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Understanding the Etiology and Pathophysiology of Acute Mesenteric Ischemia CME

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Understanding the Etiology and Pathophysiology of Acute Mesenteric Ischemia

An Overview of the Presentation

All types of acute mesenteric ischemia (AMI) have, to some extent, a similar presentation. The differences in clinical appearance for each type are discussed below. The most important finding is pain disproportionate to physical examination findings. Typically, pain is moderate to severe, diffuse, nonlocalized, constant, and sometimes colicky.[1,2]

Onset varies from type to type. Nausea and vomiting are found in 75% of affected patients. Abdominal distension and GI bleeding are the primary symptoms in up to 25% of patients. Pain may be unresponsive to narcotics. As the bowel becomes gangrenous, rectal bleeding and signs of sepsis (eg, tachycardia, tachypnea, hypotension, fever, and altered mental status) develop. A review of systems that is specifically looking for risk factors of AMI should be performed. This syndrome has a catastrophic outcome if not properly and rapidly treated. It should be considered in any patient with abdominal pain disproportionate to physical findings, gut emptying in the form of vomiting or diarrhea, and the presence of risk factors, especially if the patient is older than 50 years of age.[1,2]

Understanding the Anatomy

Typically, the celiac artery (CA) supplies the foregut, hepatobiliary system, and spleen; the superior mesenteric artery (SMA) supplies the midgut (ie, small intestine and proximal mid colon); and the inferior mesenteric artery (IMA) supplies the hindgut (ie, distal colon and rectum), but multiple anatomic variants have been observed. Venous drainage is through the superior mesenteric vein (SMV), which combines with the splenic vein to form the portal vein.[1,2]

AMI arises primarily from problems in the SMA circulation or its venous outflow. Collateral circulation from the CA and IMA may allow sufficient perfusion if flow in the SMA is reduced. The inferior mesenteric artery is seldom the site at which an embolus will become lodged. Only small emboli can enter this vessel because of its smaller lumen. When it does occur, the embolus lodges at the site of division of the inferior mesenteric artery into the left colic, sigmoidal, and superior hemorrhoidal arteries. In such instances, collateral flow from the middle colic and middle hemorrhoidal arteries (through the vascular arcades of the inferior

mesenteric artery distal to the embolus) may sustain the perfusion of the left colon.[1,2,7]

The Pathophysiology Behind the Different Types of AMI

Insufficient blood perfusion to the small bowel and colon may result from arterial occlusion by embolus or thrombosis (AMAE or AMAT), thrombosis of the venous system/mesenteric venous thrombosis (MVT), or nonocclusive processes such as vasospasm or low cardiac output (NOMI). Embolic phenomena account for approximately 50% of all cases, arterial thrombosis for about 25%, NOMI for roughly 20%, and MVT for less than 10%. Rarely, isolated spontaneous dissections of the SMA have been reported. Hemorrhagic infarction is the common pathologic pathway whether the occlusion is arterial or venous.[1-6]

Injury severity is inversely proportional to the mesenteric blood flow and is influenced by the number of vessels involved, systemic mean pressure, duration of ischemia, and collateral circulation. The superior mesenteric vessels are involved more frequently than the inferior mesenteric vessels, with blockage of the latter often being silent because of better collateral circulation.[1,2]

Damage to the affected bowel portion may range from reversible ischemia to transmural infarction with necrosis and perforation. The injury is complicated by reactive vasospasm in the SMA region after the initial occlusion. Arterial insufficiency causes tissue hypoxia, which leads to initial bowel wall spasm and gut emptying by vomiting or diarrhea. Mucosal sloughing may cause bleeding into the gastrointestinal tract. At this stage, little abdominal guarding is usually present, producing the classic intense visceral pain disproportionate to the physical examination findings.[1,2]

The mucosal barrier becomes disrupted as the ischemia persists, and bacteria, toxins, and vasoactive substances are released into the systemic circulation. This can cause death from septic shock, cardiac failure, or multisystem organ failure before bowel necrosis actually occurs. As hypoxic damage worsens, the bowel wall becomes edematous and cyanotic (see Figures 1 and 2). Fluid is released into the peritoneal cavity, explaining the serosanguineous fluid sometimes recovered by diagnostic peritoneal lavage. Bowel necrosis can occur within 8-12 hours from the onset of symptoms. Transmural necrosis leads to peritoneal signs and heralds a much worse prognosis.[1,2]

