Mesenteric Ischemia

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ABSTRACT

Mesenteric ischemia is classified as either acute or chronic. The former is a life-threatening emergency in which a sudden reduction in intestinal blood flow may ultimately result in bowel infarction. The most common causes are arterial embolism, arterial thrombosis, nonocclusive mesenteric ischemia, and mesenteric venous thrombosis. A high index of suspicion, early diagnosis and rapid intervention are necessary so that normal mesenteric perfusion is restored before fatal bowel infarction can occur. Chronic mesenteric ischemia is usually caused by stenotic or occlusive disease involving the proximal segments of the mesenteric arterial supply to the bowel, usually as a result of atherosclerosis. Intestinal angina is the classic presentation, defined as recurrent postprandial abdominal pain that subsides in 1 to 2 hours, with associated weight loss and aversion to food. When combined with the clinical presentation, physical examination, and laboratory data, imaging plays a key role in the diagnosis of either acute or chronic mesenteric ischemia. Recognition of pertinent imaging findings and various treatment options may aid in preventing the serious and possibly fatal sequelae that may occur in cases of mesenteric ischemia.

KEYWORDS: Acute mesenteric ischemia, chronic mesenteric ischemia, nonocclusive mesenteric ischemia, intestinal angina

Objectives: Upon completion of this article, the reader should be able to (1) identify the pathophysiology and most common causes of both acute and chronic mesenteric ischemia, (2) state the diagnostic role of various imaging modalities, and (3) explain how the clinical presentation and imaging findings determine the appropriate treatment options.

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MESENTERIC ISCHEMIA

Mesenteric ischemia is classified as either acute or chronic. Acute mesenteric ischemia (AMI) is a life-threatening emergency in which an acute reduction in splanchnic blood flow threatens bowel viability and may ultimately lead to bowel necrosis. Normally short episodic reductions in mesenteric perfusion are well tolerated by the bowel, as ~20 to 25% of mesenteric

capillaries may be open with considerable reserve against changes in blood flow, but if there is a prolonged discrepancy between demand and supply, ischemia results.¹ Prolonged ischemia may ultimately cause tissue damage from reperfusion injury, resulting in increased microvascular permeability.² A high index of suspicion, early diagnosis and rapid intervention are necessary so that normal mesenteric perfusion is restored before fatal

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bowel infarction can occur. AMI may result from (1) arterial embolism, (2) arterial thrombosis, (3) nonocclusive mesenteric ischemia, and (4) mesenteric venous thrombosis. Less common causes include dissection, trauma, bowel strangulation, retroperitoneal fibrosis, fibromuscular dysplasia, segmental arterial mediolysis (SAM), and vasculitis.^{3,4}

Chronic mesenteric ischemia (CMI) occurs when there is stenotic or occlusive disease involving the proximal (usually ostial) segments of the mesenteric arterial supply to the bowel, usually as a result of atherosclerosis. Autopsy studies have shown a 6 to 10% incidence of >50% stenosis in one or more mesenteric arteries, yet the incidence of symptomatic CMI is significantly less.^{5,6} Because of the rich and redundant arterial supply of the bowel and the propensity to develop collaterals, CMI typically occurs only when there is disease of at least two of the three major mesenteric arteries: the celiac axis, superior mesenteric artery (SMA), and/or inferior mesenteric artery (IMA). Intestinal angina is the classic presentation, defined as recurrent postprandial abdominal pain that subsides in 1 to 2 hours, with associated weight loss and aversion to food.³ Because intestinal angina is rare, diagnosis may be delayed and patients may be symptomatic for months or years prior to seeking medical attention.

Contrast-enhanced computed tomography (CT) and CT angiography (CTA) are typically the initial imaging modalities employed in evaluating mesenteric ischemia. CTA depicts vascular anatomy in detail and may demonstrate stenoses and/or occlusions as well as any arterial collaterals. A noncontrast CT examination, to assess for the presence of vascular calcifications, should always be included. CT may also demonstrate nonvascular causes of abdominal pain such as inflammatory disease or neoplasm. Furthermore, there may be findings that will help differentiate between an acute and a chronic process; bowel wall thickening, pneumatosis, and portomesenteric gas indicate an acute process and are absent in CMI.

