Damage control surgery--Massive small bowel gangrene

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Principles of damage control surgery

- Control hemorrhage
- Prevention of contamination
- Avoid further injury

- Any areas of bowel that are obviously necrotic and non viable should be resected at this first operation.
- Any segments of bowel that are questionable in appearance are left to be checked in 24 hrs at the second look operation

- In1881,Koeberle reported the first case of bowel resection and for a long time the term 'massive’ was used to designate resections of 200 cm or more of small bowel.
- The three commonest diseases requiring resection are *Infarction of the bowel* due to

① vascular or mechanical causes ,

② extensive involvement by Crohn's and

③ trauma to the bowel and its bloodsupply (ColcockandBraasch,1968).

Acute mesenteric ischemia

- Acute mesenteric ischemia (AMI) is a syndrome in which inadequate blood flow through the mesenteric vessels causes ischemia and eventual gangrene of the bowel wall.
- Either arterial or venous disease

•The disease may evolve in a chronic fashion as in the case of progressive luminal obstruction d/t atherosclerosis .

or , can occur suddenly in thromboembolism.

Can be described as the most catastrophic vascular disorders with mortality rates 50-75%.

History

- 1500 - First described in Florence by Antonio Beniviene
- 1815 - Guy's Hospital, London1875 - Litten ligates the SMA of an animal and records events
- 1894 - Councilman describes chronic MI
- 1895 - Venous thrombosis (MVT) described by Elliott
- 1901 - chronic mesenteric ischemia described by schnitzler
- 1926 -mesenteric venous thrombosis by cokkinis
- 1940 - Heparin first used
- 1950 - First SMA embolectomy without bowel resection
- 1957 – intestinal angina term used by mikkelsen
- 1958 - First successful revascularization after SMA thrombosis
- 1959 - Non-occlusive mesenteric ischemia (NOMI) first described in NEJM
- 1960 - Arteriography used successful and became standard of diagnosis

- since 1930, many advances have been made that allow earlier diagnosis and treatment.
- Whereas the prognosis remains grave

