Acute Mesenteric Ischemia

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Acute mesenteric ischemia is caused by a critical reduction in intestinal blood flow that frequently results in bowel necrosis and is associated with a high mortality. Clinicians must maintain a high index of suspicion because a prompt diagnosis and early aggressive treatment before the onset of bowel infarction results in reduced mortality. Medical management includes aggressive rehydration and the use of antibiotics, anticoagulation, vasodilators, and inhibitors of reperfusion injury. If acute mesenteric ischemia is suspected, early angiography is imperative, as it permits accurate diagnosis and possible therapeutic intervention. Therapeutic options during angiography depend on the cause of ischemia and include administering intra-arterial vasodilators and/or thrombolytic agents and angioplasty with or without stent placement. If interventional techniques are not possible or if the patient presents with suspicion of bowel infarction, surgery is warranted. Surgical techniques include superior mesenteric artery embolectomy or visceral artery bypass, which should be used before bowel resection to ensure only resection of nonviable bowel.

Introduction

Acute mesenteric ischemia (AMI) refers to the sudden reduction of intestinal perfusion, which can be due to occlusive or nonocclusive obstruction of arterial or venous blood flow. AMI is a potentially fatal vascular emergency with overall mortality of 60% to 80%, and its reported incidence is increasing [1–5]. Of those surviving, 20% to 60% develop short gut syndrome. The common end point in AMI is a pathophysiologic process that results in bowel ischemia and/or necrosis. Despite advances in diagnosis and treatment, the survival rate has not improved substantially during the past 70 years due to continued difficulty in recognizing the condition before bowel infarction occurs [6,7].

The clinical presentation is nonspecific in most cases. Classically, AMI is characterized by severe pain out of proportion to physical examination findings. Physical examination does not reliably differentiate between ischemic and infarcted bowel. Patients frequently present with symptoms compatible with other diagnoses, such as ileus, peritonitis, pancreatitis, or diverticulitis. The risk factors for AMI and the clinical course differ depending on the underlying etiology [8,9]. As bowel ischemia progresses, severe metabolic derangements ensue. If they are not addressed early in the clinical course, they lead to a series of events culminating in multiple organ dysfunction and death. The timely use of diagnostic and therapeutic methods to quickly restore blood flow is paramount in reducing the high mortality rate associated with AMI [10].

AMI has four specific causes: arterial embolism, arterial thrombosis, nonocclusive mesenteric ischemia (NOMI), and mesenteric venous thrombosis (MVT). Arterial embolism, the most common cause, is responsible for approximately 40% to 50% of cases [2,3]. Most mesenteric emboli originate from a cardiac source as the result of a mural thrombus that subsequently embolizes to the mesenteric arteries. Myocardial ischemia or infarction, atrial tachyarrhythmias, endocarditis, cardiomyopathies, ventricular aneurysms, and valvular disorders are all risk factors for intracardiac thrombus development. Nearly one third of all patients with a superior mesenteric artery (SMA) embolus have a history of an antecedent embolic event. Symptom onset is usually dramatic and sudden as a result of the poorly developed collateral circulation. Most arterial emboli preferentially lodge in the SMA because it emerges from the aorta at an oblique angle. More specifically, most emboli tend to lodge in the SMA distal to the origin of the middle colic artery, the SMA's first major branch [5,11]. The SMA embolism diagnosis usually can be made intraoperatively based on the distribution of the ischemic bowel. Because most SMA emboli lodge distal to the origin of the inferior pancreaticoduodenal branches, the proximal jejunum is spared, whereas the rest of the small bowel is ischemic or infarcted.

Acute mesenteric thrombosis accounts for 25% to 30% of all ischemic events and almost always occurs in the setting of severe atherosclerotic disease [2–4]. Approximately 50% to 75% of presenting patients have had prior symptoms compatible with chronic mesenteric ischemia. Patients with this condition may have a more insidious onset of disease, as its chronic nature allows important collaterals to develop. Bowel ischemia or infarction ensues when the last remaining visceral artery or

important collateral artery is compromised. Because the occlusion typically occurs at the origin of the SMA, the extent of bowel ischemia or infarction typically is greater than that with embolism, extending from the duodenum to the transverse colon.

