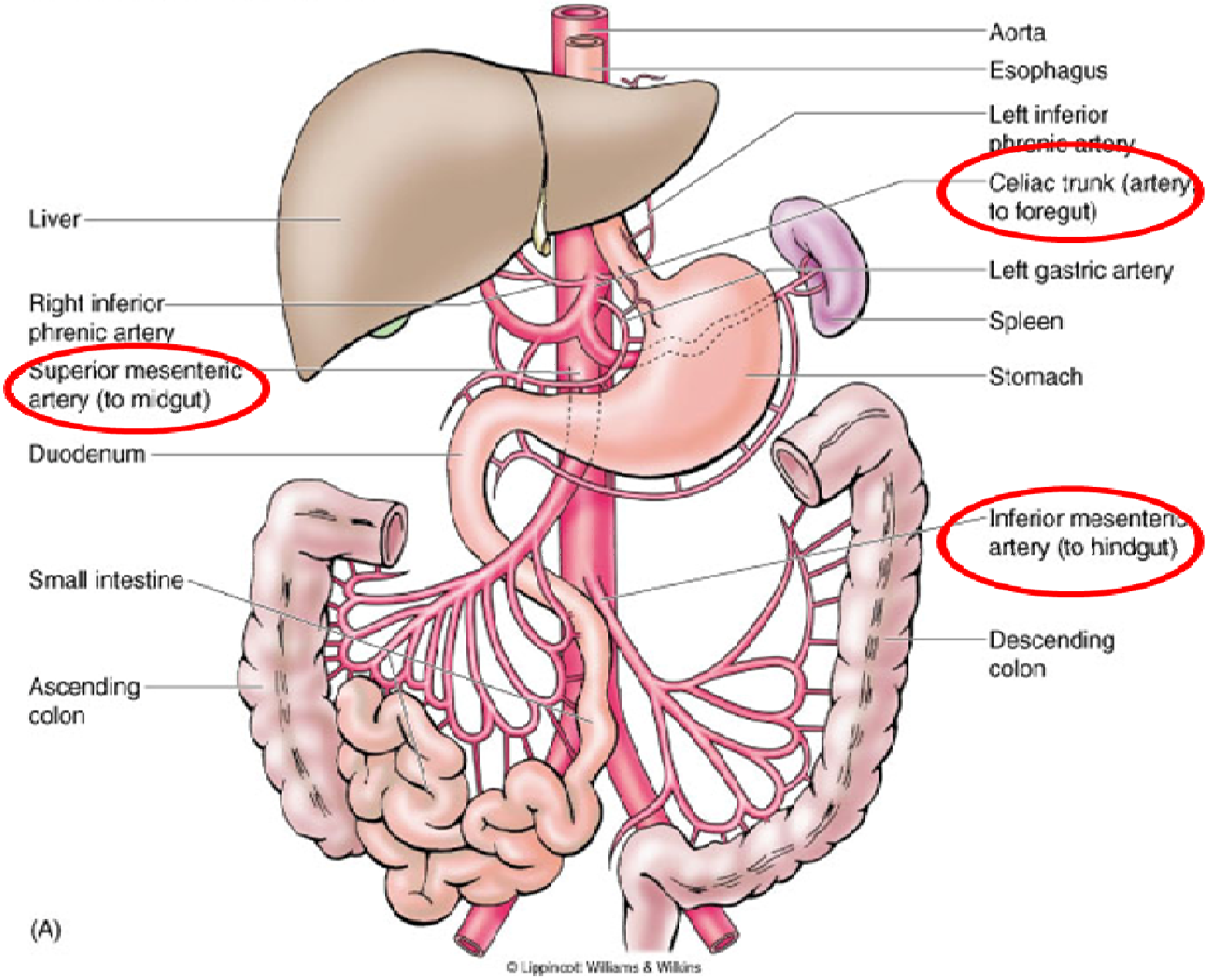
Bowel Infarction

Dr Rodney Itaki

Anatomical Pathology Discipline

2.28. Arterial supply of the GI tract.



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

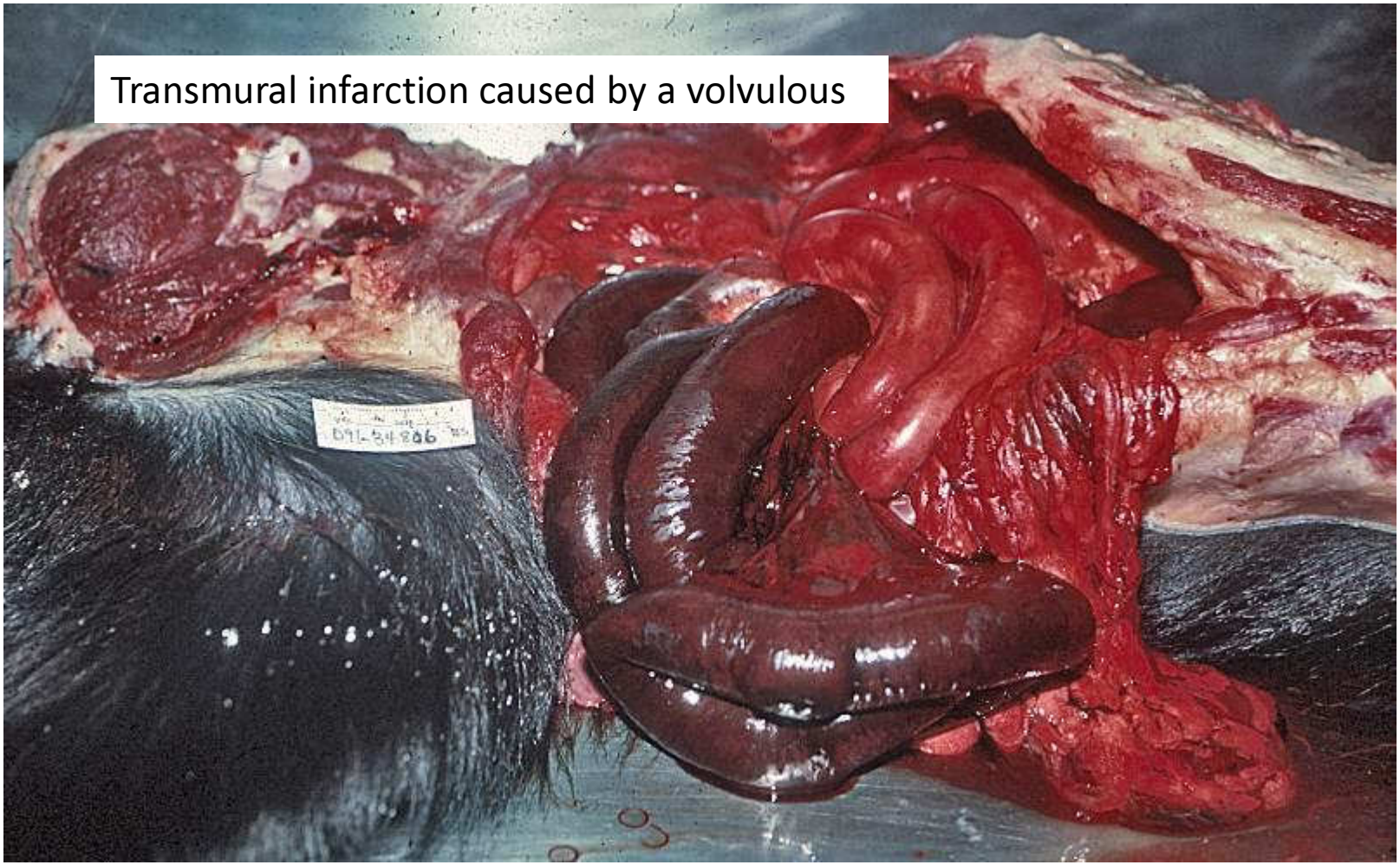
- Short segment or large portion of intestine can be affected
- Highest risk part is the spleenic flexure (watershed between distribution of superior & inferior mesenteric arteries)
- Mesenteric venous occlusion: anterograde & retrograde propagation of thrombosis may lead to extensive involvement of 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 subseroal & submucosal echymotic discoloration