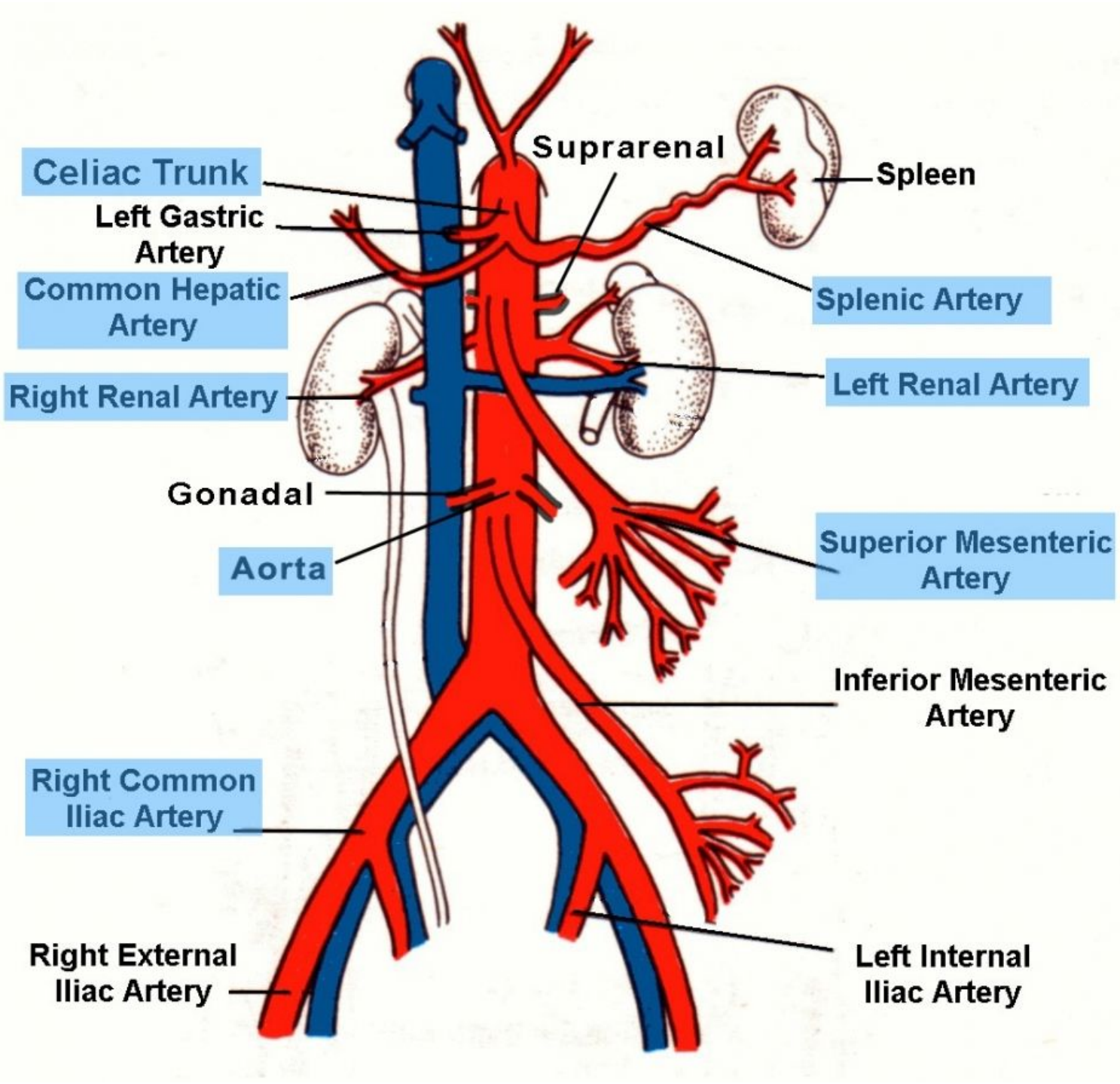
Clinical entities

- AMI comprises 4 different primary clinical entities: NOMI, AMAE, AMAT, and MVT.
- Arterial disease may be subdivided into *nonocclusive mesenteric ischemia* (NOMI)and *occlusive mesenteric arterial ischemia* (OMAI).
- OMAI may be further subdivided into *acute mesenteric arterial embolus* (AMAE) and acute mesenteric arterial thrombosis (AMAT). Venous disease takes the form of *mesenteric venous thrombosis* (MVT).

Mesenteric vasculature

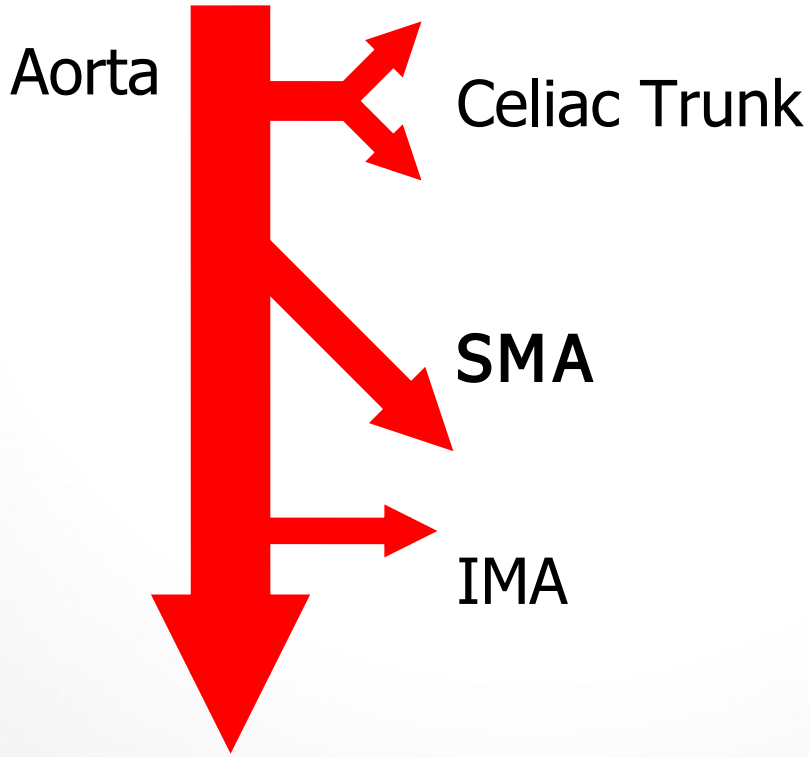
- **Coelic axis** stomach, duodenum, liver, spleen, pancreas.

- **Superior mesenteric artery**-- duodenum, pancreas, smallbowel, proximal colon upto splenic flexure
- **Inferior mesenteric artery**-- left colon, rectum.
- Venous drainage is through the superior mesenteric vein (SMV), which joins the splenic vein to form the portal vein


Celiac Trunk
Left Gastric Artery
Common Hepatic Artery
Right Renal Artery
Gonadal
Aorta
Right Common Iliac Artery
Right External Iliac Artery
Suprarenal
Spleen
Splenic Artery
Left Renal Artery
Superior Mesenteric Artery
Inferior Mesenteric Artery
Left Internal Iliac Artery

