Bowel Infarction

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Anatomical Pathology Discipline
2.28. Arterial supply of the GI tract.

- Aorta
- Esophagus
- Left inferior phrenic artery
- Celiac trunk (artery to foregut)
- Left gastric artery
- Spleen
- Stomach
- Superior mesenteric artery (to midgut)
- Duodenum
- Small intestine
- Inferior mesenteric artery (to hindgut)
- Descending colon
- Ascending colon
Ischaemic Bowel Diseases

• Ischaemic bowel diseases involvement of small & large intestine
• May be restricted to small or large intestine or may affect both depending on blood vessel affected
• Blood supply of intestines: celiac, superior & inferior mesenteric arteries.
  – Acute occlusion may lead to infarction of several meters of intestine
Ischaemic Bowel Disease

• Insidious loss of one vessel may be without effect owing to rich anastomotic interconnections

• Lesions within end arteries which penetrate gut wall produce small focal ischaemic lesions

• Severity of injuries range from:
  – Transmural infarction – involves all visceral layers
  – Mural infarction – mucosa and submucosa
  – Mucosal infarction – no deeper than muscularis mucosa
Ischaemic Bowel Disease

- Transmural infarction: almost always implies mechanical compromise of major mesenteric blood vessels
- Mucosal or mural infarction: results from hypoperfusion either acute or chronic
- Mesenteric venous thrombosis is a less frequent cause of vascular compromise
Predisposing Factors

- Arterial thrombosis
- Arterial embolism
- Venous thrombosis
- Non-occlusive ischaemia
- Miscellaneous causes
Arterial Thrombosis: Causes

- Severe atherosclerosis (at origin of mesenteric vessels)
- Systemic vasculitis: e.g. polyarteritis nodosa
- Dissecting aneurysm
- Angiographic procedures
- Aortic reconstructive surgeries
- Surgical accidents
- Hypercoagulable states
- Oral contraceptives – produce hypercoagulable state
Arterial Embolism: Causes

- Cardiac vegetations
- Angiographic procedures
- Aortic atheoembolism
Venous Thrombosis: Causes

- Hypercoagulable states
- Oral contraceptive use
- Antithrombin III deficiency
- Intraperitoneal sepsis
- Postoperative state – hypercoagulable state
- Invasive neoplasms (hepatocellular carcinoma)
- Cirrhosis
- Abdominal trauma
Non-occlusive Ischaemia: Causes

- Cardiac failure
- Hypovolumic shock
- Dehydration
- Vasoconstrictive drugs: digitalis, vasopressin, propranolol
Miscellaneous causes

- Radiation injury
- Volvulus stricture
- Internal or external herniation
Ischaemic Bowel Disease

- Embolic arterial occlusion most often involves branches of superior mesenteric artery
- Inferior mesenteric artery spared and thought to be due the course of the artery where it is oblique at its origin.
- Despite many known causes large percentage no known cause can be definitively identified
Clinical Presentation

• Bowel infarction is an uncommon disorder but with rate of 50-70% death rate

• Due to time of onset of symptoms and perforation is small

• Tends to occur in older pts – this population has high incidence of cardiac and vascular diseases
Clinical Presentation: **Transmural Infarction**

- Severe abdominal pain – sudden onset
- Associated nausea, vomiting and bloody diarrhoea or gross melanotic stool maybe present
- Pts may progress to shock & vascular collapse can occur very quickly within hours
- Diminished peristaltic sounds or none audible
- Abdominal muscle spasm creates board-like rigidity of abdominal musculature
Clinical Presentation: **Mucosal & Mural Infarction**

- Not usually fatal if cause of vascular compromise corrected
- Non-specific abdominal symptoms combined with intermittent bloody diarrhoea
- If not recognised quickly may progress to extensive infarction and sepsis
- Chronic ischaemic colitis may present as inflammatory disease: intermittent episodes bloody diarrhoea with periods of healing
Levels of infarction

• Transmural infarction involving all layers of the gut.
• Mural infarction – mucosa & submucosa
• Mucosal infarction – mucosa

