

## **Aneurysms**

- Focal Arterial Dilatation
- A localised dilatation of an artery with an increase in diameter of 50% or more than that of the non-dilated adjacent vessel
- Prevalence:
  - In men >65yrs: 7-8%
  - Six times higher in men Vs female
  - 25% of patients with AAA have Femoral or Popliteal aneurysms

## **pathogenesis**

- Aneurysm wall contains less Elastin & a considerable increase in Collagen
- Associated with loss of smooth muscle cells within the media and widespread inflammatory infiltration
- Elevated levels of MMPs, prostaglandins and proteolytic enzymes contribute to elastolysis

## **Aetiology**

- Atherosclerotic
- Hereditary:
  - Prevalence is 4 times higher in siblings of patients with known AAA
  - No single gene has yet been identified
- Connective tissue disorder:
  - Marfan's
  - Ehlers-Danlos type IV
- Inflammation (Inflammatory AAA); eg Takayasu aortitis
- Infection:
  - Syphilis
  - Salmonella Typhi
  - Staph Infection (Mycotic Aneurysm) In association with Subacute Bacterial Endocarditis

## **Most common sites**

- Aorta: infrarenal, iliac, femoral, popliteal arteries

## **Risk factors**

### **Modifiable**

- Cigarette smoking
- Hypertension
- Hyperlipidaemia
- Hyperhomocysteinaemia

### **Non-modifiable**

- Family history
- Gender (male)
- Age

## **Classification of aneurysms**

- True Vs Pseudo-aneurysms
- Fusiform Vs Saccular
- Atherosclerotic Vs Inflammatory Vs Mycotic
- Single Vs Multiple
- Location

## **Abdominal aortic aneurysms (AAA)**

- Supra-renal
- Juxta-renal
- Infra-renal

## **Iliac aneurysms**

- Usually a/w AAA
- Isolated iliac aneurysms <2% of aorto-iliac aneurysms

## **Common femoral aneurysms**

- Usually a/w AAA
- 25% AAA a/w femoral aneurysms

- true aneurysms <Pseudo-aneurysms

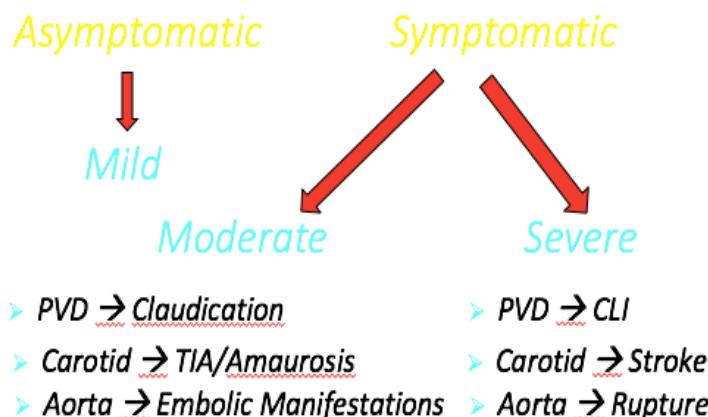
## **popliteal artery aneurysms**

- Most common peripheral aneurysm
- 80% of all peripheral aneurysms
- Popliteal aneurysm: AAA = 1:15
- 50% Bilateral
- 40-50% associated with AAA

## **abdominal aortic aneurysm**

- Def: permanent, irreversible 50% increase over the normal aortic diameter
- Normal aortic diameter for men is 3 cm and for women is 2.5 cm
- 85% of AAA happen as a result of atherosclerosis

# Presentation



## presentation

- 75% Asymptomatic
  - Incidental finding during course of investigation of unrelated symptoms
  - Pulsatile mass that the patients detect themselves
- Chronic abdominal pain with tenderness & no circulatory collapse (Inflammatory)
- Distal embolisation
  - Blue Toe Syndrome
  - Acute embolic arterial occlusion
- Severe abdominal pain and/or back pain with circulatory collapse
  - Leak

Note:

