Recent Modalities In Management Of Mesenteric Vascular Occlusion

Essay

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Presented by

Mohamed Sayed Abd-AlAzim Mahmoud Heikal

M.B,B.ch

Under supervision of

Prof. Dr. Mohamed Ahmed KhalafAllah

Professor of General Surgery

Faculty of Medicine, Ain Shams University

Dr. Mohamed Ahmed Rady

Assistant professor of General Surgery

Faculty of Medicine, Ain shams University

Dr. Mostafa Abdo Mohamed

Assistant professor of General Surgery

Faculty of Medicine, Ain Shams University

Faculty of Medicine

Ain Shams University

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LIST OF ABBREVIATIONS

ACC/AHA	American College of Cardiology/American Heart Association
AE	arterial embolism
AMI	acute mesenteric ischemia
Ant PDA	Anterior pancreaticoduodenal artery
AoCMI	*Acute-on-chronic ischemia*
AP	antero-posterior
aPTT	activated Partial Thromboplastin Time
AT	arterial thrombosis
BES	balloon expandable stents
CA	celiac artery
CACS	celiac artery compression syndrome
CE-MRA	contrast-enhanced magnetic resonance angiography
CHA	common hepatic artery
CMI	chronic mesenteric ischemia
CRP	C-reactive protein
CT	computed tomography
CTA	computed tomography angiography
DP	dorsal pancreatic
DSA	digital-subtraction angiography
DVT	deep venous thrombosis
EDV	end diastolic velocity
ER	endovascular revascularization
EVT	endovascular therapy
FFR	fractional flow reserve
FMD	fibromuscular dysplasia
GDA	gastroduodenal artery

GEA	gastroepiploic artery
GST	glutathione S transferase
HA	hepatic artery
I-FABP	intestinal fatty acid binding protein
IMA	inferior mesenteric artery
IMV	inferior mesenteric vein
IPDA	inferior pancreaticoduodenal artery
IVC	inferior vena cava
LGA	left gastric artery
LMWH	low molecular weight heparin
MALS	median arcuate ligament compression syndrome
MCA	middle colic artery
MCV	middle colic vein
MDCT	Multi-detector computed tomography
MRA	magnetic resonance angiography
MVT	Mesenteric venous thrombosis
NEC	necrotizing enterocolitis
NIS	Nationwide Inpatient Sample
NOMI	Non-occlusive mesenteric ischemia
OR	Open revascularization
OSR	open surgical revascularization
PAD	Peripheral Artery Disease
PMAS	Percutaneous Mesenteric artery Angioplasty and Stenting
Post PDA	posterior pancreaticoduodenal artery
PSV	peak systolic velocity
PTA	percutaneous transluminal angioplasty
PTFE	polytetrafluoroethylene
PV	portal vein
RAAA	ruptured abdominal aortic aneurysm

RAS	Renin-angiotensin system
RCT	randomized controlled trials
RGA	right gastric artery
RIMA	right inferior mammary artery
ROMS	Retrograde Open Mesenteric Stenting
rt-PA	recombinant tissue plasminogen activator
RVD	reference vessel diameter
SA	splenic artery
SAE	serious adverse event
SBS	short bowel syndrome
SMA	superior mesenteric artery
SMV	superior mesenteric vein
SPDA	superior pancreaticoduodenal artery
SV	splenic vein
US	Ultrasonography
VT	venous thrombosis
WBC	white blood cell

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INTRODUCTION

Mesenteric ischemia accounts for approximately 1% of acute abdomen hospitalizations. Early non-specific symptoms of these cases is a big challenge in identification, distinguishing it from less lethal cases and early diagnosis inspite of major diagnostic and treatment advances over the past decades. The mortality of acute mesenteric ischemia (AMI) in adults has been reported at 70% to 90%. It has been reported at 40% in infants with necrotizing enterocolitis (NEC). A 24 hours delay decreases survival rates by 20%.Therefore, development of new diagnostic strategies is of great importance (**Coco and Leanza, 2018)**.