The accepted diagnostic gold standard for diagnosing either acute or chronic mesenteric ischemia is angiography. If the history suggests embolic or thrombotic occlusion with an acute presentation, the best management is prompt surgery, as this has the best chance to save threatened, but uninfarcted bowel. Patients with a more delayed presentation may be considered for arteriography, as the advantages of added information probably outweigh risks of further intestinal infarction. This may be performed emergently in suspected AMI if there are no peritoneal signs. In such cases, angiography must be rapidly available, as a delay in diagnosis could ultimately prove fatal. Elective angiography is usually appropriate for suspected CMI; in addition to confirming the diagnosis, catheter-based

interventions are often employed in the treatment of either acute or chronic mesenteric ischemia. For either entity anteroposterior and lateral aortography combined with selective celiac, SMA, and IMA angiography are typically performed, and include a prolonged filming sequence that incorporates the venous phase in the evaluation.

ACUTE MESENTERIC ISCHEMIA

As previously noted, there are multiple causes for an acute reduction in arterial or venous intestinal blood flow. One of the most common causes is embolic arterial occlusion, implicated in up to 50% of cases,⁷ typically involving the SMA just distal to the middle colic artery origin (Fig. 1). This causes vasoconstriction of the arterial branches originating beyond the embolic occlusion, or additional distal embolization, further worsening the extent of bowel ischemia. The embolus usually is from a cardiac source and therefore risk factors such as myocardial infarction, arrhythmia, valvular disease, and ventricular aneurysm should be considered and appropriately excluded.⁷

Mesenteric ischemia that results from SMA thrombosis usually is caused when a preexisting atherosclerotic stenosis, typically at the vessel origin, reaches a critical diameter and acutely thromboses. Because the thrombotic occlusion occurs proximal to the middle colic artery, midgut arterial perfusion is completely

Figure 1 Superior mesenteric artery arteriogram shows a large embolus that appears as a central filling defect (large black arrow) and has occluded the distal portion of the arterial trunk (small black arrow). A very small portion of the jejunum remains perfused (small white arrow). Note the abnormally dilated bowel loops (large white arrows) in this patient with acute mesenteric ischemia.

Figure 2 Digital subtraction angiography (DSA) image shows thrombosis (small black arrows) of the superior mesenteric artery that occurred distal to a high-grade stenosis (large white arrow). There is very little perfusion distal to the thrombosis. Note the previously placed stent in the celiac trunk (large black arrow), which is also occluded.

interrupted, particularly if there are poorly formed collaterals (Fig. 2). Thus, there is usually a worse prognosis in acute ischemia due to SMA thrombosis than with embolus because emboli often lodge distally in the SMA, and allow continued partial perfusion of the proximal intestine (Fig. 3).

Nonocclusive mesenteric ischemia (NOMI) results from severe and prolonged visceral arterial vasoconstriction, and may occur in the setting of severe systemic illness with circulatory insufficiency and end-organ shock. Thus, there is no obstructive cause for the diminished perfusion, but rather there is reduced perfusion pressure. These hypotensive or hypovolemic states may persist for hours and the intestinal mucosal permeability increases in direct proportion to the duration and severity of the vasoconstriction. In a shock setting, the normal autoregulatory mechanisms fail to permit reperfusion before intestinal necrosis occurs. This entity also occurs in response to drug intoxication, e.g., cocaine, digitalis, dopamine and ergot derivatives, as well as following coarctation repair. The incidence may be decreasing, with the improved treatment of critically ill patients and with a shift in emphasis from the use of peripheral vasoconstrictors.

