Management of gut ischemia

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Ischemia of the intestine may manifest with different clinical features, each one requiring a specific diagnosis and treatment approach. Early identification and treatment of acute mesenteric ischemia is crucial to improve prognosis. For that purpose, early angiography followed by surgery or a direct laparotomy in critically ill patients is warranted. Embolectomy and bowel resection for arterial embolus, thrombectomy and revascularization for arterial thrombus, vasodilators for nonocclusive mesenteric ischemia, and heparin and eventual bowel resection for acute venous thrombosis, are the treatment modalities of choice. Chronic mesenteric ischemia has to be considered in elderly patients or those with risk factors presenting with postprandial abdominal pain and weight loss. Treatment may be performed either by surgical revascularization or angioplasty with or without stent placement according to patient conditions and surgical risk. Ischemic colitis is the most common form of intestinal ischemia, usually affects the left colon, and may resolve spontaneously. Bowel rest, IV fluids, and antibiotics is the standard treatment. Initially, surgery is indicated for patients with peritonitis, massive bleeding, or fulminant colitis. Subsequently, surgery is also indicated for patients who do not improve after 2-3 weeks of treatment, those evolving to sepsis, or those presenting late with colonic stricture or persistent chronic colitis. [Indian J Gastroenterol 2006;25(Suppl 1):S39-S42]

Under the term intestinal ischemia a number of clinical disorders are included in which blood supply to the intestine and mesentery is impaired, thus limiting tissue oxygenation and nutrient availability. This can be a consequence of reduced splanchnic or mesenteric blood flow in the overall territory or in a regional bed.

The spectrum of intestinal ischemia comprises a number of syndromes that include acute mesenteric ischemia (AMI) as a result of emboli, arterial or venous thrombi, vasoconstriction secondary to low-flow states, or small bowel loop strangulation; chronic mesenteric ischemia (CMI) (also called intestinal angina) due to transient and recurrent episodes of inadequate intestinal blood flow to sustain metabolic needs or to support increased metabolic demands such as that associated with digestion; and ischemic colitis, where circulatory insufficiency of the colon results in varying degrees of local tissue necrosis and systemic manifestations.

Ischemic colitis is the most frequent ischemic event and accounts for 70%-75% of all cases of intestinal ischemia. AMI is the second most common (25%-30%), and includes embolization to the superior mesenteric artery (SMA) (50% of all cases of AMI), nonocclusive mesenteric ischemia (NOMI) (20% to 30% of episodes of AMI), arterial thrombosis (about 15% of cases of AMI), and acute venous mesenteric thrombosis (5%-10% of AMI). Finally, CMI represents only 5% of all ischemic episodes.

Acute mesenteric ischemia

Several risk factors may contribute to each specific ischemic event. For intestinal arterial embolism these include cardiac arrhythmia, myocardial dyskinesia, prosthetic valve, cardioversion, cardiac catheterization, recent myocardial infarction, and prior emboli. For intestinal arterial thrombosis the main factors are: prior cerebral, coronary or peripheral arterial insufficiency, old age, low-flow state, diabetes, hypercholesterolemia, hypertension, aortic mesenteric aneurysm, congestive heart failure, hypercoagulable state, vasculitides, and trauma. For NOMI, the factors are cardiogenic or hypovolemic shock, congestive heart failure, pulmonary edema, aortic insufficiency, major cardiac or abdominal surgery, dialysis, and the use of vasoconstrictor drugs. Concerning acute mesenteric venous thrombosis, hypercoagulable states, hyperviscosity syndromes, infection or inflammation, trauma, and portal hypertension are the most common precipitating factors.

Early diagnosis of the mesenteric ischemic event is crucial to enhance prognosis. Thus, if diagnosis of AMI is made within the first 24 hours after the symptoms have begun, the mortality ranges from 40%-50%, whereas the mortality increases up to 80% if the diagnosis is delayed more than 24 hours. Early diagnosis is hindered by the fact that the symptoms may be nonspecific. The main symptom for AMI is acute abdominal pain out of proportion to physical findings in a patient with one of the above mentioned predisposing factors. A history of chronic
postprandial abdominal pain is present when acute arterial thrombosis is superimposed on chronic mesenteric ischemia, but abdominal pain may not be appreciated in confused or critically ill patients. If the clinical picture of occlusive intestinal ischemia may often be blurred and misleading, this is even more true for NOMI. Patients will often already be in intensive care for other reasons, quite often the clinical presentation is dominated by one of the underlying diseases, and abdominal pain may be absent in 25% of the cases.