In 70% to 80% of cases, intestinal ischemia is caused by an arterial embolus or thrombosis within the superior mesenteric artery. In cases of embolic occlusion, the absence of a well-developed collateral circulation causes earlier ischemia and transmural necrosis compared to other causes of mesenteric ischemia. Other causes are venous thrombosis, vascular anomalies and non-thrombotic mechanical causes such as strangulated hernia. Vasculitis is a common cause of mesenteric ischemia in younger people with auto-immune disease. ***(Menke, 2010)***

In the presence of hypotension, local vasodilation, increasing oxygen extraction and internal intestinal redistribution of blood flow to the metabolically more demanding mucosa ensures adequate perfusion within a wide pressure range**.** Within 6 hours, an acute complete disruption to intestinal blood supply leads to irreversible mucosal ischemia with cellular energy loss and leucocyte infiltration, accompanied by the formation of oxygen radicals ***(Hokama A, 2012)***.

There are a wide variety of clinical presentations for mesenteric ischemia. Classically, acute mesenteric ischemia (AMI) is associated with a dramatic onset of severe cramp-like abdominal pain disproportionate to physical exam findings, followed after approximately 3 to 6 hours by a deceptive pain-free interval caused by a decline in intramural pain receptors as a result of sustained under-perfusion of the intestinal wall. Peritonitis and septicemia develop once the ischemia has progressed transmurally. (**Murugesan et al., 2015)**

In patients with chronic mesenteric ischemia, the association of pain and nausea with meals leads to fear of eating and subsequent weight loss (**Guo et al., 2017).**

Classically, patients with mesenteric ischemia have leukocytosis (may exceed 20×109/L), metabolic acidosis, an elevated D-dimer and elevated serum lactate. In a study by Acosta et al, said that initial blood gas analysis showed metabolic alkalosis more frequently than metabolic acidosis; this finding results from profound vomiting during early bowel ischemia. Ischemia modified albumin, a biochemical marker detected in a number of acute ischaemic conditions, was found to be elevated in patients with acute mesenteric arterial occlusion but not in healthy controls ***(Sise, 2010).***

In AMI, ischemia starts at the mucosa and extends toward the serosa. An ideal biomarker for mesenteric ischemia should originate at the mucosa to detect ischemia at the earliest stage. A highly specific, sensitive and non-invasive marker is needed to identify patients earlier. According to a recent review intestinal fatty acid binding protein (I-FABP), alpha-glutathione S transferase (GST) and D-lactate are the most promising plasma markers for mesenteric ischemia ***(Acosta, 2012).***

New diagnostic strategies aim for early identification (e.g. biochemical markers) or seek to optimize accurate diagnosis using existing modalities such as contrast-enhanced magnetic resonance angiography of the splanchnic vessels (CE-MRA). Although dynamic CE-MRA yielded a sensitivity and specificity of 0.95 and 1.00 in a clinical trial designed to diagnose severe stenosis or occlusion of the origins of the celiac axes and superior mesenteric artery, this modality is limited in identification of more distally located occlusions and does not have the same spatial resolution and acquisition time as CTA. If better spatial resolution becomes available in the future, CE-MRA has the potential to become the diagnostic modality of choice because it is non-invasive and avoids the nephrotoxicity and allergic risks associated with iodinated contrast agents ***(Wyers, 2010).***

Multi-detector computed tomography (MDCT) technique with contrast enhancement allows fast scanning of the mesenteric vasculature with high spatial resolution enabling multi-planar image reconstruction. CTA is a non-invasive, widely available and fast imaging modality with high sensitivity and specificity for evaluation of the mesenteric vasculature. Despite the radiation and intravenous nephrotoxic contrast exposure, CTA is the first-line approach for AMI and CMI. (**Loeffler et al., 2017)**