Intestinal ischemia that results from venous thrombosis is uncommon. One study reported that 0.38% of laparotomies performed for “acute abdomen” revealed intestinal ischemia and only 17% of those were from venous thrombosis.⁷ Primary occlusions have no

Figure 3 A small embolus (white arrow) has lodged distally in the superior mesenteric artery and this location allows for continued perfusion of most of the bowel in this arterial territory. This contrasts with the severe bowel ischemia that occurs with proximal embolic or thrombotic occlusion.

known underlying cause whereas secondary thromboses are associated with other disorders such as hypercoagulable states, trauma, abdominal surgery, neoplasm, inflammatory bowel disease, sepsis, peritonitis, and pancreatitis. Most disorders causing SMA thrombosis have variants of Virchow's triad: conditions promoting portal venous stasis, splanchnic venous injury, and hypercoagulability. The process typically originates in the venous arcades and extends centrally to involve the SMV and the portal vein, with the inferior mesenteric vein (IMV) less often involved. SMV thrombosis may cause hemorrhagic gut infarction typically confined to the small intestine, as a result of bowel wall edema that reduces arterial perfusion.

Clinical Aspects

Acute mesenteric ischemia occurs more frequently in elderly patients who have associated comorbidities such as systemic atherosclerotic disease, hypertension, congestive heart failure, cardiac arrhythmias, coronary artery disease, or hypovolemia and hypotension. The onset of symptoms is typically acute and consists of severe abdominal pain that is disproportionate to the findings on physical examination. Pain may be accompanied by nausea, vomiting, gastrointestinal hemorrhage, leukocytosis, and acidosis, as evidenced by an altered pH and elevated serum lactate.

Figure 4 Noncontrast computed tomography (CT) image shows characteristic abnormalities that may be present in the later stages of acute mesenteric ischemia: pneumatosis intestinalis (small white arrows), intravascular air within the main portal vein (large white arrow) as well as within intrahepatic branches and small foci of free air (small black arrows) representing pneumoperitoneum.

Imaging of Acute Mesenteric Ischemia

Plain radiographs are normal in up to 25% of cases of AMI and when abnormal, the findings are nonspecific ones that may include bowel dilatation, thumb printing from bowel wall edema or hemorrhage, and pneumatosis, portal venous gas, or pneumoperitoneum from bowel infarction.

CT may demonstrate bowel dilatation and wall thickening, as well as ascites and mesenteric edema in the early stages of AMI, whereas pneumatosis, pneumoperitoneum, and intravascular gas are seen in later stages (Fig. 4).⁸ In cases of embolic occlusion, other abdominal organs such as the kidneys may show evidence of infarction.

CTA is more specific and will often demonstrate the site of embolic occlusion as a central filling defect within the SMA or may show an abrupt point where the artery is not opacified, with poor or absent collaterals. Similar findings of arterial nonopacification are usually present in cases of SMA thrombosis, and there may be accompanying vascular calcifications at the origins of the celiac, SMA, and/or IMA along with well-formed collaterals. AMI that results from venous thrombosis may have CTA findings of a central persistent low-attenuation filling defect within the vein lumen that may be surrounded by mural rim-enhancement.⁹ The SMV and portal veins may exhibit these findings and multiple sites of involvement may be evident (Figs. 5, 6). CTA is

Figure 5 Axial image from the venous phase of a contrast-enhanced computed tomography (CT) scan shows an eccentric central filling defect (black arrow) in the superior mesenteric vein representing mesenteric vein thrombosis.

nonspecific in cases of NOMI, although the arterial calibers may be extremely diminutive.

