Management Of Intestinal Ischemia

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In General Surgery

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LIST OF ABBREVIATIONS

AMI	Acute Mesenteric Ischemia.
CA	Celiac Artery.
CI	Colonic Ischemia.
CMI	Chronic Mesenteric Ischemia.
CT	Computed Tomography.
CTA	Computed Tomography Angiography.
DVT	Deep Venous Thrombosis.
IMA	Inferior Mesenteric Artery
LMWH	Low Molecular Weight Heparin.
MDCT	Multidetector Computed Tomography
MDCTA	Multidetector Computed Tomography Angiography.
MRA	Magnetic Resonance Angiography.
MVT	Mesenteric Venous Thrombosis
NOMI	Nonoclusive Mesenteric Ischemia.
OMAI	Occlusive Mesenteric Arterial Ischemia.
PTFE	Polytetrafluroethelyne
ROMS	Retrograde Open Mesenteric Stenting.
rt-PA	recombinant tissue plasminogen activator.
SMA	Superior Mesenteric Artery
SMV	Superior Mesenteric Vein.
TIPS	Transjugular Intrahepatic Portosystemic Shunting.
VKA	Vitamin K Antagonist.

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Summary

Mesenteric ischemia occurs when perfusion of the visceral organs fails to meet normal metabolic requirements. This disorder is categorized as either acute or chronic, based on the duration of symptoms. The most common cause of AMI is embolization to the SMA. Arterial thrombosis constitutes the next most common cause of AMI and occurs in 20% to 35% of cases. Preexisting atherosclerotic plaque affecting all visceral vessels is the most common finding. Impaired intestinal perfusion in the absence of thromboembolic occlusion is termed nonocclusive mesenteric ischemia (NOMI). Symptomatic patients are frequently found to have extensive atherosclerosis, with involvement of all three visceral arteries. MVT constitutes 5% to 15% of all cases of mesenteric ischemia. Involvement is usually limited to the superior mesenteric vein but can also involve the inferior mesenteric vein and portal vein.The most common symptom of AMI associated with arterial thromboembolic disease is the sudden onset of abdominal pain. Lack of collateral flow to the visceral organs leads to a more dramatic presentation in AMI, with severe, rapid clinical deterioration. Nausea, vomiting, diarrhea, and abdominal distention can also occur.Postprandial abdominal pain and progressive weight loss are the most common symptoms in patients with CMI. Pain is often described as dull and crampy and located in the midepigastric region ***(Cronenwett and Johnston, 2010).***

Duplex ultrasonography is a useful tool for the early, noninvasive diagnosis of visceral ischemic syndromes. Color Doppler scanning can be used to assess the flow velocities and resistance index in the splanchnic arteries and their arterial beds. Computed tomography (CT) is an accurate,

noninvasive imaging modality for diagnosing mesenteric ischemia. Advantages over conventional angiography include the relative ease and speed of performance. Common radiographic findings in the bowel wall related to AMI include increased thickening, dilatation, and attenuation, which can be easily detected using CT. Disadvantages of CT include the risk of contrast nephropathy and hypersensitivity reactions to iodinated contrast agents. Inaccurate timing of contrast infusion during the arterial phase may provide indeterminate images and delay diagnosis. Magnetic resonance angiography (MRA) is useful for diagnosing mesenteric occlusive disease. Because MRA takes significantly longer to perform than CTA, its role in evaluating patients with AMI is limited. Conventional angiography remains the "gold standard" in the diagnosis of mesenteric ischemia. Anteroposterior and lateral views of the visceral aorta, as well as selective catheterization of the celiac trunk, SMA, and IMA, provide the most accurate and specific localization of stenotic and occlusive lesions ***(Cronenwett and Johnston, 2010).***

Therapeutic alternatives such as balloon angioplasty, stenting, and thrombolysis and percutaneous thrombus extraction can all be used to restore luminal visceral blood flow. Medical treatment alone is not effective in these patients. Preventive risk factor modification helps control the progression of atherosclerosis in the mesenteric circulation. Patients with known risks for inheritable hypercoagulable disorders should undergo screening and should be treated with systemic anticoagulation. Advances in endovascular techniques have greatly expanded the role of percutaneous interventions for patients with mesenteric ischemia. However, endovascular management remains largely limited to patients with CMI. Because patients

with AMI frequently require intestinal resection, laparotomy with open revascularization is the preferred method of treatment. In those with short-segment stenoses, cardiac and pulmonary co-morbidities, prior abdominal surgery, coagulopathy, or malnutrition, endovascular therapy is often favored. More complex lesions and complete arterial occlusions traditionally favor open revascularization ***(Sreenarasimhaiah, 2005).***

Laparotomy with visceral revascularization can be used to treat patients with both AMI and CMI. Several techniques for the restoration of intestinal perfusion are available to the vascular surgeon ***(Park et al., 2002).***

Introduction

Intestinal ischemia is the term used to describe the result of a variety of disorders which reflects a state of insufficient blood flow to the small intestine, the colon, or both. The injury of this (low -flow) state can range from a mild bout of abdominal pain , to a more grave situation which may require surgery or possibly embolectomy depending on the particular vessel or vessels involved ***(Brandt and Boley, 2000).***

A. Acute intestinal ischemia :

1- Arterial thrombosis as:

Severe atherosclerosis (usually at the origin of the mesenteric vessel), Systemic vasculitis, Dissecting aneurysm, Angiographic procedures, Aortic reconstructive surgery, Surgical accidents, Hypercoagulable states and oral contraceptives.