The general management of acute mesenteric ischemia is based on clinical suspicion from the patient's presentation followed by immediate laparotomy if signs of peritonitis are present or obtaining diagnostic imaging tests if peritonitis is not present. Thus, laboratory tests are most commonly used to exclude other possible diagnoses ***(Oldenburg et al, 2004).***

Every patient with AMI should concomitantly receive emergency diagnosis and treatment according to the principles of intensive care. The first

procedure is intravascular fluid replacement to stabilize hemodynamics, as volume displacement to the ischemic portions of the intestines and general endothelial disintegration occur within a few hours. In order to prevent exacerbation of thromboembolic occlusion, immediate anticoagulation should be performed. Antibiotic treatment must be started concomitantly (e.g. second-generation cephalosporin plus metronidazole). **(Stone and Wilkins, 2015)**

Endovascular treatment includes the possibility of angiographically-directed catheter-aspiration embolectomy and/or catheter lysis with recombinant tissue plasminogen activator (rt-PA), urokinase, or pharmacotherapy with prostaglandin E1. **(Wasnik et al., 2015)**

Immediate surgical intervention remains the treatment of choice for central occlusion of the superior mesenteric artery, failure of endovascular treatment, or peritonitis. Cooperation between visceral and vascular surgeons is essential for this. Treatment is guided by the principle of arterial reperfusion before intestinal resection is considered. From a vascular point of view, mastery of embolectomy as well as reconstruction techniques for visceral arteries is required ***(Acosta S, 2012).***

Acute mesenteric ischemia is a vascular emergency in need for early diagnosis and rapid onset of optimum treatment during its initial stage to lower the mortality rate. **(Gifford et al., 2015)**

AIM OF THE WORK

The aim of this study is to highlight the recent trends in management of patients with mesenteric vascular occlusion.

Chapter **(1)**

ANATOMY AND PHYSIOLOGY OF THE MESENTERIC CIRCULATION

I. ARTERIAL ANATOMY

A. Celiac Axis :

The celiac axis (CA) originates at the level of the T12 or L1 vertebral body at a relatively acute angle, traveling for 1 to 2 cm before trifurcating into the left gastric , common hepatic , and splenic arteries. The left gastric artery anastomose with the right gastric artery, which emanates from the common hepatic artery (CHA). (**Skinner and Van Fossen, 2018)**

The CHA gives off the right gastric artery and the gastroduodenal artery before becoming the proper hepatic artery. The proper hepatic artery branches into the right and left hepatic arteries. **(Kotsis et al.,2018)**

The splenic artery is the largest branch of the celiac axis and follows a tortuous course along the superior aspect of the pancreas. **(Carmichael and Mills, 2016)**

B. Superior Mesenteric Artery

The SMA arises from the anterior surface of the aorta at the level of the first lumbar vertebral body. Its first branch is the inferior pancreaticoduodenal artery that divides into the anterior inferior-pancreaticoduodenal and posterior inferior-pancreaticoduodenal arteries. These arteries anastomose with the anterior and posterior superior-pancreaticoduodenal arteries originating from the gastroduodenal artery to form two robust arterial arcades supplying the head of the pancreas and the

duodenum (**Fig**. **1**). The second branch of the SMA is the middle colic artery. The third branch of the SMA is the right colic artery which commonly arises from the SMA as a common trunk with the middle colic artery. The fourth branch of the SMA is the ileocolic artery which arises from the right side of the SMA and subdivided into superior and inferior branches. The appendiceal artery commonly originates from its inferior branch. **(Henry et al., 2018)**