Gadolinium-enhanced magnetic resonance angiography (MRA) can be used to demonstrate arterial causes of AMI such as embolus or thrombotic occlusion

Figure 6 Coronal venous-phase maximum intensity projection (MIP) image from contrast-enhanced CT shows that the caliber of the superior mesenteric vein is diminutive (large white arrows) relative to that of the inferior mesenteric vein (black arrowheads) and that there is a further size discrepancy to the superior mesenteric vein (SMV) at the junction (large black arrows) of the splenic vein (small white arrow) with the portal vein (small black arrow), representing partial SMV thrombosis. Note the resulting engorgement of the venous arcades (white arrowheads).

and is an excellent modality to evaluate mesenteric vein thrombosis, but the long examination times and often poor availability may result in unacceptable delays in diagnosis and treatment.¹⁰

As previously noted, angiography is the gold standard for the detection of SMA embolism and may also demonstrate collateral vessels and visceral arterial stenoses and occlusions in cases of thrombotic occlusion. An acute embolus appears as a filling defect, which completely or partially obstructs the artery, with absent collateral vessels. The so-called tram-track appearance occurs when the embolus is partially outlined by contrast that courses between the embolus and the arterial wall (Fig. 7). Selective mesenteric arteriography can be performed if the vessel origin is patent and can assess for distal occlusion; additionally catheter-directed intervention may be employed, if appropriate. The invasive and time-consuming nature of angiography, coupled with potential nephrotoxicity procedure-related morbidities have served to reduce its role in the initial evaluation of acute mesenteric ischemia, particularly given the wealth of diagnostic information that CT and CTA provide. Angiography is now typically reserved for cases in which the diagnosis is equivocal and a delay in treatment is acceptable, or when catheter-based therapeutic options such as thrombolysis, aspiration of a thrombus or embolus, or vasodilator therapy may potentially be used.

Figure 7 An acute embolus within the superior mesenteric artery appears as a filling defect (large black arrow) partially obstructing the artery. Smaller filling defects distally (small black arrows) represent fragmentation of the embolus. Note the so-called tram-track appearance in which the filling defects are partially outlined by contrast that courses between the emboli and the arterial wall.

Because of the poor ability of noninvasive imaging modalities to accurately assess the terminal mesenteric vasculature it has limited usefulness in evaluating vasospastic disorders. Therefore, at present, if NOMI is suspected angiography may be valuable in clarifying the diagnosis, and may be coupled with vasodilator infusion therapy. Typical findings in NOMI seen at selective mesenteric angiography include diffuse constriction of vessels with reduced opacification of the bowel parenchyma, alternating areas of segmental constriction, producing a “string of sausages” sign in the major arterial branches, a reflux of contrast material into the aorta, and a normal mesenteric venous phase (Fig. 8).⁷

Treatment Options

Once the diagnosis of AMI is established, prompt treatment is imperative as delay may result in irreversible and potentially fatal bowel ischemia. Initial therapy should include fluid resuscitation, nasogastric tube decompression, and if possible, anticoagulation and avoidance of vasoconstricting agents.¹¹ Broad-spectrum antibiotics should be administered early. Anticoagulation is the primary therapy in cases of mesenteric vein thrombosis, potentially with mandatory long-term anticoagulation if a primary clotting disorder is discovered.

Figure 8 Selective superior mesenteric artery arteriogram in a patient with nonocclusive mesenteric ischemia (NOMI) secondary to vasopressor therapy shows a diminished caliber to the main arterial trunk (white arrows) and vasoconstriction of the branch vessels (black arrowheads). Alternating areas of vasoconstriction (white arrowheads) have produced a “string of sausages” sign.

For any cause of AMI, laparotomy is indicated if there are peritoneal signs. In most patients, emergent surgical embolectomy or bypass, with resection of any nonviable bowel is usually necessary. It is not possible to predict accurately the viability of ischemic-appearing bowel, so revascularization should precede resection, as restoration of pulsatile flow to the SMA may produce remarkable changes in bowel segments that initially appeared irreversibly ischemic. A "second-look laparotomy" is often necessary after the initial surgery, as postoperative vasospasm of previously normal vessels may further compromise bowel viability in some patients. Additionally, the initial intraoperative assessment of the bowel may have failed to detect all nonviable segments and a second-look is needed to ensure all obviously necrotic bowel is removed.¹¹