2- Arterial embolism as:

Vegetations (as with endocarditis or myocardial infarction with mural thrombosis), Angiographic procedures and Aortic atheroembolism.

3- Venous thrombosis as:

Hypercoagulable states induced for example, by oral contraceptives or antithrombin III deficiency, Intraperitoneal sepsis, Post-operative state, Vascular- invasive neoplasms (particularly hepatocellular carcinoma) and Cirrhosis and abdominal trauma ***(Cronenwett and Johnston, 2010).***

Acute occlusion of one of the three major supply trunks of the intestine which is celiac, superior, and inferior mesenteric arteries may lead to infarction of extensive segments of intestine. However, insidious loss of one vessel may be without effect, owing to the rich anastomotic inter-

communication between the vascular beds. Lesions within the end-arteries that penetrate the gut wall produce small, focal ischemic lesions. The severity of injury ranges from transmural infarction of the gut involving all visceral layers, to mural infarction of the mucosa and submucosa sparing the muscular wall, to mucosal infarction, if the lesion extends no deeper than the muscularis mucosa ***(Crawford, 2000).***

B. Chronic intestinal ischemia:

In which blood flow to the intestines is reduced over time caused by: Atherosclerotic involvement of the large mesenteric arteries is almost always the cause of this form of intestinal ischemia, Thromboangitis obliterans (Buerger'sdisease) and Polyarteritis nodosa also can produce chronic intestinal ischemia. Early and accurate diagnosis of intestinal ischemia depends on careful history taking, repeated clinical examination, and serial laboratory, radiographic and endoscopic evaluation of the patient. The more severe cases can be difficult to distinguish from other causes of acute abdomen, whereas the less severe cases can mimic acute or chronic idiopathic enterocolitis. Undetected and untreated intestinal ischemia may be fatal. This condition, though uncommon, is serious and often requires immediate medical care. Depending on the cause of intestinal ischemia, treatment options may include medications (as resuscitation by papaverine hydrochloride infusion, vasodilator and anticoagulant drugs) or surgery (as resection of ischemic intestine) or a procedure to open arteries (as synthetic or autogenous vein bypass graft circumventing the stenotic segment) ***(Edward, et al. 2005)***.

Aim of The Work

The aim of this work is to review the Literatures regarding intestinal ischemia for better understanding of its pathology and management aiming at improving the outcome of this surgical dilemma.

Anatomy and Embryology

The primitive dorsal aorta gives rise to the abdominal aorta during fetal development. Ventral segmental arteries emerge from the primitive ventral aorta, which disappears around the fourth week of gestation. Multiple segmental branches from the primitive ventral aorta-the 10th, 13th, and 21st-persist and develop into the celiac artery, SMA, and inferior mesenteric artery (IMA), respectively ***(Cronenwett and Johnston, 2010).***

Mesentery:

It is a fold of peritoneum and extraperitoneal tissue which attatches the whole length of the jejunum and ileum (up to 6m) to an oblique line across the posterior abdominal wall from the duodenojejunal flexure towards the ileocaecal junction. The short (15cm) root contains the superior mesenteric vessels and cross the anterior surfaces of the third part of the duodenum, aorta, inferior vena cava and the testicular or ovarian vessels and ureter on the right psoas muscle. The mesentery contains the jejunal and ileal blood vessels, large lacteal lymph vessels draining to the lymph nodes in and at the base of the mesentery, a considerable plexus of autonomic nerves, and extraperitoneal fatty tissue ***(Yasuhara, 2005).***

Blood supply of the mesentery:

The arterial supply to the gut is derived from three main arteries; the celiac artery [CA] which is the artery of the foregut and supplies the gastro intestinal tract from the lower one-third of the esophagus down as far as the middle of the second part of the duodenum. The superior mesenteric artery [SMA] is the artery of the midgut and supplies gastrointestinal tract from the middle of the second part of the duodenum as far as the distal one-third of the transverse colon. The inferior mesenteric artery [IMA] is the artery of the

hindgut and supplies the large intestine from the distal one-third of the transverse colon to half way down the anal canal ***(Yasuhara, 2005).***

[1] The Celiac Artery [CA]:

The celiac artery arises from the abdominal aorta just caudal to the diaphragm at the level of L1 and is bordered by the median arcuate ligament at the aortic hiatus superiorly and the superior border of the pancreas inferiorly. Traditionally, the three branches from this common trunk include the left gastric, splenic, and common hepatic arteries ***(Cronenwett and Johnston, 2010).***

Branches Of Celiac Artery

(A) ***Left gastric artery****:* the small left gastric artery runs to the cardiac end of the stomach, gives off few esophageal branches and then turns to the right along the lesser curvature of the stomach. It anastomoses with the right gastric artery ***(Agur and Daliey, 2005).***

(B) ***Splenic artery***: splenic artery runs in a course along the upper border of the pancreas and behind the stomach. On reaching the left kidney, the artery enters the lienorenal ligament and runs to the hilum of the spleen. It gives off pancreatic branches, left gastroepiploic artery and short gastric arteries ***(Agur and Daliey, 2005).***

(C) Hepatic artery: This medium sized hepatic artery runs forward and to the right and then ascends between the two layers of the free border of lesser omentum, It lies in front of the opening into the lesser sac and is placed to the left of the bile duct and in front of the portal vein. At the porta hepatis, it divides into right and left branches to supply the corresponding lobes of the liver. It gives the right gastric artery, the gastroduodenal artery and the right and left hepatic arteries ***(Agur and Daliey, 2005).***