the same dissection described earlier for antegrade bypass. Blunt tunneling underneath the pancreas can again be used to bring the graft up to this region for anastomosis to the common hepatic artery, thus allowing for less anastomotic angulation and kinking associated

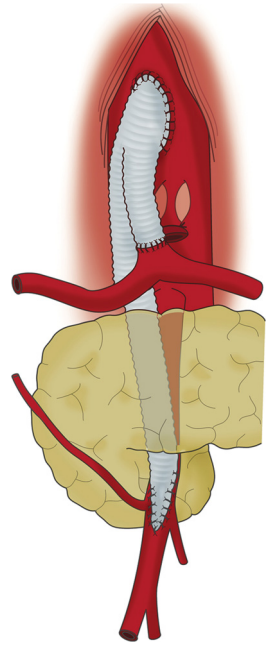

Fig. 3. Antegrade aortoceliac–superior mesenteric artery bypass. The proximal anastomosis originates from the supraceliac aorta, and the limbs of the graft are oriented on top of each other. The body of the graft should be left as short as possible. The celiac anastomosis can be performed in an end-to-end fashion. The inferior limb to the superior mesenteric artery is tunneled deep to the pancreas. The superior mesenteric anastomosis is performed end to side. (*From* Huber TS, Lee WA. Mesenteric vascular disease: chronic ischemia. In: Cronenwett JL, Johnston W, editors. Rutherford's vascular surgery. 7th edition. Philadelphia: Saunders/Elsevier; 2010; with permission.)

with the celiac revascularization. The bypass limb to the SMA should follow a "lazy reverse C" in the retroperitoneum to minimize the risk of kinking when the abdominal contents are returned to their native position (**Fig. 4**).[50]

Open surgical endarterectomy also remains a viable and durable option for chronic mesenteric ischemia. Endarterectomy offers definitive clearance of the obstructing lesions, and can be performed safely with excellent long-term results.[51] This procedure is best performed via a left thoracoabdominal incision. The diaphragm can be partially or completely divided radially; the retroperitoneal space is entered behind the left kidney, and the abdominal content rotated medially, along with the left kidney and spleen. The distal thoracic aorta is dissected free, and the crura overlying the CA divided only after the left renal vessel is clearly identified. The dissection is then carried distally to identify the SMA at its origin. Once proximal and distal control is obtained an aortic arteriotomy can be made, allowing eversion endarterectomy of the renal and visceral vessels. On completion, the arteriotomy can be closed primarily, or with a patch.

More recently, some have investigated the role of endovascular techniques for chronic mesenteric ischemia. Many groups have noted a reduced early mortality when endovascular techniques are used.[36,52] As with many endovascular techniques, the reduced early complication rate and mortality reduction comes with reduced long-term durability and increased need for reintervention.[53] When compared with open surgical revascularization, Kasirajan and colleagues[54] found an increased recurrence of symptoms in patients treated endovascularly. The Mayo group found that nearly

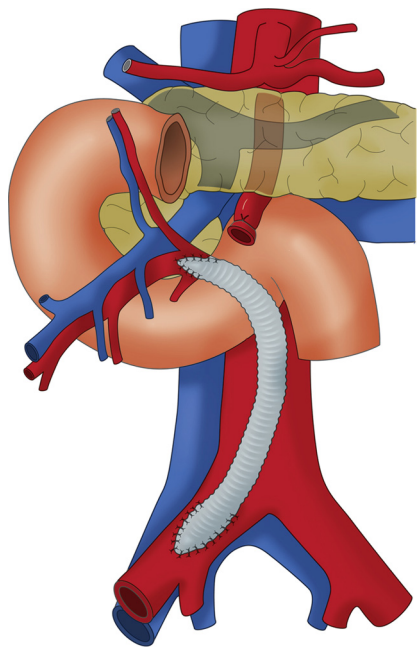

Fig. 4. Retrograde aorta–superior mesenteric artery bypass. The distal anastomosis is performed in an end-to-end or end-to-side fashion to the superior mesenteric artery after mobilization of the ligament of Treitz and the other duodenal peritoneal attachments. The bypass graft takes a gentle "reverse-C loop" as it transitions. (*From* Huber TS, Lee WA. Mesenteric vascular disease: chronic ischemia. In: Cronenwett JL, Johnston W, editors. Rutherford's vascular surgery. 7th edition. Philadelphia: Saunders/Elsevier; 2010; with permission.)