- Severe pain when aneurysms expands or ruptures
- Often begins in the central abdomen and radiates to the back (erosion of vertebral bodies), or flank. Can a/w abdominal distension, pulsating abdominal mass, shock due to massive blood loss
- Shock (hypotension, pallor, pale, threading pulse, drop urine output, tachycardia)
- Expansile is worrying, pulsatile can sometimes be seen in men as the diameter size is 3 cm
- Haematemesis, melena, PR bleed if it erodes into intestine causing aorto-intestinal fistula
- Embolic events (macro/ microemboli); the appearance of micro-embolic lower limb infarcts in patients with easily palpable pedal pulses suggest popliteal/ abdominal aneurysms- Blue toe syndrome= operate soon as risk of macroembolism
- High-output heart failure (CO higher than normal) due to aorto-caval fistula (where aneurysms erode into IVC)
- Haematuria-fistula or erodes into ureters

## Rupture AAA

- Sudden onset
  - Severe abdominal or back pain
  - Hypovolemic shock

- Pulsatile abdominal mass
- 5 year rupture rates:
  - 5-5.9cm → 25%
  - 6-6.9cm → 35%
  - >7cm → 75%

#### other Px

- High output heart failure
  - Erosion into Inferior Vena Cava (Aorto-Caval Fistula)
  - Loud machinery murmur
  - Hematemesis, melena or PR bleed
  - Aorto-Intestinal fistula
  - Sudden massive, but usually preceded with Sentinel bleeds
- General → Signs of shock!!!
- Abdomen → Pulsatile expansile mass
  - Differentiate from transmitted Epigastric pulsations
  - Tenderness
  - Iliac pulsations
  - Femoral & peripheral pulses

#### Investigations

- Identify patients in whom the balance of risks favours operative intervention
- Reduce peri-operative morbidity & mortality
- Assess the anatomical suitability for EVAR vs open repair
  1. General investigations + Investigation of Risk factors
    - a. FBC → Hb, WCC, Platelets
    - b. U&E's → Contrast
    - c. LFT's → Statins
    - d. Coag → Thrombophilia screen
    - e. Lipid Profile
    - f. Fasting Glucose & HbA1C
    - g. Homocysteine
    - h. CXR, ECG
    - i. LFT: to check before starting on statins
  2. Investigation of the Current Problem
    - a. ABI + Toe pressures
    - b. Duplex scan
    - c. CTA-if planning for surgery use this

Minimally invasive and highly accurate to determine size and extent of aneurysms and its location in the aorta. Precise imaging of aorta and iliac vessels is vital preoperatively especially if patient is to undergo EVAR (look for other peripheral artery aneurysms: popliteal, ileac)

- d. MRA
- e. Ultrasound

Ultrasound-has 98% accuracy in measuring the size of aneurysms and is safe and noninvasive view can be obscured by obesity or bowel gas

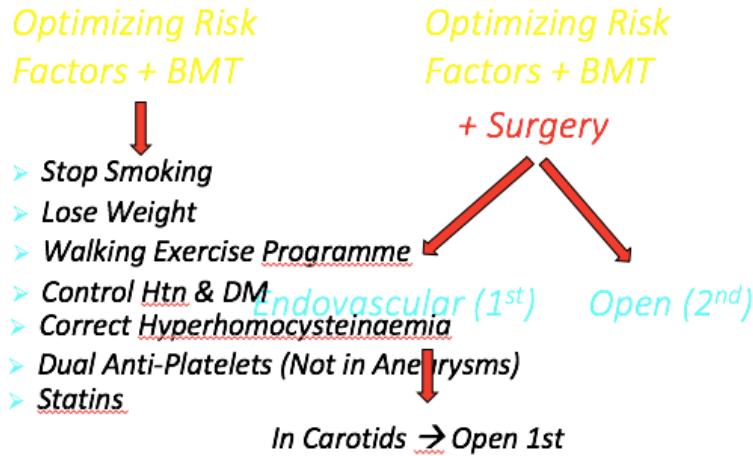
#### For surveillance

- Aneurysms measuring <4.0 cm should be rechecked by U/S every year to monitor for potential enlargement and dilation
- 4.0-4.5 cm every 6 months
- greater 4.5 cm should be evaluated by surgeon for potential repair
- 3. Investigation of the other Vascular Beds
  - a. ECG
  - b. ECHO
  - c. ABI + Toe Pressures
  - d. Duplex scan