NOMI accounts for approximately 20% of patients with mesenteric ischemia [8,12]. NOMI's pathogenesis is poorly understood but often involves a low cardiac output state associated with diffuse mesenteric vasoconstriction. Rather than having a functional anatomic obstruction, splanchnic vasoconstriction in response to hypovolemia, decreased cardiac output, hypotension, or vasopressors best explains the difference between this entity and other forms of AMI. Vasoactive drugs, particularly vasopressin, α-agonists, and digoxin, all have been implicated in the pathogenesis of NOMI [13]. Watershed areas of circulation are more vulnerable in NOMI. Because this condition frequently affects critically ill patients who have considerable comorbidities, the onset may be insidious, and the mortality rates are high. A form of nonocclusive ischemia has been described in critically injured trauma patients or patients who have undergone the stress of a major surgical procedure and are receiving enteral nutrition. AMI's reported incidence in these patients is 0.3% to 8.5% [14•]. The proposed mechanism is an imbalance between demand (created by the enteral feedings) and supply (decreased by systemic hypoperfusion and mesenteric vasoconstriction).

MVT is the least common cause of mesenteric ischemia, representing 10% to 15% of patients with AMI [2,3]. Historically, most cases have been classified as idiopathic. However, with improved diagnostic techniques, more cases have been shown to be related to primary clotting disorders, with only 10% now being classified as idiopathic [15,16]. MVT usually occurs in a segmental fashion and is characterized by edema and hemorrhage of the bowel wall that ultimately leads to necrosis and focal sloughing of the mucosa. Hemorrhagic infarction occurs when the intramural vessels are occluded. Because arterial flow is not compromised until late, the clinical presentation is usually more vague and protracted. The superior mesenteric vein is most often affected, with involvement of the inferior mesenteric vein and the large bowel being uncommon. Mortality depends on the acuity or chronicity of the venous thrombosis and the extent of venous involvement. Patients with acute mesenteric venous thrombosis have a 30-day mortality of 60% to 70%, whereas patients with the chronic form of the disease have a mortality of 20% [16].

Diagnosis

Because AMI may rapidly progress toward intestinal infarction and subsequent death, prompt diagnosis and treatment are paramount. A high index of suspicion in the setting of a compatible history and physical examination serves as the cornerstone of early diagnosis of mesenteric ischemia [7,17]. AMI should be considered in the differential diagnosis of any patient more than 60 years old who has a recent history of myocardial infarction or congestive heart failure and/or a prior history of atrial fibrillation, arterial emboli, or postprandial abdominal pain and weight loss. Survival approaches 50% when the diagnosis is made within 24 hours after symptom onset but drops sharply to 30% or less when the diagnosis is delayed [11]. Laboratory studies are nonspecific, with the most common laboratory abnormalities being hemoconcentration, leukocytosis, and an anion gap acidosis. High levels of serum lactate, amylase, aspartate aminotransferase, lactate dehydrogenase, and creatine phosphokinase are commonly observed, but none is sufficiently sensitive or specific to be diagnostic. Hyperkalemia and hyperphosphatemia usually are late signs and frequently are associated with bowel infarction [18].

The findings on abdominal plain films are also nonspecific. The classic radiographic findings of thumbprinting or thickening of bowel loops occur in less than 40% of patients at presentation. Abdominal CT scanning in general has poor sensitivity and specificity in the diagnosis of most types of AMI. Multislice technology recently has been introduced for CT angiography, and this may further improve the CT results in AMI [19]. CT scanning may be helpful in excluding other pathologies. CT is more sensitive (90%) in diagnosing venous thrombus than other types of AMI and is the investigation of choice when MVT is suspected [20]. The presence of pneumatosis intestinalis on CT does not necessarily indicate the presence of transmural infarction; however, transmural infarction is more common in patients with pneumatosis and portomesenteric venous gas [21]. Several recent studies suggest that magnetic resonance angiography and magnetic resonance venography may be highly sensitive for diagnosing MVT [22,23].

Duplex ultrasonography can visualize stenoses or occlusions in the celiac or superior mesenteric arteries. However, this study is often technically limited by distended, air-filled bowel loops. Also, its sensitivity is very limited in the setting of more distal emboli or in assessing NOMI. For these reasons, duplex ultrasound has no role in diagnosing AMI.