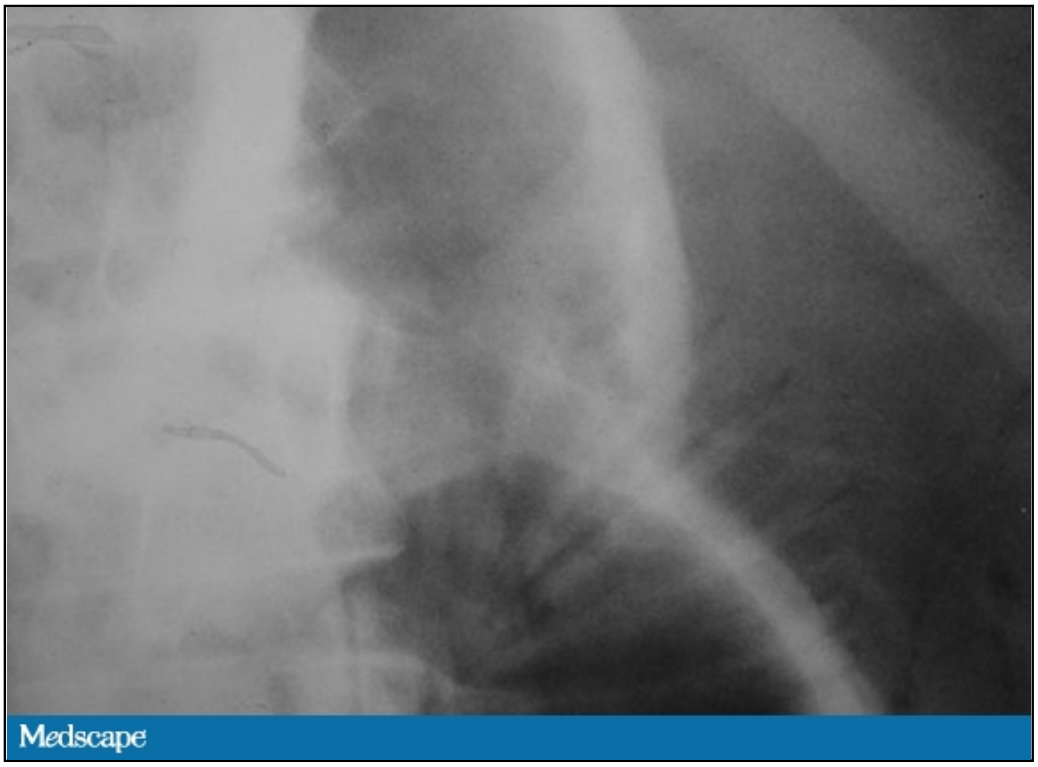

Figure 1. Pneumatosis intestinalis (black stripes of air outlining the valvulae conniventes) in advanced acute mesenteric ischemia (AMI) with gangrenous bowel.