## Surgical decision making

- Risk of rupture
- Risk of surgery
- Symptomatic aneurysm
- Ruptured aneurysm

## Management

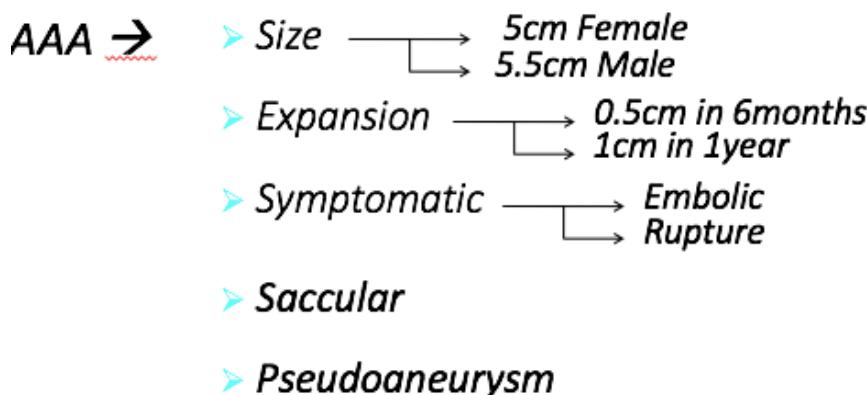


### Management

- Surveillance
  - BMT
    - Smoking cessation
    - BP control → β Blockers
    - Statin
  - Duplex at safe intervals
    - < 4cm → annually
    - > 4cm → 6 monthly

1. Optimize Risk factors-once aneurysms detected, goal is to prevent it from enlarging. Life-long control of risk factors is a must and include the following:
  - a. Lifestyle-quit smoking, reduce obesity, increased exercise, good glycaemic control
  - b. Pharmacology- antihypertensive, diabetic control, vitamin B complex (for homocysteine)
2. Optimal medical therapy- Aspirin (dual antiplatelets not required unless other indication) and Statin
3. Surgery- Open/ Endovascular

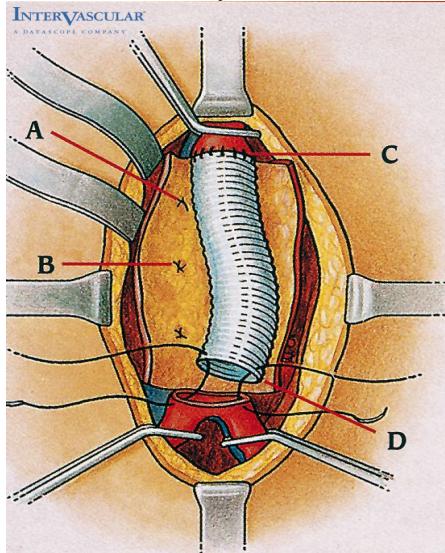
### Indications for surgery



the principle of repair in both open and EVAR is to produce a sealed conduit for blood to pass through the affected segment of the aorta without leaking into the aneurysm sac

#### management of rupture

- Successful emergency repair depends on intervention during the “Window of Opportunity”  
... Active bleeding is temporarily arrested by hypotension & tamponade of the hematoma by the posterior parietal peritoneum
- Minimal resuscitation (Permissive Hypotension)
  - Systolic BP 60 – 80mmHg accepted



#### open repair

- Midline laparotomy and dissect down to aorta at back
- Aortic and iliac clamping is done before the aorta is opened and any thrombus is removed
- Section of aorta that is dilated is removed and replaced with inlay graft or Y graft that is sutured into place
- Materials of graft: Dacron or polytetrafluoroethylene (PTFE)
- Done under general anesthesia, takes 4-6 hours. After surgery to ICU
  - Urinary catheter-to drain the bladder
  - Central venous catheter- to monitor pressures in the heart
  - Arterial catheter- to monitor BP
  - Epidural catheter- to give pain medicine
  - NG tube- used to keep stomach empty
- Go home 4-7 days after surgery, back on normal activities after 4 weeks