In the absence of clinical signs that would indicate emergent laparotomy (eg, peritonitis, perforated viscus), mesenteric angiography remains the investigation of choice when AMI is suspected. Several studies demonstrate improved survival rates when early angiography is implemented [15,21,24]. Angiography must be biplanar [25]. Although the anteroposterior view is best for visualizing the distal mesenteric vasculature, lateral aortography is better for visualizing the origins of the major visceral arteries, as they project anteriorly and overlay the aorta in the anteroposterior plane. Mesenteric angiography usually can differentiate embolic from thrombotic occlusions by the nature of the filling defect patterns. MVT is characterized by a generalized slowing and vasoconstriction of the arterial flow in conjunction with lack of opacifi-

cation of the corresponding mesenteric or portal venous outflow tracts. In MVT, this is usually segmental. This is in contrast to NOMI, which is usually diffuse and shows normal venous runoff. Also, NOMI characteristically shows narrowing and multiple irregularities of the major SMA branches, the "string of sausages" sign.

Treatment

AMI treatment can be categorized into four key principles: prevention; early diagnosis and treatment; proper intraoperative decision making; and aggressive postoperative management to avoid reperfusion injury, prevent clot propagation, and thwart septic complications. Studies have shown that 50% to 75% of patients with mesenteric artery thrombosis had prior symptoms of chronic mesenteric ischemia, and 33% to 75% of patients had a history of atrial fibrillation and were not on anticoagulants [26,27].

Once the AMI diagnosis is suspected, the stable patient without peritoneal signs should proceed to angiography without delay. When angiography is used to establish the diagnosis, the angiographic catheter should be left in the SMA for infusions of papaverine or vasodilators, if possible. Papaverine, a phosphodiesterase inhibitor, increases mesenteric blood flow to the marginally perfused viscera and typically is used in the setting of arterial embolic disease or NOMI. Although tissue-type plasminogen activator delivered as an infusion directly to the thrombus has been reported, especially in patients with mesenteric venous disease, this must be weighed against the possibility of the patient developing bowel infarction during the time it takes to potentially lyse the clot and the potential for bleeding [28]. Percutaneous suction SMA embolectomy or percutaneous transluminal angioplasty/stenting rarely has been described in AMI and should only be attempted in very specific instances [29,30••].

Concomitant treatment should include active resuscitation, aggressive rehydration, and treatment of the underlying condition. If there are no contraindications to anticoagulation, then intravenous heparin should be administered to achieve an activated partial thromboplastin time of 50 to 70 seconds to reduce the chance of further progression of the arterial or venous thrombosis. Once the patient's hemodynamics have improved and anticoagulation has been initiated, efforts should aim to reduce the mesenteric vasospasm associated with reperfusion. Intravenous glucagon can be infused initially at 1 μg/kg per minute and titrated up to 10 μg/kg per minute as tolerated. Allopurinol, angiotensin-converting enzyme inhibitors, and other oxygen radical scavengers may help to reduce the reperfusion syndrome and improve overall mortality [31]. Preventing septic complications includes the liberal use of broad-spectrum antibiotics to cover gram-positive, gram-negative, and anaerobic bacteria.

If the decision is made to go to the operating room, revascularization should take place before bowel resection in an effort to salvage as much small bowel as possible and to avoid the devastating effects of "short gut" syndrome. In situations in which the bowel viability is questionable, a "second-look" operation 24 to 48 hours later may be warranted [32].

Pharmacologic treatment

Crystalloids

Most patients presenting with AMI will be dehydrated and will require large amounts of intravenous fluids.

Lactated Ringer's solution

Standard dosage for lactated Ringer's solution is up to 100 mL/kg. Contraindications include cardiac insufficiency and pulmonary edema; the main side effects are peripheral edema and pulmonary edema. It is low in cost and very cost effective.

Broad-spectrum antibiotics

Broad-spectrum antibiotic therapy is used to protect against bacterial translocation from the ischemic bowel segment. Coverage should include gram-positive, gram-negative, and anaerobic bacteria. All the broad-spectrum antibiotics described subsequently are relatively inexpensive and very cost effective.

Levofloxacin

Levofloxacin is to be used with metronidazole or with piperacillin/tazobactam, as described subsequently. Standard dosage is 500 mg intravenously every 24 hours. Contraindications include hypersensitivity, prolonged QT interval, and concomitant class IA or III antiarrhythmic. It also should be used with caution in older adult patients and those with renal failure or seizure disorder. Its main drug interactions are mesoridazine, ranolazine, thioridazine, acarbose, acetohexamide, benfluorex, chlorpropamide, gliclazide, glimepiride, glipizide, gliquidone, glyburide, insulin, lidocaine, metformin, miglitol, tolazamide, tolbutamide, and troglitazone. Side effects include diarrhea, abdominal pain, dyspepsia, pseudomembranous colitis (rare), and congestive heart failure (rare).