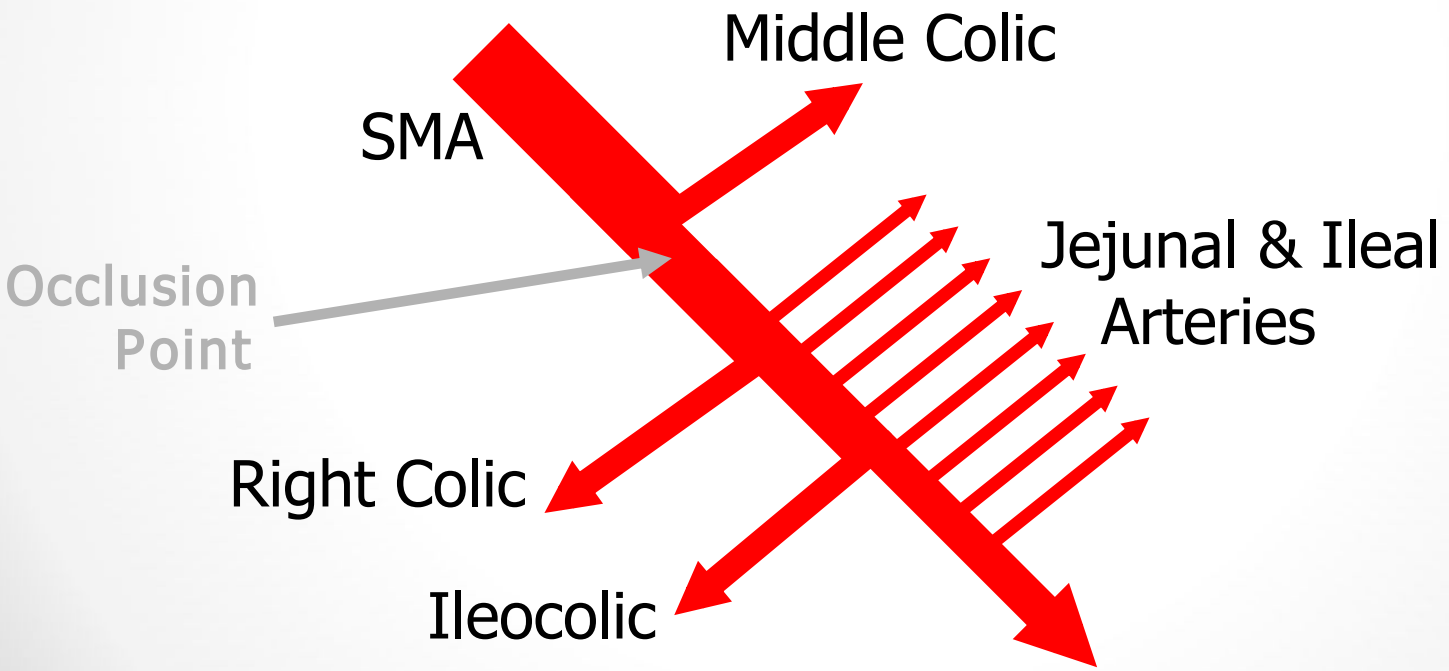
Superior Mesenteric Artery (SMA)

- Largest caliber vessel + 45-degree angle makes it most commonly occluded



Superior Mesenteric Artery (SMA)

- Emboli occlude past the middle colic, causing small bowel ischemia



Etiologies of Acute Mesenteric Ischemia (AMI)

- SMA Occlusion (at least 60% of cases)
  - Embolism: MI, Afib, Endocarditis, Valve d.
  - Thrombosis: Atherosclerosis – plaque rupture
- Nonocclusive Mesenteric Ischemia (NOMI)
  - Atherosclerosis + shock + vasopressors
- Mesenteric Venous Thrombosis (MVT)
  - Primary clotting disorder

Etiologies of Acute Mesenteric Ischemia (AMI)

- Focal small bowel ischemia - rare
  - Partial malrotation, volvulus, mesenteric hematoma, strangulated hernia
- Unknown
  - ?Mesenteric small vessel disease

- **Causes of embolic AMI (AMAE)** include the following:
- Cardiac emboli - Mural thrombus after myocardial infarction, auricular thrombus associated with mitral stenosis and atrial fibrillation, septic emboli from valvular endocarditis (less frequent)
- Emboli from fragments of proximal aortic thrombus due to a ruptured atheromatous plaque
- Atheromatous plaque dislodged by arterial catheterization

- Causes of **thrombotic AMI** (AMAT) include the following:
- Atherosclerotic vascular disease (most common)
- Aortic aneurysm
- Aortic dissection
- Arteritis
- Decreased cardiac output from myocardial infarction or CHF (thrombotic AMI may cause acute decompensation)
- Dehydration from other causes

