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MINIREVIEWS

Mesenteric ischemia: Pathogenesis and challenging diagnostic and therapeutic modalities

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Abstract

Mesenteric ischemia (MI) is an uncommon medical condition with high mortality rates. MI includes inadequate blood supply, inflammatory injury and eventually necrosis of the bowel wall. The disease can be divided into acute and chronic MI (CMI), with the first being subdivided into four categories. Therefore, acute MI (AMI) can occur as a result of arterial embolism, arterial thrombosis, mesenteric venous thrombosis and non-occlusive causes. Bowel damage is in proportion to the mesenteric blood flow decrease and may vary from minimum lesions, due to reversible ischemia, to transmural injury, with subsequent necrosis and perforation. CMI is associated to diffuse atherosclerotic disease in more than 95% of cases, with all major mesenteric arteries presenting stenosis or occlusion. Because of a lack of specific signs or due to its sometime quiet presentation, this condition is frequently diagnosed only at an advanced stage. Computed tomography (CT) imaging and CT angiography contribute to differential diagnosis and management of AMI. Angiography is also the criterion standard for CMI, with mesenteric duplex ultrasonography and magnetic resonance angiography also being of great importance. Therapeutic approach of MI includes both medical and surgical treatment. Surgical procedures include restoration of the blood flow with arteriotomy, endarterectomy or anterograde bypass, while resection of necrotic bowel is always implemented. The aim of this review was to evaluate the results of surgical treatment for MI and to present the recent literature in order to provide an update on the current concepts of surgical management of the disease. Mesh words selected include MI, diagnostic approach and therapeutic management.

Key words: Acute mesenteric ischemia; Mesenteric ischemia; Chronic diagnostic approach; Therapeutic management; Surgical strategy

Core tip: Mesenteric ischemia (MI) is an uncommon medical condition with high mortality rates. MI includes inadequate blood supply, inflammatory injury and eventually necrosis of the bowel wall. Because of a lack of specific signs or due to its sometime quiet presentation, this condition is frequently diagnosed only at an advanced stage. Therapeutic approach refers to both medical and surgical treatment. Surgical procedures include restoration of the blood flow with arteriotomy, endarterectomy or anterograde bypass, while resection of necrotic bowel is always implemented.

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INTRODUCTION

Mesenteric ischemia (MI) is an uncommon medical condition that accounts 0.1% of all hospital admissions, with high mortality rates ranging from 24%-94%[1]. MI includes inadequate blood supply, inflammatory injury and eventually necrosis of the bowel wall. The disease can be divided into acute and chronic MI (CMI), with the first being subdivided into four categories[2]. Therefore, acute MI (AMI) can occur as a result of arterial embolism, arterial thrombosis, mesenteric venous thrombosis (MVT) and non-occlusive causes (NOMI), such as hypo-perfusion due to low cardiac output or mesenteric arterial vasoconstriction[3]. Bowel damage is in proportion to the mesenteric blood flow decrease and may vary from minimum lesions, due to reversible ischemia, to transmural injury, with subsequent necrosis and perforation[4]. CMI is associated to diffuse atherosclerotic disease in more than 95% of cases, with all major mesenteric arteries presenting stenosis or occlusion. Other causes include fibromuscular dysplasia, vasculitis, Takayasu arteritis, malignancy and radiation. Because of a lack of specific signs or due to its sometime quiet presentation, this condition is frequently diagnosed only at an advanced stage[5]. Constant, diffuse, non-localized or periumbilical abdominal pain remains the most common symptom[6-8]. Postprandial pain, nausea and weight loss occur in patients with CMI[4]. While laboratory studies or plain films of abdomen are not indicative, computed tomography (CT) imaging and CT angiography contribute to differential diagnosis and management of AMI. Angiography is also the criterion standard for CMI, with mesenteric duplex ultrasonography (US) and magnetic resonance angiography (MRA) also being of great importance[9-11]. Therapeutic approach of MI includes both medical and surgical treatment. Papaverine, heparin, warfarin and thrombolytic drugs are the most common medications[12]. Surgical procedures include restoration of the blood flow with arteriotomy, endarterectomy or anterograde bypass, while resection of necrotic bowel is always implemented[13]. The aim of this review was to evaluate the results of surgical treatment for MI and to present the recent literature in order to provide an update on the current concepts of surgical management of the disease. Mesh words selected include MI, diagnostic approach and therapeutic management.