Metronidazole

The standard dosage of metronidazole is a loading dose of 15 mg/kg (1 g for a 70-kg adult) administered intravenously over 1 hour for life-threatening conditions. Maintenance infusion is 7.5 mg/kg (500 mg for a 70-kg adult) intravenously over 1 hour, every 6 to 8 hours starting 6 hours after loading dose. Total dose should not exceed 4 g/d. Alternatively, 500 mg intravenously may be administered every 6 hours.

Hypersensitivity is the only contraindication to metronidazole. However, it is to be used with caution in cases of central nervous system disorder. The dose should be adjusted

in hepatic disease, and the patient should be monitored for seizures and development of peripheral neuropathy. It may increase toxicity of anticoagulants, lithium, and phenytoin; cimetidine may increase toxicity of metronidazole. A disulfiram-like reaction may occur as and 48 hours after ethanol is orally ingested. Main side effects include dyspepsia, diarrhea, metallic taste, dry mouth, and pruritis.

Piperacillin/tazobactam
Standard dosage of piperacillin/tazobactam is 3.375 mg intravenously every 6 hours. Contraindications include hypersensitivity to penicillins, cephalosporins, or β-lactamase inhibitors; main drug interactions are methotrexate and vecuronium, and the main side effects are pruritus, rash, diarrhea, nausea, vomiting, headache, and anaphylaxis (rare).

Anticoagulation
Anticoagulation is to be used initially, perioperatively, and potentially long term depending on AMI's etiology to reduce the chance of progressive thrombosis.

Heparin
Standard dosage of heparin is an initial bolus of 5000 to 10,000 U (or 100 U/kg), then 1000 to 1250 U/h, titrating for a goal of partial thromboplastin time of 50 to 70 seconds. Contraindications include hypersensitivity, active bleeding, history of heparin-induced thrombocytopenia, severe thrombocytopenia, and subacute bacterial endocarditis. Digoxin, nicotine, tetracycline, and antihistamines may decrease its effects, whereas NSAIDs, aspirin, dextran, dipyridamole, and hydroxychloroquine may increase heparin toxicity. Its main side effects are bleeding and thrombocytopenia, and it is low in cost and very cost effective.

Thrombolytics
Thrombolytics are angiographically infused to lyse thrombi in selected patients with AMI (MVT).

Alteplase (tissue plasminogen activator)
Standard dosage is 2 to 5 mg intra-arterially initially, followed by 1 to 2 mg/h intra-arterially. Among the contraindications are documented hypersensitivity; active internal bleeding; stroke within last 2 months; intracranial or intraspinal surgery or trauma; intracranial hemorrhage on pretreatment evaluation; suspicion of subarachnoid hemorrhage; intracranial neoplasm; arteriovenous malformation or aneurysm; bleeding diathesis; and severe, uncontrolled hypertension. Alteplase's main side effects are bleeding and stroke; its cost is high, and its cost effectiveness has not been demonstrated.

Vasodilators
Vasodilators dilate the mesenteric arterial system, reversing reactive arterial vasospasm in AMI.

Papaverine
Standard dosage is 30 to 60 mg/h intravenously via selective angiography catheter. Contraindications include hypersensitivity and complete atrioventricular block. It should be used with caution in patients with angina, recent myocardial infarction, recent stroke, and glaucoma, and it will precipitate when administered in the same intravenous line together with heparin. The main side effects are hypertension, tachyarrhythmia, hepatotoxicity, acidosis, pruritus, rash, nausea, vomiting, headache, somnolence, and priapism. Papaverine is high in cost and moderately cost effective.

Glucagon
Glucagon produces intestinal vasodilation and hypotonicity to reduce oxygen demand. Standard starting dosage is 1 μg/kg intravenously titrated up to 10 μg/kg per minute as tolerated. Contraindications are hypersensitivity to glucagon and known pheochromocytoma, and warfarin is its main drug interaction. Side effects include hyperglycemia, rash, nausea, vomiting, hypertension, and hypokalemia. It is high in cost and moderately cost effective.

Vasopressors
Vasopressors are used in the setting of systemic hypotension (vasopressin and α-agonists should be avoided).

Dopamine
Standard dosage is 2 to 5 μg/kg per minute intravenously (low dose). Contraindications include hypersensitivity, pheochromocytoma, and tachyarrhythmia, whereas alkaloids represent its main drug interaction. Dopamine's main side effects are hypertension, tachyarrhythmia, rash, nausea, vomiting, and headache. It is moderate in cost and very cost effective.