- **Causes of NOMI** include the following:
- Hypotension from CHF, myocardial infarction, sepsis, aortic insufficiency, severe liver or renal disease, or recent major cardiac or abdominal surgery
- Vasopressive drugs
- Ergotamines
- Cocaine
- Digitalis (whether digitalis use causes NOMI or patients who develop NOMI are older and are more likely to have been prescribed digitalis is unclear)

- **Causes of MVT** include the following (>80% of patients with MVT are found to have predisposing conditions):
- Hypercoagulability from protein C and S deficiency, antithrombin III deficiency, dysfibrinogenemia, abnormal plasminogen, polycythemia vera (most common), thrombocytosis, sickle cell disease, factor V Leiden mutation, pregnancy, and oral contraceptive use
- Tumor causing venous compression or hypercoagulability (paraneoplastic syndrome)
- Infection, usually intra-abdominal (eg, appendicitis, diverticulitis, or abscess)
- Venous congestion from cirrhosis (portal hypertension)
- Venous trauma from accidents or surgery, especially portocaval surgery
- Increased intra-abdominal pressure from pneumoperitoneum during laparoscopic surgery
- Pancreatitis

Pathophysiology

- Insufficient perfusion of the small bowel and colon may result from arterial occlusion by embolus or thrombosis (AMAE or AMAT), thrombosis of the venous system (MVT), or nonocclusive processes such as vasospasm or low cardiac output (NOMI).

- Bowel mucosa is most vulnerable to inadequate blood flow with ischemia as well as reperfusion injury resulting in tissue damage

Clinical features

Classic Presentation:

- Rapid onset of severe, unrelenting periumbilical pain
- *Pain out of proportion to findings on physical examination.*
- Nausea and vomiting
- Forceful/urgent bowel evacuation

SMA Thrombosis:

- Prodrome of postprandial pain/nausea and weight loss
- Presentation with classic symptoms

Non-occlusive Mesenteric Ischemia:

Unexplained decline in clinical status or failure to follow expected recovery

Mesenteric Venous Thrombosis:

- Fever
- Abdominal distension
- Hemoccult positive stool

Physical Examination

- The different etiologies notwithstanding, physical examination findings are generally similar in patients with AMI. The main distinction is between early and late presentation. Early in the course of the disease, in the absence of peritonitis, physical signs are few and nonspecific. Tenderness is minimal to nonexistent. Stool may be guaiac positive.

Diagnostic Considerations

- Because acute mesenteric ischemia (AMI) is a condition with an unclear initial presentation, serious morbidity, and a high mortality rate without proper treatment, clinical suspicion should remain high. Obtain early angiography if any suspicion of AMI exists. Subsequent treatment should be initiated as rapidly as possible. No patient in whom AMI is suspected should be discharged unless AMI can be ruled out.

Investigations

- Elevated wbc count
- Plain radiograph(rule out perforation and obstruction)
- Duplex scanning
- Ct angiography
- Mesenteric angiography
- MR angiography
- Diagnostic laparoscopy

Mesenteric angiography

advantages

- Intra arterial vasodilatation.
- Thombolysis.
- Angioplasty
- stenting

disadvantages

- Invasive
- Limited emergency avilability
- Contrast associated nephrotoxicity
- Inability to assess intestinal infarction

Differential diagnosis

- Intestinal obstruction
- Perforated viscus
- Pancreatitis
- Cholecystitis
- diverticulitis

MANAGEMENT

- ✓ General measures
- ✓ Medical and endovascular treatments
- ✓ Surgical management

General measures

- Oxygen inhalation
- Pain relief
- Fluid and electrolyte management - Aggressive fluid resuscitations is critical (significant fluid shifts and sequestration)
- Broad septrum antibiotics (bacterial translocation)
- Naso gastric tube decompression decrease intraluminal pressure and minimize reduction of blood flow

Medical and endovascular

- Indicated only when no signs of peritonitis
- SMAE/SMAT—aspiration embolectomy , Cather directed embolectomy, mechanical thrombus fragmentation , Stent insertion
- NOMI---intra arterial vasodilator papavarin at the time of angiography
- SMVT---rapid anticoagulation with heparin f/l by warfarin

Surgical management

Main goals of the operative treatment

- Revascularization
- Assessment of bowel viability and resection of the necrotic bowel
- Endovascular approach if no evidence of bowel ischemia
- Restore mesenteric flow
- Preserve small bowel

- 50 cm of viable small bowel required to sustain life if colon is present
- Life long parenteral nutrition or intestinal transplantation if less than 50cm small bowel or less than 100cm small bowel with out colon are present
- SMA is assessed intraoperatively by palpation ,handheld Doppler , intra operative angiography.

Complications

- Bowel necrosis necessitating bowel resection
- Septic shock
- Death

Prognosis

- The prognosis of AMI of any type is grave. 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%. Even with good treatment, up to 50-80% of patients die.