#### Complications

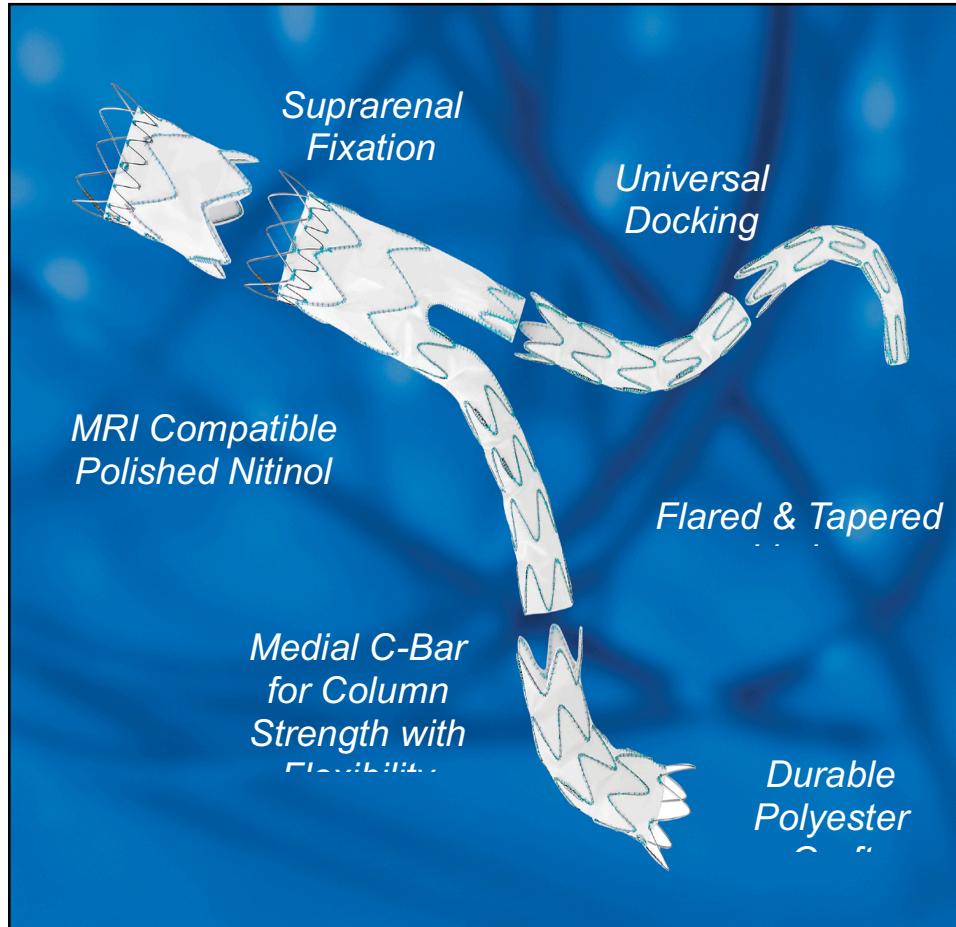
- Sudden increase in afterload
  - Clamping
- Sudden hypotension
  - Declamp
  - Reduced preload-Venous sequestration
  - Reperfusion injury
- Haemorrhage
  - Suture line
  - Venous
- DIC
- Hypothermia
- Distal embolisation
- Paraplegia
- Impaired sexual function
- DVT
- Stroke

- Renal function
- GI complication
  - Ileus
  - Sigmoid ischaemia

#### Late complications

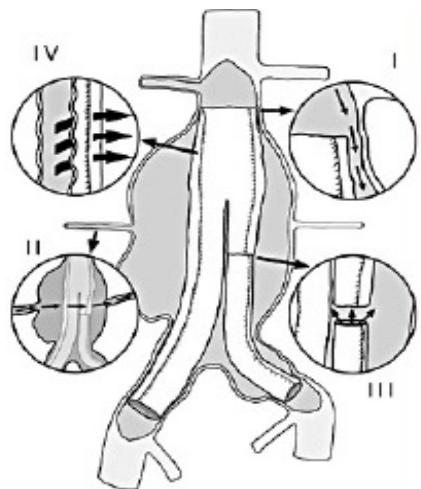
- Aortic anastomotic disruption / Pseudoaneurysm
- Graft infection / AE fistula
- Thrombosis
- Secondary aneurysm