HISTOLOGY AND PATHOGENESIS

In the process of embryogenesis, segmental arteries regress until three major vessels remain: The celiac artery (CA), the superior mesenteric artery (SMA) and the inferior mesenteric artery (IMA) as well. Typically, the CA supplies the foregut, hepatobiliary system and spleen, the SMA supports the midgut including small intestine and proximal mid colon up to the splenic flexure and the IMA refers to the hind gut with regard to distal colon beginning from the splenic flexure, and rectum[14]. The venous system, mainly, parallels the arterial branches. Therefore, the superior mesenteric vein drains the small intestine and cecum, while the inferior mesenteric vein includes the descending and sigmoid colon and the rectum[15]. The gastrointestinal (GI) system presents significant collateral circulation at all levels that provides protection from ischemia and can compensate for approximately a 75% acute reduction in mesenteric blood flow for up to 12 h, without substantial injury[16].

AMI caused by arterial embolism accounts for 50% of acute ischemic conditions. The SMA is the visceral vessel the most susceptible to emboli due to its small take-off angle from the aorta and higher flow. Most often, emboli lodge about 6-8 cm beyond the arterial origin, distal to the origin of the middle colic artery[6,17]. Typical causes of emboli include mural thrombi after myocardial infarction, atrial thrombi associated with mitral stenosis, atrial fibrillation, septic emboli from valvular 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 anywhere between the heart and the origin of SMA[8,17].

The thrombosis of mesenteric arteries typically occurs at their origin causing extensive infarction and often affects at least two of the major visceral vessels. It is mainly a complication of preexisting visceral atherosclerotic lesions and involves acute worsening of the already compromised blood flow. It may also be attributed to arterial aneurysm or other vascular pathologies, such as dissection, trauma, mesenteric aneurysm rupture, fibromuscular dysplasia or vasculitis[18,19]. In inflammatory vascular disease, smaller vessels are commonly affected. On the other hand, NOMI results from hypoperfusion with secondary severe and prolonged visceral vasoconstriction. The most common initiating conditions involve systemic shock

due to decreased cardiac output following a myocardial infarction or congestive heart failure, septic shock or hypovolemia. Additionally, NOMI can be the result of compression by intra-abdominal tumors[6]. A secondary clinical entity of MI occurs due to mechanical obstruction, such as internal hernia with strangulation and volvulus.

Finally, MVT includes primary disorder with no identifiable predisposing factor and secondary MVT as well. The most common cause of secondary MVT is hypercoagulability. Additionally, in more than 95% of patients, diffuse atherosclerotic disease decreases the blood flow to the bowel and causes CMI. Other causes refer to fibromuscular dysplasia, vasculitis including Takayasu arteritis, giant cell arteritis, polyarteritis nodosa, systemic lupus erythematosus, thromboangiitis obliterans, malignancy and radiation. Subsequent complications conclude to thrombotic and embolic phenomena leading to manifestations of AMI[15,20,21].

Current pathophysiologic understanding of visceral perfusion suggests a pivotal role for the visceral circulation in the cardiovascular homeostasis regulation. GI perfusion is usually impaired early in situations including critical illness, major surgery and exercise, all of which are characterized by increased demands on the circulation to maintain tissue oxygen delivery[16,18,22,23]. This relative hypoperfusion often outlasts the period of the hypovolemic insult or low-flow state. Tissue damage results either from ischemic or reperfusion injury. Within 3-4 h after the onset of ischemia, there is necrosis of the mucosal villi and within 6 h, transmural, mural or mucosal infarction can be observed. The splenic flexure is more susceptible to ischemic injury as it is the separation point of distribution between the SMA and the IMA. In the early stages, the intestinal wall appears congested and later it becomes edematous, friable and hemorrhagic. Without treatment, patients present with bowel hemorrhage within 1-4 d while enteric bacteria can cause gangrene, which leads to perforation and sometimes severe sepsis[24,25].

CLINICAL PRESENTATION

With regard to clinical presentation, acute and chronic MI differs significantly dependent on the underlying etiology[26]. AMI's most typical symptom is abdominal pain that is disproportionate to physical examination findings. As ischemia is the pathologic process, the pain initially is visceral, diffused, non-localized and it may be moderate to severe, constant, sometimes colicky and occasionally unresponsive to opioid analgesics. Other common signs are nausea and vomiting while diarrhea progressing to constipation may be also present. Examination findings early in the course of the disease are limited and non-specific, including minimal abdominal tenderness. If ischemia is attributed to embolic disease, the pain is severe and abrupt, as occlusion is rapidly installed and collateral circulation is completely absent[13,22]. On the other hand, AMI due to arterial thrombosis has a significantly more gradual progression of ischemia and infarction and less severe presentation, because the artery is usually already partially blocked and a collateral supply has been established[18]. Once the ischemia has progressed transmurally, signs of peritonitis and septicemia are encountered[27]. Bowel necrosis, septic shock and death are common complications in AMI. On the contrary, CMI typically causes postprandial abdominal pain, generally epigastric or periumbilical, nausea and weight loss. Examination findings include signs of malnutrition, pain disproportionate to clinical features and abdominal bruit[5,28].