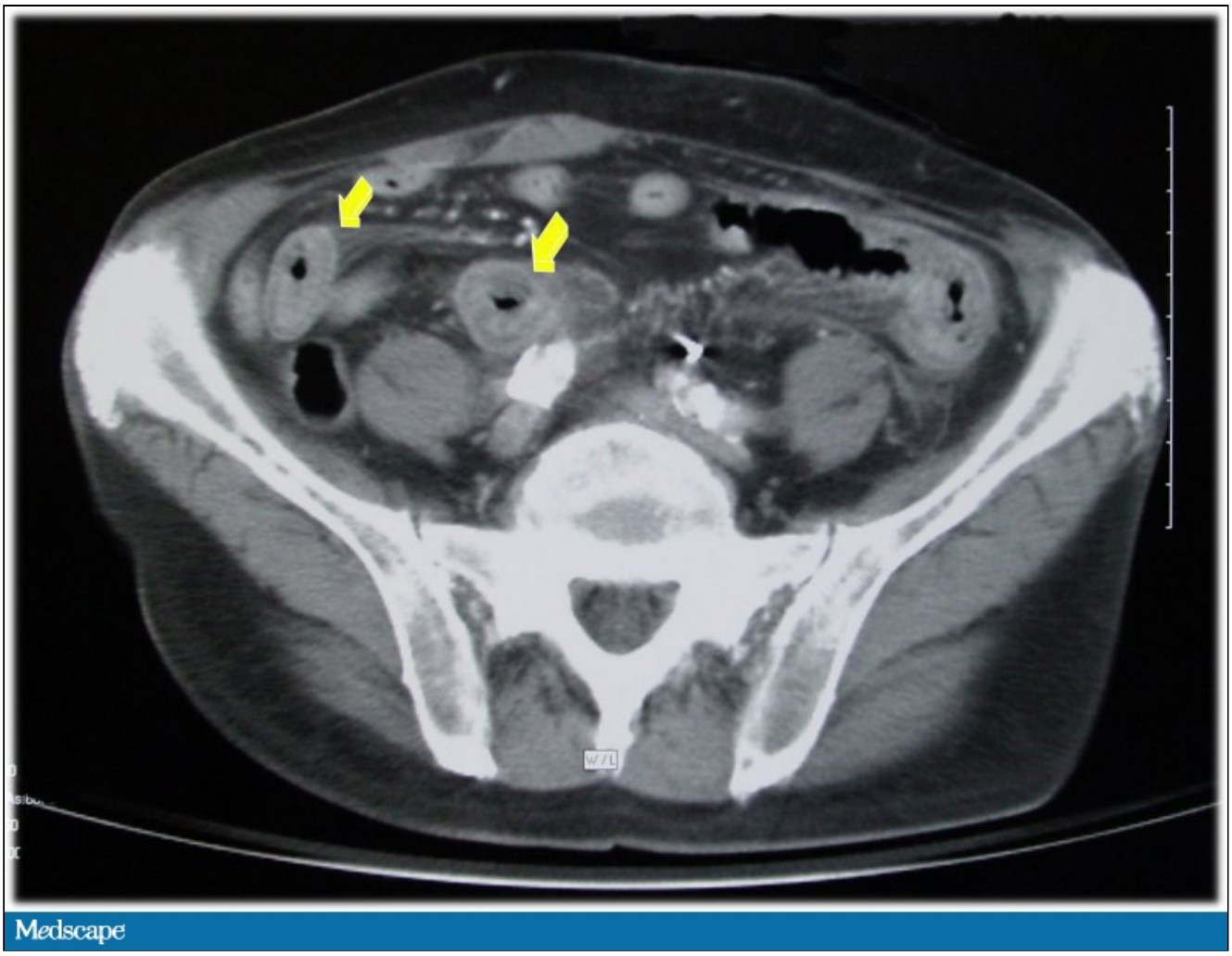

Figure 2. CT scan (with contrast) of nonocclusive mesenteric ischemia with resulting bowel edema (arrows).

Embolic AMI is usually caused by an embolus of cardiac origin. Typical causes include mural thrombi after myocardial infarction, atrial thrombi associated with mitral stenosis and atrial fibrillation, vegetative endocarditis, mycotic aneurysm, and thrombi formed at the site of atheromatous plaques within the aorta or at the sites of vascular aortic prosthetic grafts interposed between the heart and the origin of the SMA. The vascular occlusion is sudden, so the patients have not developed a compensatory increase in collateral flow. As a result, they experience worse ischemia than patients with thrombotic AMI. The SMA is the visceral vessel most susceptible to emboli because of its small branching angle from the aorta and higher flow. Most often, emboli lodge about 6-8 cm beyond the arterial origin, at a narrowing near the emergence of the middle colic artery.[1,2]

AMI from embolic causes typically has the most abrupt and painful presentation of all types. This is caused by the rapid onset of occlusion and inability to form additional collateral circulation. It has been described as abdominal apoplexy. Often, vomiting and diarrhea (gut emptying) are observed. Patients are usually found to have a source of embolization. Because most emboli are of cardiac origin, patients often have atrial fibrillation or a recent myocardial infarction (with mural thrombus). Patients may report a history of valvular heart disease or previous embolic episode; however, this is not frequently the case.[1,2]