#### EVAR



#### complications

- Endoleak; improper seal, communication into arteries eg IMA lumbars into intercostals, device failure, porosity of graft-reaction to device (rare)
  - Type I (Graft attachment site leaks)
    - Type Ia: proximal end of endograft
    - Type Ib: distal end of endograft
  - Type II (Branch leaks)
  - Type III (Graft defect)
    - Type IIIa: Junctional leak or modular disconnection
    - Type IIIb: Fabric disruption
  - Type IV (Graft wall porosity)



- Graft Migration
  - Proximally
  - Distally
- Graft Infection
- Access site complications
  - Pseudo-aneurysm
  - Hematoma

Anatomical requirements for EVAR

- Infra-Renal
- Proximal landing zone (Infra-renal neck)
  - 15 – 20mm
  - <60° angulation
- Distal landing zone (Iliacs)
  - Adequate length
  - Non-aneurysmal
  - Minimal tortuosity
  - Patent

## Endo Vs Open

<i>Endovascular</i>	<i>Aorta</i>	<i>Open</i>
↓		↓
➤ Age over 65	➤ Age under 65	
➤ Infra-Renal	➤ Regardless of Anatomy	
➤ Adequate Neck		
➤ 1.5cm		
➤ Angulation <60°		
➤ Patent Iliacs with minimal Tortuosity		

### Iliac aneurysms

- Risk of rupture has not been accurately quantified
- Symptomatic & ruptured aneurysms mandate immediate intervention
- Commonly accepted guidance → Elective repair for asymptomatic Iliac aneurysms >3cm

### Common femoral aneurysms

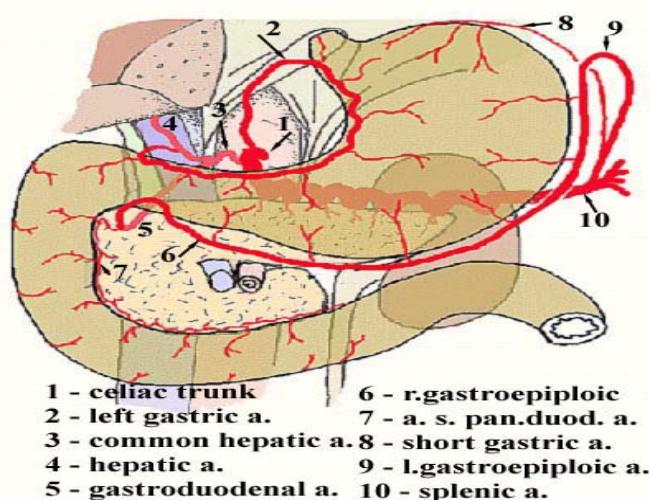
- Surgical treatment indicated at 3 cm or more

### Popliteal artery aneurysms

- Indications for surgery include
  - High thrombus load
  - Distal embolisation
  - Otherwise 2cm or larger

## MESENTERIC ISCHEMIA

- Defined as occlusive/ non occlusive mechanisms leading to hypoperfusion of one or more mesenteric vessels and ischaemic colitis. Most of blood supply of bowel goes to the mucosa so when impaired it quickly leads to ischaemia
- Meal → digestion requires oxygen → severe intestinal ischaemia will cause induction of anaerobic metabolism → production of metabolites that cause pain in a similar manner to other vascular beds (results in acidosis, increased amylase, leukocytosis)
- Acute
- Chronic



What happens after a meal. Increase blood flow. CA flow barely changes. Major changes occur in SMA. Mucosal blood flow 75% though it comprise 50% of small bowel mass.

Digestion requires oxygen which is simply not there. Anaerobic metabolism.

Mucosal barrier.

Reperfusion injury.

### Chronic ischaemia

- Intestinal/ mesenteric angina
- Median arcuate ligament compression syndrome-due to hypertrophy of crus muscle- coeliac compressed by crus of diaphragm

### Pathophysiology

#### Intestinal angina

- Following a meal, digestion requires increased oxygen consumption by the gut. In the presence of severe intestinal ischemia, significant induction of anaerobic metabolism will result in the production of metabolites that cause pain in a similar manner to other vascular beds