DIAGNOSTIC MODALITIES

Due to the unclear manifestation of AMI, this condition is often misdiagnosed causing serious morbidity and high mortality. Laboratory studies are non-specific. Initially, complete blood cell count may be normal but leukocytosis and/or leftward shift may be observed later in the clinical course. The levels of amylase and lactate dehydrogenase may be increased[29]. Metabolic acidosis is a common but nonspecific disorder. In addition, prothrombin time and activated partial thromboplastine time should be evaluated and especially when MVT occurs, patients should be examined for protein C and S and antithrombin Ⅲ deficiencies, abnormalities in lupus anticoagulant, anticardiolipin antibody and platelet aggregation[16,17,30].

In the absence of physical signs of peritonitis imaging studies are implemented. The majority of patients firstly undergo CT scanning in order to exclude other causes with similar clinical features and it presents a sensitivity of 71%-96% and a specificity of 92%-94% for AMI. CT findings include intramural (pneumatosis intestinalis) as well as portal vein gas, focal edematous bowel wall, mesenteric edema, abnormal gas patterns, streaking of mesentery and solid organ infarction. Arterial occlusion may present lack of enhancement of the vessels, but unlike embolic infarction, thrombosis of the SMA occurs more commonly in the origin of the vessel[31,32]. In MVT, CT scans may demonstrate an enlarged mesenteric or portal vein with sharp definition of the venous wall and low density within the vessel. Magnetic resonance imaging (MRI) along with MRA yields similar findings to those of CT scanning in AMI[15,21].

Angiography has been traditionally the most reliable method to assess the presence and the extent of occlusive disease. Anteroposterior views demonstrate collateral pathways and lateral projections depict the origins of visceral branches. Patients with thrombosis demonstrate complete lack of the visualization of the SMA and its branches, whereas those with embolism of the SMA present filling of the proximal SMA only with a sharp cutoff of the artery. Angiography is now superseded by CT angiography, as it is noninvasive and easily available[33]. However, if CT scanning is inconclusive and there is strong clinical suspicion of AMI, angiography is performed to verify the diagnosis. In addition, as

CT findings of NOMI are nonspecific, angiography reveals diffuse stenosis of the mesenteric vessels in the absence of occlusive lesions and reduced delineation of intestinal parenchyma[34]. Duplex US is highly specific (92%-100%) but not as sensitive as angiography (70%-89%) as the examination cannot detect clots beyond the proximal main vessels.

In CMI laboratory evaluation may confirm anemia, leucopenia or lymphopenia secondary to chronic malnourishment, as well as, hypoalbuminemia and electrolyte abnormalities[28]. As regards imaging studies, arteriography remains the criterion standard. Occlusion of two visceral branches of the aorta with severe stenosis of the remaining visceral branch, usually the celiac or SMA is observed. Multi-sliced CT is a noninvasive technique, which can play a pivotal role in diagnosing vascular disease of the celiac trunk and SMA in chronic ischemia. In addition, MRI and MRA are very promising diagnostic modalities when combined with fast contrast-enhanced (CE) techniques[5,35]. While AMI is an emergency where CT scanning is the most appropriate diagnostic tool, CMI is best examined with CE-MRA, coupled with measurements of flow. With this functional approach, MRA is the only modality that can completely assess abdominal vascular disease. Mesenteric duplex (US) is helpful to assess vascular patency, following visceral bypass grafting or endovascular stenting when combined with angiography to estimate potential existence of severe stenosis. However, the sensitivity is limited owing to intraperitoneal gas, obesity and previous abdominal surgical interventions[36,37].

THERAPEUTIC APPROACH

AMI high mortality rates indicate the importance of urgent medical treatment. Maintenance of hemodynamic stability and adequate oxygen saturation as well as correction of any electrolyte imbalance is of outmost importance. Vasopressors should be avoided. Blood products can be provided and 2-4 units of red blood cells should be available. Administration of broad spectrum antibiotics starts early to prevent and treat sepsis and should cover the colonic flora**.** Nasogastric tube decompression, correction of any acid/base abnormalities, bladder catheterization and intravenous fluid administration are always implemented preoperatively. Pain control is mainly accomplished with opioids[8,12,30].

Different drug treatment protocols have been suggested according to the subtypes of AMI. Targeted infusion of papaverine through angiography catheter at the affected vessels relieves the reactive vasospasm and therefore is proposed for all arterial forms of AMI as well as NOMI. Subsequent continuous administration of 30-60 mg/h papaverine after angiography with appropriate dose adjustment in accordance with clinical response for at least 24 h is warranted. Caution should be taken though as significant hypotension may occur if the catheter invades aorta. Relevant therapeutic intervention remains the treatment modality of choice for NOMI[12,17,30]. Finally, VEGF has been recently thoroughly proposed as a future potential therapeutic approach to MI[37].