Dobutamine
In cases of decreased cardiac output, standard dosage initially is 0.5 to 1.0 μg/kg per minute intravenously. Maintenance dosage is 2.5 to 20 μg/kg per minute intravenously and should be titrated according to response (maximum dose 40 μg/kg per minute intravenously).

Contraindications for dobutamine include hypersensitivity, idiopathic hypertrophic subaortic stenosis, arrhythmias, hypovolemia, myocardial infarction, and severe coronary artery disease. Its main drug interactions are isocarboxazid and entacapone. Main side effects include chest pain, hypertension, palpitations, tachyarrhythmia, hypokalemia, nausea, headache, dyspnea, eosinophilia (rare), and thrombocytopenia (rare). It is moderate in cost and very cost effective.

Modulators of reperfusion injury
These act as free-radical scavengers that are thought to have protective effects in ischemia/reperfusion injury.

Allopurinol
Standard dosage is 200 to 400 mg/d intravenously for 24 to 48 hours. Hypersensitivity is the only contraindication, and its main drug interactions are azathioprine and enalapril (angiotensin-converting enzyme inhibitors). The main side effects are pruritus, rash, nausea, vomiting, renal failure, Stevens-Johnson syndrome (rare), agranulocytosis (rare), anemia (rare), myelosuppression (rare), and hepatotoxicity (rare). Allopurinol is low in cost, but its cost effectiveness is unclear.

Enalapril
Standard dosage is 1.25 mg intravenously every 6 hours. The only contraindications are hypersensitivity and history of angioedema. Its main drug interactions are allopurinol and aspirin. The main side effects are hyperkalemia, nausea, vomiting, and rash. It is moderate in cost with unclear cost effectiveness.

Surgical management
Stable patients should proceed urgently to angiography, with which proper diagnosis, correct delineation of vascular compromise, and potential intervention can be undertaken. Patients with peritoneal signs or signs consistent with a perforated viscus should proceed to the operating room emergently. Whenever feasible, visceral revascularization should precede bowel resection. In this way, the extent of the resection can be more accurately determined to ensure preservation of as much bowel as possible.

Embolectomy
Embolectomy is to be performed in the setting of embolic disease. Standard procedure consists of transverse arteriotomy with standard embolectomy followed by primary vessel closure.

Thromboendarterectomy
Thromboendarterectomy is to be performed in the setting of arterial thrombosis affecting the proximal mesenteric vasculature. Standard procedure consists of a longitudinal incision on the mesenteric vessel with subsequent thromboendarterectomy followed by patch angioplasty. If adequate inflow cannot be established, bypass surgery should ensue.

Mesenteric artery bypass
Grafts may originate in an antegrade fashion from the supraceliac aorta or in a retrograde fashion from the infrarenal aorta or iliac artery.

Advantages for saphenous vein bypass are that it uses autologous vein and is less prone to infection. Disadvantages are that it takes additional time to harvest.

An advantages for prosthetic graft bypass is that no procurement time is required (faster). Disadvantages are that it is prone to infection in a contaminated field plus additional cost.

Bowel resection
Bowel resection is to be performed in case nonviable bowel is found. It should preferentially be performed after revascularization procedure. In cases in which viability is questionable, it is appropriate to return to the operating room in 12 to 36 hours for a "second-look" operation (previously stapled ends of bowel may be reanastomosed at this time).

Endovascular management
Endovascular interventions may take place during the initial mesenteric angiography. In the setting of NOMI, a selective catheter may be left in the SMA, and a continuous infusion of papaverine initiated.

Angioplasty (percutaneous transluminal angioplasty)
An angiography catheter is inserted percutaneously into the femoral or brachial artery and advanced into the aorta under fluoroscopic guidance. Once the target lesion has been identified, a guidewire is advanced through the ostium of the affected splanchnic artery, and a balloon catheter is placed and subsequently used to dilate the lesion. Complications may include target vessel dissection, peripheral embolism, and stroke (if a transbrachial access is used).

Stenting
Angioplasty proceeds as described previously, then a balloon-expandable or self-expanding metallic stent is deployed across the lesion. Stent placement is associated with higher patency rates than angioplasty alone.

Conclusions
AMI is associated with a high mortality. Maintaining a high index of suspicion can lead to an early diagnosis, which lends itself to a reduced mortality. The treatment of AMI depends on the specific etiology and can range from nonoperative management to emergent laparotomy.

Disclosures
No potential conflicts of interest relevant to this article were reported.

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have been highlighted as:
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