In a patient with suspicion of acute mesenteric ischemia, prompt diagnosis and therapeutic approach after resuscitation is the single most important factor to improve prognosis. Prognosis also varies according to the cause of ischemia, and to ascertain the possible etiology of the ischemic event it is necessary to evaluate first the possible history of deep venous thrombosis or hypercoagulatable state. If it is the case, the possibility of acute venous thrombosis is high and CT dynamic scan or magnetic angoressionance is warranted to confirm such diagnosis. If not, the existence of positive peritoneal findings has to be evaluated. In case of signs of peritonitis, urgent laparotomy is necessary, but if those signs are not present, abdominal angiography is the preferred diagnostic tool to distinguish between emboli, thrombosis and nonocclusive ischemia. In a review of mesenteric angiography in the diagnosis of AMI, sensitivities in five of six studies have ranged between 90%-100%; specificity was reported in two of these studies to be 100%.

Various therapeutic approaches have been proposed for embolus, including surgical revascularization, intra-arterial perfusion of thrombolytic agents or vasodilators, and systemic anticoagulation. The therapeutic option should be chosen taking into account the presence or absence of peritoneal signs, whether arterial occlusion is partial or complete, and whether location of the embolus is above the origin of the ileocolic artery or in more distal branches. There is uniform agreement that exploratory laparotomy is mandatory when signs of peritonitis are present and that embolectomy and resection of any infarcted bowel should be performed as necessary. When abdominal contamination is absent, primary anastomosis is generally performed. When extensive bowel segments are questionably viable, only unambiguously necrotic bowel is resected, and a second-look operation is performed about 24 hours later to permit demarcation between viable and nonviable bowel and to minimize the extent of bowel resection.

Anticoagulation with heparin is administered postoperatively to prevent recurrent embolization. Successful nonoperative treatment of acute mesenteric arteriopathy using heparin and thrombolytic agents including urokinase, streptokinase, and recombinant tissue plasminogen activator is likely to be successful when the embolus is partially occluding, or is in one of the branches of the SMA or in the main SMA distal to the origin of the ileocolic artery, and the treatment is applied within 12 hours of the onset of symptoms. There is some controversy as to the use of vasodilators as a complementary treatment in patients with SMA embolus. There is evidence showing that vasocostriction of both the unobstructed and obstructed branches occurs with a SMA embolus. Successful nonoperative treatment of acute mesenteric ischemia, prompt diagnosis and therapeutic approach after resuscitation is the single most important factor to improve prognosis. Prognosis also varies according to the cause of ischemia, and to ascertain the possible etiology of the ischemic event it is necessary to evaluate first the possible history of deep venous thrombosis or hypercoagulatable state. If it is the case, the possibility of acute venous thrombosis is high and CT dynamic scan or magnetic angoressionance is warranted to confirm such diagnosis. If not, the existence of positive peritoneal findings has to be evaluated. In case of signs of peritonitis, urgent laparotomy is necessary, but if those signs are not present, abdominal angiography is the preferred diagnostic tool to distinguish between emboli, thrombosis and nonocclusive ischemia. In a review of mesenteric angiography in the diagnosis of AMI, sensitivities in five of six studies have ranged between 90%-100%; specificity was reported in two of these studies to be 100%.

If a diagnosis of acute thrombosis of the SMA is made, emergency surgical revascularization is recommended. Simple surgical thrombectomy is unlikely to be successful in the long term, and it has to be accompanied by revascularization performed via bypass.

Management of NOMI is essentially pharmacological and is readily achieved by selective infusion of vasodilators into the SMA. Splanchnic vasodilators include papaverine, tolazoline, nitroglycerin, glucagon, prostaglandin E, phenoxybenzamine, and isoproterenol. The greatest clinical experience is with papaverine, which is administered as a continuous infusion into the SMA at 30 to 60 mg/h. Immediate laparotomy is indicated during papaverine therapy if peritoneal signs do not rapidly remit with onset of infusion, if peritoneal signs develop during infusion, or if the patient deteriorates clinically.