In case of embolic AMI, infusion of thrombolytics within 8 h of symptoms onset is recommended for selected patients. As the main complication is GI bleeding, absolute contraindications for intense thrombolysis are the presence of peritonitis signs or bowel necrosis. Recent investigations suggest the tenecteplase or reteplase infusion angiographically to lyse thrombi instead of alteplase, as they appear to cause less non-intracranial bleeding[38-40]. Moreover, Ⅳ administration of anticoagulants to prevent further extension of thrombus in MVT or post-revascularization in arterial occlusive AMI has been advocated[41]. Heparin inhibits further thrombogenesis and prevents additional clot accumulation with particular caution to the possibility of GI bleeding. Conversion to oral warfarin with suitable dose adjustment is always indicated and should be continued for at least 6 mo[21,42].

Recent knowledge has generated a multidisciplinary surgical management of AMI. Initial aim is to restore intestinal oxygenation and to minimize or prevent severe complications such as peritonitis and gangrenous bowel perforation. Thus, surgical treatment of AMI with signs of peritonitis mainly includes exploratory laparotomy with meticulous assessment of bowel viability[6,30]. Resection of infarcted intestine is strongly indicated. In addition, intraoperative Doppler US and Ⅳ infusion of fluorescein and bowel examination under Wood lamp illumination can differentiate poorly perfused bowel. Resection of necrotic bowel plays a pivotal role for patient resuscitation while an attempt for anastomosis remains controversial. A second look operation is strongly suggested along with clinical examination and diagnostic imaging. Intense screening of intestinal revascularization is always implemented[26,43].

More specific, in case of embolic AMI an attempt of reperfusion remains of vital importance[6]. Surgical team can determine the location of the blockage by palpation and proceed to transverse arteriotomy proximal to the occlusion, using a balloon-tipped Fogarty catheter in order to extract the clot. The embolectomy can be performed most expeditiously by exposing the SMA at the base of the transverse mesocolon. The arteriotomy can be sealed primarily or vein-patched. Alternative approach refers to bypass technique with venous or arterial graft[17,30,44]. In case of thrombotic occlusion with absence of gangrenous bowel, revascularization is attempted either with antegrade or retrograde aorto-mesenteric bypass or with trans-aortic endarterectomy. In case of spontaneous dissection of the SMA before the onset of intestinal infarction successful percutaneous stent placement has been reported[39,45,46].

Significant incidence of AMI has also been referred after aortic aneurysm repair. Prevalence of clinically evident bowel ischemia after endovascular abdominal aortic reconstruction is comparable to open surgical

approach. However, small bowel ischemia higher prevalence in patients with endovascular repair has been detected and is associated with extremely high mortality. The direct pathologic evidence and the patterns of segmental, skipped, or patchy ischemia in most patients imply that micro-embolization plays a pivotal role[47,48].

CMI's management is mostly surgical. Due to the high rates of thrombosis, medical management as a sole treatment is warranted only in patients whose surgical risk outweighs potential benefits. Additionally, conservative treatment, such as bowel resting, smoking cessation and administration of vasodilator drugs, will not ameliorate disease progression[49]. Medicines used in CMI includes heparin and warfarin to prevent an acute thrombotic/embolic event, intra-arterial papaverine as a vasodilator prior to surgery to reduce risk of arterial spasm and nitrate therapy to provide short-term relief. Parenteral nutrition is warranted because of the long period of malnutrition[5,28].

Open revascularization (OR) and endovascular revascularization (ER) are the alternative modalities of treatment in patients with CMI[50-52]. ER appears to have lower postoperative mortality and morbidity rates and shorter intensive care unit and hospital stay. Therefore, ER has been proposed for high risk surgical candidates or those with short life expectancy due to its minimally invasive nature[35,52,53]. In ER, a short stump of the patent artery is necessary to gain wire access. Excessive endovascular manipulation can result in arterial dissection, perforation, or embolization. Moreover, severely calcified or long lesions and small-diameter mesenteric arteries are also associated with an increased risk of distal embolization and restenosis. Nevertheless, OR presents early as well as long term symptomatic relief and significantly lower restenosis rates compared to ER. Thus, the majority of the patients should be treated with conventional reconstruction especially if ER has previously attempted unsuccessfully. In conclusion, OR is still predominantly proposed although ER had recently gained popularity as an alternative therapeutic approach[37,52,54].

Surgical management includes transaortic endarterectomy of the celiac or SMA, antegrade bypass from the supraceliac aorta and retrograde bypass from the infrarenal aorta or the common iliac artery. Bypasses may be achieved using vein or prosthetic material. Primary stenting is favored in highly calcified, thrombotic, occlusive, or dissected lesions. Little is known about the impact of stent placement in terms of distal embolization. An embolic protection device may be considered in the presence of a large thrombus burden. However, there is limited evidence showing its efficacy during ER for CMI[37,50,51].

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