### Diagnosis

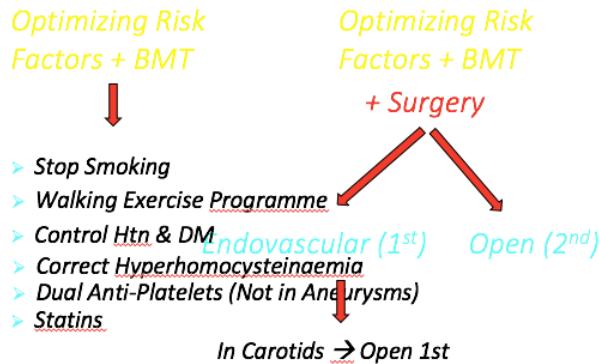
#### Non-invasive test

- PFA; to rule out perforated viscus, may have dilated loops and pneumatosis intestinalis (gas cysts and oedema in mucosa) and thumb printing (thickened wall) which is highly suggestive for necrotizing enterocolitis
- Duplex-noninvasive but impaired by increase in intestinal gas or adiposity and is operator dependent so not gold standard
- CT
- MRA

## Indications for surgery

### Chronic Mesenteric ischaemia- loss of weight

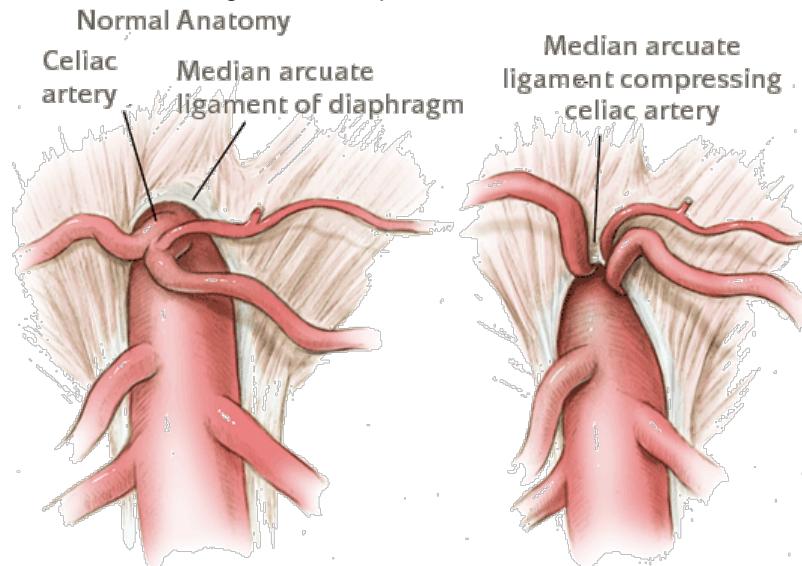
#### Management



#### Open surgery

- Bypass vs resection & re-implantation

#### Median arcuate ligament compression



- Division of tough crural fibres
- Arterial reconstruction

#### Acute mesenteric ischaemia

- Occlusive or non-occlusive mechanism leading to hypoperfusion of one or more mesenteric vessels

#### Pathophysiology

- Following a meal, digestion requires increased oxygen consumption by the gut. In the presence of severe intestinal ischemia, significant induction of anaerobic metabolism will result in the production of metabolites that cause pain in a similar manner to other vascular beds
- Prolonged hypoxia with persistent severe pain out of proportion to the clinical findings (No peritonism)
- Early development of;
  - Acidosis
  - Hyperamylasaemia
  - Leocytosis
- Ischemic damage to the mucosa
  - Translocation of bacteria, endotoxins and cytokines
  - Major systemic effects

- Septicaemia
- Multi-organ failure

#### Aetiology

- Arterial embolic disease
- Arterial thrombotic disease
- Venous thrombotic disease
- Non-occlusive mesenteric ischaemia

#### Arterial Embolism

- >50% cases : SMA occlusion
- Origin of middle colic artery
- Cardiac(80%) & aortic plaque
- Coeliac and IMA –usually tolerated
- SMA occlusion- Death
- SMA is more feared
- Normally due to AF or mural thrombus following MI

#### Thrombotic disease

- 15%
- Preexisting disease
- Ostium
- Delayed onset of symptoms
- Often pt. will describe a hx of chronic ischaemia & have developed some degree of collaterals which protect the bowel for a short time before critical ischaemia develops.
- These lesions are found at the ostium of these vessels.