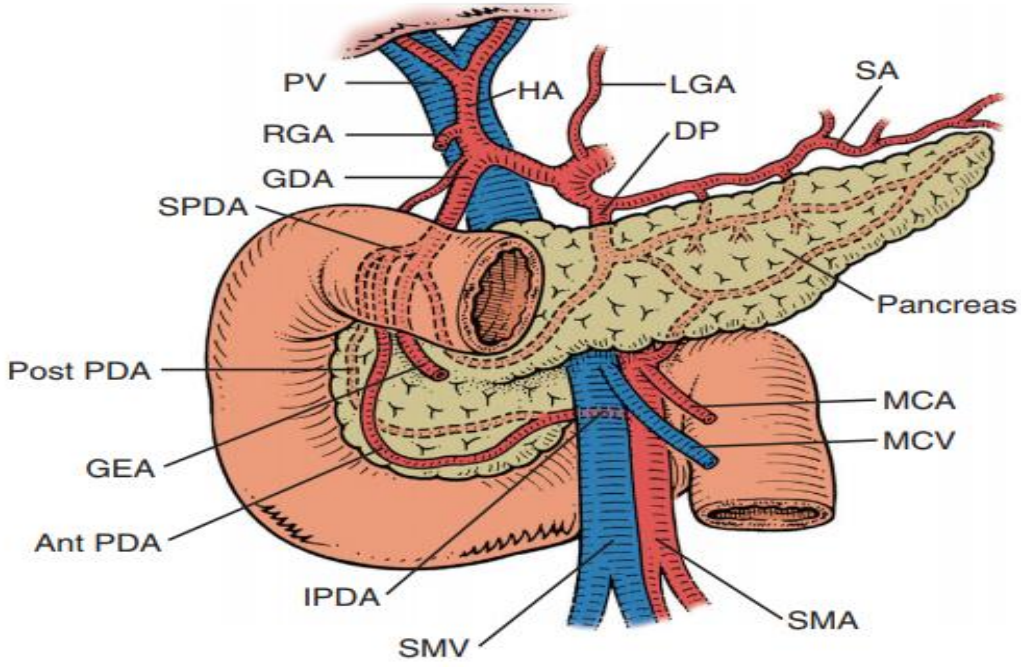

FIGURE 1 Arterial vascular arcades supplying the duodenum and pancreas. Ant PDA, Anterior pancreaticoduodenal artery; DP, dorsal pancreatic artery; GDA, gastroduodenal artery; GEA, right gastroepiploic artery; HA, hepatic artery; IPDA, inferior pancreaticoduodenal artery; LGA, left gastric artery; MCA, middle colic artery; MCV, middle colic vein; Post PDA, posterior pancreaticoduodenal artery; PV, portal vein; RGA, right gastric artery; SA, splenic artery; SMA, superior mesenteric artery; SMV, superior mesenteric vein; SPDA, superior pancreaticoduodenal artery. (**Henry et al., 2018)**

C. Inferior Mesenteric Artery

The IMA arises from the left anterior surface of the aorta at the level of the third lumbar vertebral body. The IMA giving rise to the left colic, sigmoidal, and superior rectal vessels. (**Nair and Kumar, 2016)**

D. Collateral Pathways

Natural collateral pathways between these mesenteric vessels can circumvent chronic vascular occlusive disease, provided the efficiency of this collateral network adequately meets the metabolic demand. The mesenteric circulation is well known for its rich collateral network and resilience to chronic ischemia. However, these pathways may not be sufficient in acute ischemic events. (**Sahin et al., 2018)**

1. *Celiac Axis—Superior Mesenteric Artery* :

The major collateral pathway of critical importance, especially in the presence of occlusive disease affecting the celiac axis or the SMA, is between the superior pancreaticoduodenal vessels, from the gastroduodenal artery, and the inferior pancreaticoduodenal vessels, from the SMA. Additional important collateral channels can also exist between the dorsal pancreatic artery and the SMA (**Fig**. **2**, see also **Fig**. **1**). (**Olewnik et al., 2017)**

2. *Inferior Mesenteric Artery—Hypogastric Artery* :

A rich and sometimes important collateral pathway exists between the mesenteric circulation and the systemic circulation because of the anastomosing channels between the superior rectal and middle and inferior rectal vessels (see **Fig**. **2**). The superior rectal vessels supply circulation to the upper two-thirds of the rectum, and the middle and inferior rectal vessels supply the remaining one-third with the inferior rectal vessels also supplying the anal canal. (**Olewnik et al., 2017)**