Catheter-directed therapies have been employed in treating AMI with various degrees of success. Outcomes are dependent upon the severity of ischemia, as percutaneous therapies such as catheter-directed thrombolysis or vasodilator infusion may require longer treatment times than surgery.¹² There have been multiple reports of the successful use of thrombolysis in cases of both acute embolic and thrombotic SMA occlusion (Fig. 9),¹³ but this requires a patient without clinical evidence of impending bowel necrosis. Direct intraarterial vasodilator infusion therapy should be considered as

Figure 9 Catheter-directed thrombolysis was used to treat the patient with superior mesenteric artery thrombosis who was illustrated in Fig. 2, with restoration of patency (black arrows) to the previously thrombosed main arterial trunk. Note the improved opacification of the distal superior mesenteric artery branches. The thrombosed celiac artery stent (black arrowheads) was not treated.

the primary treatment in cases of NOMI; typically a 60 mg bolus of papaverine is administered selectively into the SMA, followed by continued selective transcatheter infusion at rates of 30 to 60 mg/hour. Recently, successful treatment of NOMI with continuous intravenous high-dose PGE₁ administration (dose, 0.01–0.03 μ g/kg per min; mean administration period, 4.8 days) has been reported; as in all causes of AMI, a high index of suspicion and early intervention resulted in better survival.¹⁴

CHRONIC MESENTERIC ISCHEMIA

Symptomatic mesenteric ischemia that results from chronic occlusive disease of the intestinal arterial supply is less common than AMI. There is a failure to achieve normal postprandial hyperemic arterial intestinal flow as a result of stenotic or occlusive disease of the mesenteric arteries (Fig. 10). The limited arterial flow causes an imbalance between the oxygen demand and supply, anabolic byproducts are produced and postprandial pain analogous to angina pectoris occurs.

Figure 10 Lateral aortogram shows high-grade stenoses of the celiac (large white arrow) and superior mesenteric artery (small white arrow) origins, a typical location for an atherosclerotic etiology for chronic mesenteric ischemia. Chronic mesenteric ischemia usually occurs only when there is disease of at least two of the three major mesenteric arteries.

Clinical Aspects

The typical patient is an older woman who has multiple risk factors for atherosclerosis or has a history of prior abdominal surgery. The classic history is a chronic one of abdominal pain that occurs 30 to 45 minutes after eating, sometimes associated with nausea, vomiting, diarrhea, or malabsorption. With progression patients may develop fear of eating, with resultant weight loss, so that abdominal malignancy may be suspected. Although atherosclerosis is the most common etiology, CMI may also result from extrinsic vascular compression by the median arcuate ligament (Fig. 11), tumor, inflammatory processes, vasculitis, dissection, fibromuscular dysplasia, radiation, vasoconstricting medications, and prior abdominal surgery. Untreated cases of CMI may progress to acute mesenteric ischemia, usually as a result of thrombosis of a high-grade orificial arterial stenosis.

Imaging of Chronic Mesenteric Ischemia

The initial imaging evaluation of suspected CMI should involve a noninvasive modality such as CT, magnetic resonance imaging (MRI), or ultrasound. Further evaluation with angiography may be necessary if the initial studies are inconclusive or if surgical planning or a catheter-based intervention are indicated. As in acute

mesenteric ischemia, contrast-enhanced CT and CTA are the primary modalities initially employed in the evaluation of suspected CMI. CT can confirm or exclude alternate etiologies for the abdominal pain, whereas CTA may detect vascular calcifications, arterial stenoses and/or occlusions, and collateral vessels (Fig. 12).

As in cases of AMI, gadolinium-enhanced MRA provides an accurate evaluation of the mesenteric vasculature, but is not as sensitive as CT in screening for other abdominal abnormalities. The mesenteric vasculature may be better demonstrated through the use of three-dimensional imaging techniques and postprocessing such as maximum intensity projections and volume rendering. The severity of arterial stenoses may be overestimated and calcifications are poorly demonstrated, however.