40% of endovascularly treated patients required reintervention within the first 2 years, but that this could be completed with low mortality, although complications related to the secondary procedure were as high as 27%.[53] The group at Dartmouth also confirmed the increased risk of restenosis and the need for reintervention with endovascular treatment.[55] However, these investigators did note that endovascular treatments served well to temporize some poor open surgical candidates, allowing for medical optimization, nutritional recovery, and subsequent open revascularization.[55]

COMPRESSIVE SYNDROMES

Median Arcuate Ligament Syndrome

Median arcuate ligament syndrome (MALS) shares many of the same presenting symptoms as the other forms of mesenteric ischemia. Episodic, crampy, upper abdominal pain associated with meal intake is a hallmark. Often an associated recent weight loss is also present, and many patients will also have symptoms of delayed gastric emptying. This process results from dynamic compression of the CA.[56] The hallmark diagnostic finding is dynamic compression of the celiac artery on expiration. On inspiration, the compression lessens or is absent. This disorder can be diagnosed with duplex ultrasonography, MR angiography, and conventional catheter-based angiography.[57] There has been much debate over the extent to which MALS contributes to potentially symptomatic mesenteric ischemia states, and MALS remains a diagnosis of exclusion.[58] Surgical treatment for MALS has been somewhat limited, and at present only case reports and small case series exist in the literature. There have been multiple reports of open or laparoscopic division of the ligament and crus with or without celiac ganglionectomy resulting in symptomatic relief.[59–62] The specific aim of treatment should encompass division of the restrictive fibroligamentous bands, with endovascular attempts at angioplasty and stenting as a secondary measure for persistent celiac stenosis.[61,63] Angioplasty alone, with or without stenting, is ineffective, if not contraindicated without proper decompression first and foremost.

SUMMARY

Mesenteric ischemia remains a rare clinical entity, but because of the grave consequences of missed or significantly delayed diagnosis, clinical suspicion must remain high. Abdominal pain, bloating, nausea, vomiting, and pain out of proportion to physical examination findings remain the hallmark of presentation. Catheter-based angiography is still the gold standard of diagnosis, but high-quality CTA is an acceptable alternative.

With regard to AMI, treatment options include open embolectomy, catheter-directed thrombolysis, visceral bypass, and/or mesenteric angioplasty and stenting. If percutaneous therapies are selected it is essential that the abdominal examination can be followed closely, and any concern over bowel integrity should prompt an exploratory intervention. Treatment of nonocclusive mesenteric ischemia includes therapy for the primary disease process, and some promising results have been seen with catheter-directed vasodilator infusions. Mesenteric venous thrombosis most often is a marker for systemic hypercoagulable states or occult malignancy. This disease can be treated most often with anticoagulation and a careful search for the inciting hypercoagulable-inducing condition.

Chronic mesenteric ischemia remains a disease of women in their fifth and sixth decade. Prolonged symptomatology and delayed diagnosis is often seen. Classic food fear, meal avoidance, and postprandial epigastric or periumbilical pain are hallmarks of this disease state. Open surgical revascularization is the mainstay of therapy,

but in nonideal surgical candidates endovascular techniques are an acceptable alternative. These endovascular interventions are associated with lower early mortality rates but seem to be less durable in the long term, often requiring repeat intervention to maintain patency.

Finally, MALS results in dynamic compression of the celiac access, and can be confirmed as a diagnosis of exclusion. Laparoscopic or open division of the compressive bands is the treatment of choice. After complete operative lysis, persistent symptoms with residual angiographic findings of stenosis can be treated with endovascular approaches, including angioplasty and stenting. Endovascular therapy alone should not be attempted until external compression has been completely removed by other techniques.

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