According to the US Centers for Disease Control and Prevention Injury Center (CDC), a special form of mesenteric ischemia may result from systemic air embolism in those who sustain high-energy blast injuries. These patients sustain severe primary blast injury to the lung, a condition referred to as "blast lung".[1,2]

Thrombotic AMI is a late complication of preexisting visceral atherosclerosis. Symptoms do not develop until 2 of the 3 arteries (usually the celiac and superior mesenteric arteries) are stenosed or completely blocked. Progressive worsening of the atherosclerotic stenosis before the acute occlusion allows time for the development of additional collateral circulation.[1,2]

Most patients with thrombotic AMI have atherosclerotic disease at other sites, such as coronary artery disease, stroke, or peripheral arterial disease. A drop in cardiac output from myocardial infarction or congestive heart failure (CHF) may cause AMI in a patient with visceral atherosclerosis. Thrombotic AMI may also be a complication of arterial aneurysm or other vascular pathologies, such as dissection, trauma, and thromboangiitis obliterans. In inflammatory vascular disease, smaller vessels are affected. Thrombosis tends to occur at the origin of the SMA, causing widespread infarction. These patients frequently present with a history of chronic mesenteric ischemia in the form of intestinal angina before the emergent event.[1,2]

AMI caused by a thrombus, such as a myocardial infarction, typically happens when an artery that is already partially blocked by atherosclerosis becomes completely occluded. Similarly to angina pectoris preceding a myocardial infarction, 20%-50% of these patients have a history of abdominal angina. Abdominal angina is a syndrome of postprandial abdominal pain starting soon after eating and lasting for up to 3 hours. The digestion of food requires increased perfusion of the intestine, so the mechanism is similar to that of exercise-induced angina pectoris. Weight loss, "food fear," early satiety, and altered bowel habits may be present.[1,2]

The precipitating event that initiates thrombotic AMI may be a sudden drop in cardiac output from myocardial infarction, CHF, or a ruptured plaque. Dehydration from vomiting or diarrhea caused by an unrelated illness may also precipitate thrombotic AMI. These patients have undergone a gradual progression of arterial occlusion and frequently have a better collateral supply. Bowel viability is better preserved, often leading to a less severe presentation than with embolic AMI. Symptoms tend to be less intense and of more gradual onset. As might be expected, these patients typically have a history of atherosclerotic disease at other sites, such as coronary artery disease, cerebral arterial disease, peripheral artery disease (especially aortoiliac occlusive disease), or a history of aortic reconstruction.[1,2]

NOMI is precipitated by a severe reduction in mesenteric perfusion, with secondary arterial spasm from such causes as cardiac failure, septic shock, hypovolemia, or the use of potent vasopressors in patients in critical condition. Because bowel perfusion, similar to cerebral perfusion, is preserved in the setting of hypotension, NOMI represents a failure of autoregulation. Many vasoactive drugs may also cause regional vasoconstriction, such as digitalis, cocaine, diuretics, and vasopressin. Gross pathologic arterial or venous occlusions are not observed in patients with NOMI.[1,2,7]

Nonocclusive AMI occurs more frequently in older patients than other forms of AMI. These elderly patients are often already in an ICU setting with acute respiratory distress syndrome or severe hypotension from cardiogenic or septic shock, or they are receiving vasopressive drugs. Most of these patients are taking digitalis. Symptoms typically develop over several days, and patients may have had a prodrome of malaise and vague abdominal discomfort. When infarction occurs, the patients develop increased pain associated with vomiting. They may become hypotensive and tachycardic, with loose, bloody stool.[1,2,7]

MVT often (ie, >80% of the time) is the result of some processes that make the patient more likely to form a clot in the mesenteric circulation (ie, secondary MVT). Primary MVT occurs in the absence of any identifiable predisposing factor. The list of causes for MVT is long and includes infection, usually from an intra-abdominal source; phlebitis or pylephlebitis (portal pyemia) secondary to inflammatory diseases of the bowel, such as diverticulitis, appendicitis, and secondarily infected carcinoma of the bowel; hypercoagulable states, such as those caused by polycythemia, oral contraceptives, or genetic abnormalities (protein C or S deficiency); mesenteric venous stasis from portal hypertension or mass effect of abdominal tumors; and direct trauma to the mesenteric veins from a surgical procedure.[1,2]