In asymptomatic individuals in whom the diagnosis of acute venous thrombosis has been made on a CT scan obtained for reasons other than abdominal pain, either no therapy or a 3-6 month course of anticoagulation is reasonable.

In symptomatic patients in whom an acute thrombosis of the superior mesenteric vein (SMV) is diagnosed either by CT scan, MRI, or angiography, treatment is determined by the presence or absence of peritoneal signs. As in all patients with AMI, signs of peritonitis mandate laparotomy and resection of infarcted intestine. If the diagnosis of SMV thrombosis is established intra-operatively, anticoagulation should start immedi-
ately. Intravenous anticoagulation with heparin has been shown to prevent thrombus propagation and recurrence, and to improve survival. In the absence of peritoneal signs, heparin should be immediately initiated, with an initial bolus of 5000 units followed by continuous infusion at 1000 units/h.

**Chronic mesenteric ischemia**

Chronic mesenteric ischemia (intestinal angina) is a clinical syndrome characterized by recurrent abdominal pain and weight loss as a result of repeated transient episodes of insufficient intestinal blood flow, usually related with the increased metabolic demand associated with digestion. It is seen in middle-aged or elderly people, predominantly in females, and with direct or indirect evidence of atherosclerosis.

In most cases, patients undergo extensive workup for obscure chronic abdominal pain, including routine laboratory evaluation, plain abdominal radiographs, upper and lower GI endoscopy, small bowel barium series, and ultrasonography or abdominal CT. When common causes of abdominal pain have been ruled out and suspicion of intestinal ischemia is well established from epigastric pain after meals and substantial weight loss, an angiography, CT angiographic study, or angioresonance of the splanchnic arteries is warranted, especially in patients with risk factors.

Complete revascularization, or alternatively bypass grafting of the SMA alone, is the treatment of choice for patients with low surgical risk. In those patients with high surgical risk the treatment should be performed by angioplasty with or without stent placement.

**Ischemic colitis**

Ischemic colitis, which is predominantly seen in the left colon, may occur from either occlusive or nonocclusive events, mainly in the territory of the inferior mesenteric artery (IMA), in colonic branches of the SMA, or in the superior and inferior mesenteric veins. Large arterial vessel occlusion may be caused by thrombi or atheromatous lesions. Aortic surgery can cause ischemic colitis because of unnoticed ligation of the IMA, the occurrence of intraoperative hypoperfusion in the presence of IMA occlusion, or embolization of atheromatous debris to the hypogastric arteries tissue beds during endovascular manipulations.

Abdominal pain and rectal bleeding are the most common symptoms of ischemic colitis, but the signs and symptoms of ischemic colitis overlap with other colonic diseases such as infectious colitis, inflammatory bowel disease, colon cancer, radiation colitis, diverticulitis, pseudomembranous colitis, NSAID-induced colonic lesions, or pancreatitis. Data supporting the diagnosis of ischemic colitis are age of onset, segmental distribution of injury, abrupt transition between damage and undamaged mucosa, rectal preservation (only useful for distinction from ulcerative colitis), and rapid resolution of lesions in subsequent colonoscopies.

Most cases of ischemic colitis resolve spontaneously. However, bowel rest and general supportive measures including parenteral fluids are recommended. Precipitating factors should be corrected. This includes stopping any medication with potential vasoconstrictor effects on the mesenteric vasculature or narcotics that may worsen colonic distension. Measures should be implemented to correct low-flow states due to either hypovolemia or cardiac dysfunction. Intravenous infusion of broad-spectrum antibiotics is usually recommended, although no definitive clinical evidence is available of the beneficial effects of such policy. Patients showing evident signs of peritonitis require urgent surgery and bowel resection. In patients in whom severe symptoms such as abdominal tenderness or fever, or laboratory findings such as leukocytosis or metabolic acidosis persist or deteriorate despite medical treatment, surgery is also warranted. Other infrequent indications for surgery are massive bleeding, persistent ischemic lesions with protein-losing colopathy, and recurrent sepsis in a patient apparently recovered from an acute episode of ischemic colitis. In case of chronic evolution of ischemic colitis, surgery is required only when colonic stricture becomes symptomatic. Segmental resection is the procedure of choice in such cases, although transendoscopic dilatation may be an alternative in selected patients at high risk for surgery.

**References**