There are also MR techniques that may be used to evaluate mesenteric perfusion before and after a food challenge. Cine phase contrast imaging (PC) assesses superior mesenteric vein (SMV) flow and MR oximetry (MRO) evaluates the oxygenation of SMV blood.¹⁰ The mesenteric blood flow should normally increase after a food challenge and thus a decreased SMV flow response implies CMI on PC imaging.¹⁰ With the inability to increase mesenteric blood flow in CMI,

Figure 11 Chronic mesenteric ischemia may rarely be caused by extrinsic compression of the celiac artery by the median arcuate ligament of the diaphragm. This results in proximal arterial narrowing (white arrow) ~5 to 10 mm from the origin, with a characteristic upward hooking of the celiac artery. This controversial condition may be associated with upper abdominal pain, weight loss, hyperemesis, and an epigastric bruit caused by inadequate blood flow through the celiac artery, with resultant ischemia.

Figure 12 Three-dimensional computed tomography angiography shows an extremely severe superior mesenteric artery stenosis (small white arrow), an occluded celiac trunk (large white arrow), and an enlarged and patent inferior mesenteric artery (black arrow) in a patient with symptoms of intestinal angina. The initial imaging evaluation of suspected chronic mesenteric ischemia should involve a noninvasive modality such as this.

Figure 13 Duplex ultrasound of the superior mesenteric artery demonstrates a peak systolic velocity (PSV) >275 cm/second correlating with a stenosis of at least 70%.

Figure 14 Lateral aortogram shows high-grade stenoses of both the celiac (black arrow) and superior mesenteric (white arrow) arteries in a patient who had a chronic history of postprandial epigastric pain and weight loss.

oxygen extraction is increased in the mesenteric blood and MR oximetry takes advantage of this physiology. The increased oxygen extraction reduces the oxygen saturation and consequently reduces the T2 value of SMV blood.¹⁰ Postprandial reduction of the T2 value of blood in the SMV has been demonstrated in patients with symptomatic CMI, in contrast to a rise in T2 in asymptomatic patients.¹⁰

Duplex ultrasonography may identify high-grade stenotic lesions by demonstrating elevated fasting SMA and/or celiac arterial peak systolic velocities, with a peak systolic velocity >275 cm/second correlating with a stenosis of at least 70%. A comparison of pre- and postprandial SMA and/or celiac peak systolic velocities may suggest vascular compromise, but currently this is not useful as a true stress test for CMI (Fig. 13).

Angiography is often used in cases of CMI, either for confirmation of the diagnosis and surgical planning or for endovascular treatment. The imaging sequence is the same as in AMI and includes anteroposterior (AP) and lateral aortography with selective catheterization of the mesenteric arteries. If appropriate, angioplasty and/or stenting may be then used for revascularization.

Treatment Options

Surgical or endovascular revascularization are indicated both for relief of symptoms and to prevent progression to life-threatening AMI. Surgical treatment options include endarterectomy, vascular bypass,

Figure 15 The patient in Fig. 14 underwent endovascular treatment with placement of an intravascular stent (black arrows) in the superior mesenteric artery, as seen on this SMA arteriogram.

Figure 16 Stent placement has restored a normal luminal caliber to the superior mesenteric artery (white arrow), as seen on this lateral aortogram. Although the initial technical success rates usually exceed 90%, recurrent stenosis occurs frequently, requiring reintervention. The reported cumulative primary and assisted patency rates after these treatments currently are lower than with surgical revascularization.

and arterial reimplantation.¹⁵ In most instances, revascularization of only one vessel (usually the SMA) is needed despite the presence of multivessel disease, with high long-term patency rates (70 to 93%), but reported associated morbidities of up to 29% and mortalities of 7%.¹⁵

Endovascular treatment usually consists of angioplasty and stenting of the stenotic vascular segment, with reported high initial technical success rates that exceed 90% (Figs. 14–16).^{16–19} Recurrent stenosis occurs frequently, however, requiring reintervention. The reported cumulative primary and assisted patency rates after these treatments are lower than surgery^{16–19} and the current recommendation is one of individualized application of surgery or endovascular treatment in accordance with individual patient anatomic and comorbidity considerations.^{15,16}

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