Mural-Of Wall
Transmural Infarction

- Short segment or large portion of intestine can be affected
- Highest risk part is the splenic flexure (watershed between distribution of superior & inferior mesenteric arteries)
- Mesenteric venous occlusion: anterograde & retrograde propagation of thrombosis may lead to extensive involvement of splanchnic bed.
Morphology: Transmural

• Infarcted intestines appear haemorrhagic regardless of whether arterial or venous occlusion

• Early:
  – congestion and dusky to purple-red color
  – Small & large foci of subserosal & submucosal echymotic discoloration
Morphology: Transmural

• With progression:
  – Intestinal wall becomes edematous, thickened, rubbery and haemorrhagic.
  – Lumen will contain blood or mucus

• **Arterial occlusions**: demarcation between normal & infarcted tissue well defined

• **Venous occlusions**: area of dusky edema fades gradually into normal adjacent normal tissue
  – No clear demarcation between viable and non-viable bowel
Transmural infarction caused by a volvulus

Ref: www.studyblue.com via Google Images
Morphology: Transmural

• Histologically:
  – Obvious edema
  – Interstitial haemorrhage
  – Sloughing necrosis of mucosa
  – 1-4 day old infarcted bowel gangrene and perforation can occur.
  – Little to no inflammatory response visible
Hemorrhagic transmural haemorrhagic infarction
Morphology: mucosal & mural

- Lesions maybe multifocal or continuous and widely distributed
- Affected bowel appear dark red or purple (due to accumulated luminal haemorrhage)
- No haemorrhage or inflammatory exudate on serosal surface
- Open bowel will show haemorrhagic edematous thickening of mucosa
- Superficial ulceration maybe present
Morphology: mural & mucosal

• Histology:
  – **Mild form of ischaemic injury:** superficial epithelium of colon & tips of small intestinal villi will be necrotic or sloughed.
  – Inflammation absent
  – Mild vascular dilation
  – **Complete sloughing seen if complete mucosal necrosis. Only acellular lamina propria will be visible**
  – **Severe form:** extensive haemorrhage & necrosis of multiple layers
  – Secondary acute & chronic inflammation evident along margin of viable bowel adjacent to affected area
Morphology: mucosal & transmural

- Bacterial superinfection and formation of enterotoxic bacterial products may induce formation of pseudomembrane inflammation
- This particularly affects colon and can mimic enterocolitis of nonvascular origin
Mural infarction showing early necrosis and hyperemia in mucosa & submucosa

Ref: dastyari.parsmedic.com via Google Images
Morphology: chronic ischaemia

- Mucosal ulceration & inflammation may develop
- This mimic both acute entercolitis (from other causes) & idiopathic inflammatory bowel disease
- Stricture maybe seen due to submucosal chronic inflammation & fibrosis
- Colonic stricture typically occur in the watershed area of splenic flexure
- But acute & chronic mucosal ischaemia are commonly segmental & patchy (microscopically & macroscopically)
Chronic non-occlusive mesenteric ischemia

Ref: gastrointestinalatlas.com via Google Images
Differential Diagnosis

Appendicitis
Trauma
Pseudomembranous colitis  Adenocarcinoma
Diverticulitis
Crohn Disease
Necrotizing Enterocolitis
Pneumatosis Intestinalis
Typhlitis
Ulcerative Colitis
Complications:

• Bowel necrosis (requiring bowel resection)
• Septic shock
• Death

• Patients in whom the diagnosis is missed until infarction occurs have a mortality rate of 90%. Even with good treatment, up to 50-80% of patients die.
• Survivors of extensive bowel resection face lifelong disability.
Laboratory Diagnosis

- Abdominal x-ray
- Enema
- Angiogram
- USS/CT/MRI
- Bloods – FBC/UEC/Coagulation studies/Lipase/Amylase
Treatment:

• NPO : prepare for surgery and to reduce oxygen demand on the ischemic bowel

• surgery

• Interventional radiology: angiographic drug infusions or angioplasty.
Treatment:

• **acute occlusive mesenteric ischemia**: usually surgical resection of the infarcted bowel segment.

• **Chronic mesenteric ischemia**: not a surgical emergency and may be treated conservatively.

• **Nonocclusive mesenteric ischemia**: usually nonsurgically. Depending on the cause