#### Venous Thrombosis

- 5-10 %
- Younger
- 80% hypercoaguable disorder (factor V leiden, protein C or protein S deficiency, antithrombin III, anticardiolipin antibodies)
- DVT/PE, OCP, Malignancy, portal hypertension,
- May limit arterial flow-Oedema, infarction

#### Non-occlusive

- Severe illness
- CCF, Sepsis, MODS
- Abdominal pain 75%
- High index suspicion
- Angiography
- Papaverin- delivered to SMA by catheter
- Glucagon
- Mortality 70-80%

Note: Can be very easy and very difficult to diagnose. The pt. present with systemic hypoperfusion and visceral vasoconstriction for cerebral protection leading to bowel ischaemia. It is mediated by sympathetic nervous system and can be found in any critical setting leading to hypotension.

A no. of drugs have also been implicated.

Clinical presentation of acute mesenteric ischaemia-commonly seen in elderly patient. Need high level of suspicion if patient looks unwell with severe pain

- Patient ill out of proportion of physical signs- distended tender abdomen and bowel signs may be normal/ absent
- Variable symptoms
- Non specific pain
- Vomiting - & bloody diarrhea; haemochezia
- Hypotension
- Tachycardia-metabolic acidosis
- Peritonitis- late sign if ischaemia leads to perforation

Diagnosis

## Laboratory

- WBC >20 000 is common
- U&E
- ABG-may show metabolic acidosis
- Lactate-often a late sign if raised
- Amylase raised
- Phosphatase, D-dimers may raise as well

## Non-invasive tests

- PFA
- CT
- MRA

## Investigations



pneumatosis intestinalis



dilated bowel

plain films can occasionally show pneumatosis, portal gas, and thumb printing consisting with ischaemia. Main utility of the plain film is to outrule perforate viscus

computed tomography- most important tool to diagnose this condition.

- Pneumatosis
- Venous gas
- Bowel wall thickening
- wall enhancement
- Solid organ infarction
- Arterial Occlusion
- Venous thrombosis

## Radiology

### MRI

- Mostly good to see proximal disease- proximal SMA can detect stenosis between 70-100%
- Poor delineation of the small vessels

- Availability
- Its operator dependent, bowel gas can obscure the arteries

#### Principles of treatment

- Diagnose
- Restore flow
- Resect Non-viable tissue
- Supportive care
- Second look

#### Therapy

##### Supportive measures

- IV resuscitation-crystalloid and correction of electrolyte imbalance
- Optimize cardiac status-insert invasive monitoring devices (CVP; Art Line; urinary catheter with hourly urine output)
- Broad spectrum antibiotics (amoxycillin, gentamicin, metronidazole) until cultures come back
- NG decompression

#### Pharmacological therapy

##### Anticoagulation

- IV Heparin- 5000 IU as a bolus and continuous infusion to maintain APTT >2 to prevent propagation of thrombus. Mainstay Rx for veno-occlusive disease and NOMI-often not enough time to work in acute risk situation and risk of distal embolisation
- Warfarin

##### Vasodilators

- Papaverine

##### Thrombolysis

#### Open surgery

- Embolectomy Vs Bypass Vs Resection & Re-implantation

##### Non Viable bowel must be resected

- Second look

##### Thrombolysis

#### Appearance of frankly ischaemic bowel (use Doppler if unsure)

- Grey, foul smelling
- Loss of peristalsis
- Duskeness
- Loss of bowel sheen
- Loss of pulsation in the mesenteric arcade
- Lack of bleeding

#### Surgical options: for all unstable, peritonitis, evidence of perforation

- Revascularization- if bowel appears salvageable Embolectomy or bypass may be considered especially in Arterial thromboembolism with no infarction
- Resection and re-implantation-non-viable bowel must be resected and viable bowel brought out as a stoma to allow monitoring of mucosal perfusion, a temporary abdominal closure is recommended to allow a second look at 24-48 hours to ensure all dead tissue removed
- Palliation for elderly and bowel is non-viable. Often the whole small bowel is affected with sparing of proximal jejunum, likely to render the patient dependent on TPN or needing small bowel transplant. Minimum of 70 cm of small bowel is required for absorption to maintain life
- Division of tough crural fibers laparoscopically or mini-laparotomy-in median arcuate ligament compression

## Management