MVT may also occur after ligation of the portal vein or the superior mesenteric vein as part of "damage-control surgery" for severe penetrating abdominal injuries. Other associated causes include pancreatitis, sickle cell disease, and hypercoagulability caused by malignancy.[1,2]

MVT is often observed in a much younger patient population than other types of AMI. Symptoms may be present longer than in the typical cases of AMI, sometimes exceeding 30 days. Infarction from MVT is rarely observed with isolated SMV thrombosis, unless collateral flow in the peripheral arcades or vasa recta is compromised as well. Fluid sequestration and bowel wall edema are more pronounced than in arterial occlusion. The colon is usually spared because of better collateral circulation. The chronic form of SMV thrombosis may manifest as esophageal varices bleeding. MVT patients can present with an acute or subacute abdominal pain syndrome related to involvement of the small intestine rather than the colon. The symptoms are frequently less dramatic. Diagnosis can be even more difficult, because symptoms may have been present for weeks (ie, 27% have symptoms for >30 d). Typical symptoms of AMI may have been experienced for a prolonged period, with gradual worsening. The chronic form may manifest as esophageal varices bleeding.[1,2]

Many patients have a history of one or more of the risk factors for hypercoagulability. These include oral contraceptive use,

congenital hypercoagulable states, deep vein thrombosis (DVT), liver disease, tumor, or portocaval surgery.[1,2]

Characteristics of the Disease

The overall prevalence of AMI in the United States is 0.1% of all hospital admissions; this may be expected to rise as the population ages. The exact prevalence of MVT is unknown because many cases are presumed to be limited in symptomatology and to resolve spontaneously. In 1989, the incidence of diagnosed MVT was reported to be 2 per 100,000 admissions over 20 years at the Albert Einstein College of Medicine Montefiore Medical Center.[1,2]

Rates of AMI have not been demonstrated to be significantly different outside of the United States; however, because it is primarily a disease of older individuals, rates are probably lower in countries whose populations have a shorter life expectancy.[1,2]

Overall, the mortality rate in the last 15 years from all causes of AMI averages 71%, with a range of 59%-93%. Once bowel wall infarction has occurred, the mortality rate is as high as 90%. Survivors of mesenteric resection face significant long-term morbidity because of the reduced intestinal mucosal surface available for absorption. In a report from Madrid of 21 patients with SMA embolus with little delay in initiating maximal treatment, intestinal viability was achieved in 100% of patients if the duration of symptoms was shorter than 12 hours, 56% if it was 12-24 hours, and only 18% if it was longer than 24 hours. Early recognition and treatment of NOMI has been shown to reduce the mortality rate to 50%-55%. MVT has a 30-day mortality rate of 13%-15%.[1,2]

No racial predilections are known for AMI; however, people of races with a higher rate of conditions leading to atherosclerosis, such as black people, might be at a higher risk. No overall sex preference exists for AMI. Men might be at a higher risk for occlusive arterial disease because they have a higher incidence of atherosclerosis. Conversely, women who are on oral contraceptives or are pregnant are at a higher risk for MVT. AMI is frequently considered a disease of people older than 50 years. Younger people with atrial fibrillation or risk factors for MVT, such as oral contraceptive use or hypercoagulable states (eg, those caused by protein C or S deficiency), may present with AMI.[1,2]

Return to the Case of the Week, "A 76-Year-Old Woman With Abdominal Pain."

CME Test

A patient suspected of having mesenteric ischemia presents with the rapid onset of severe pain accompanied by nausea and vomiting. Of the etiologies discussed above, which of the following is the most likely?

- Embolic acute mesenteric ischemia
- Thrombotic acute mesenteric ischemia
- Nonocclusive mesenteric ischemia
- Mesenteric venous thrombosis

In an embolic occlusion, which of the following diagnostic studies may be useful in determining the underlying cause of the embolism?

- Electrocardiogram
- Abdominal CT scan
- Chest radiograph
- Angiography

Save and Proceed

Return to the Case of the Week, "A 76-Year-Old Woman With Abdominal Pain."

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