Morphology: Transmural

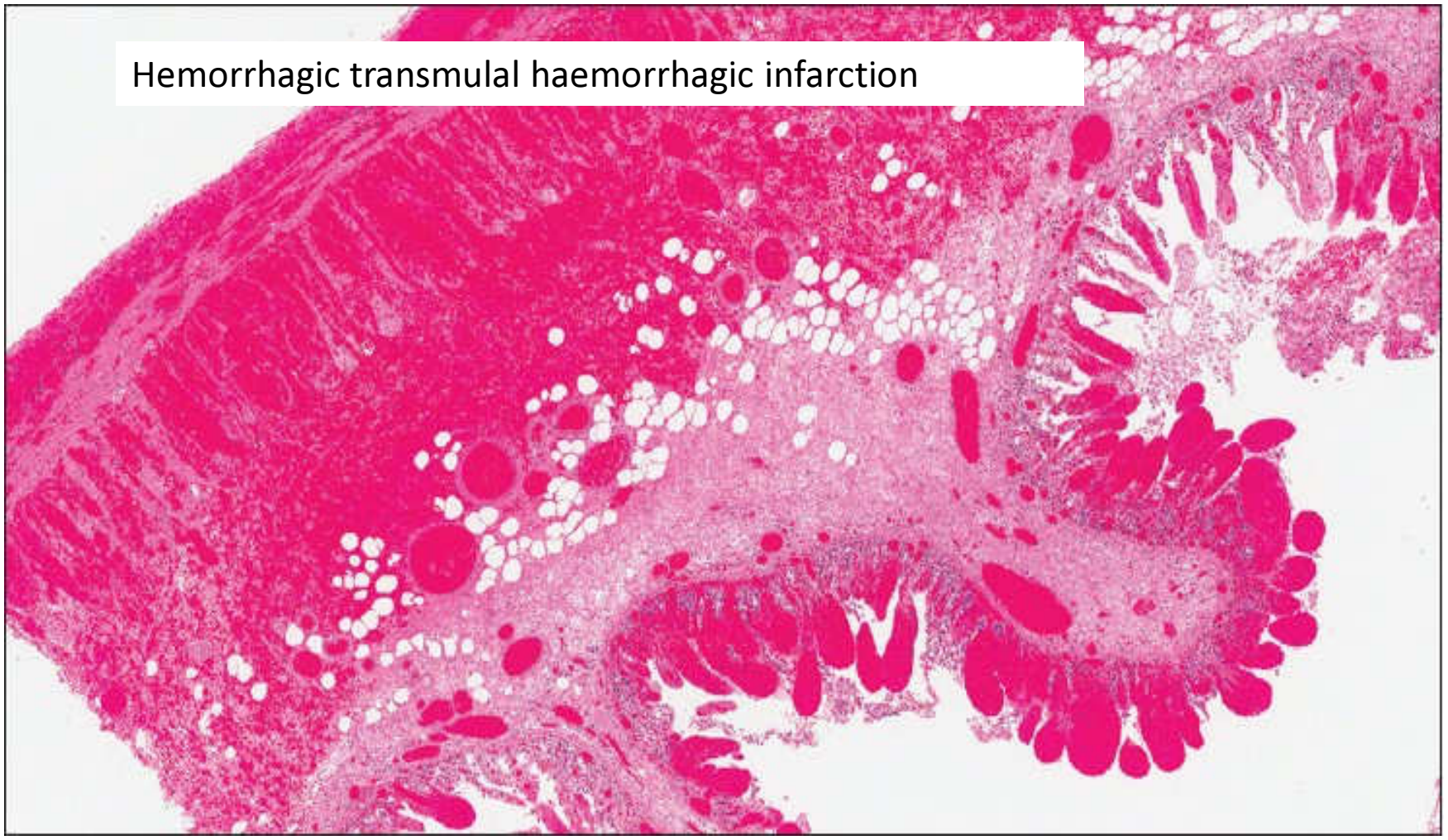
- With progression:
  - Intestinal wall becomes edematous, thickened, rubbery and haemorrhagic.
  - Lumen will contain blood or mucus
- Arterial occlusions: demarcation between normal & infarcted tissue well defined
- Venous occlusions: area of dusky edema fades gradually into normal adjacent normal tissue
  - No clear demarcation between viable and non-viable bowel



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



Ref: jahjournal.org via Google Images

Morphology: mucosal & mural

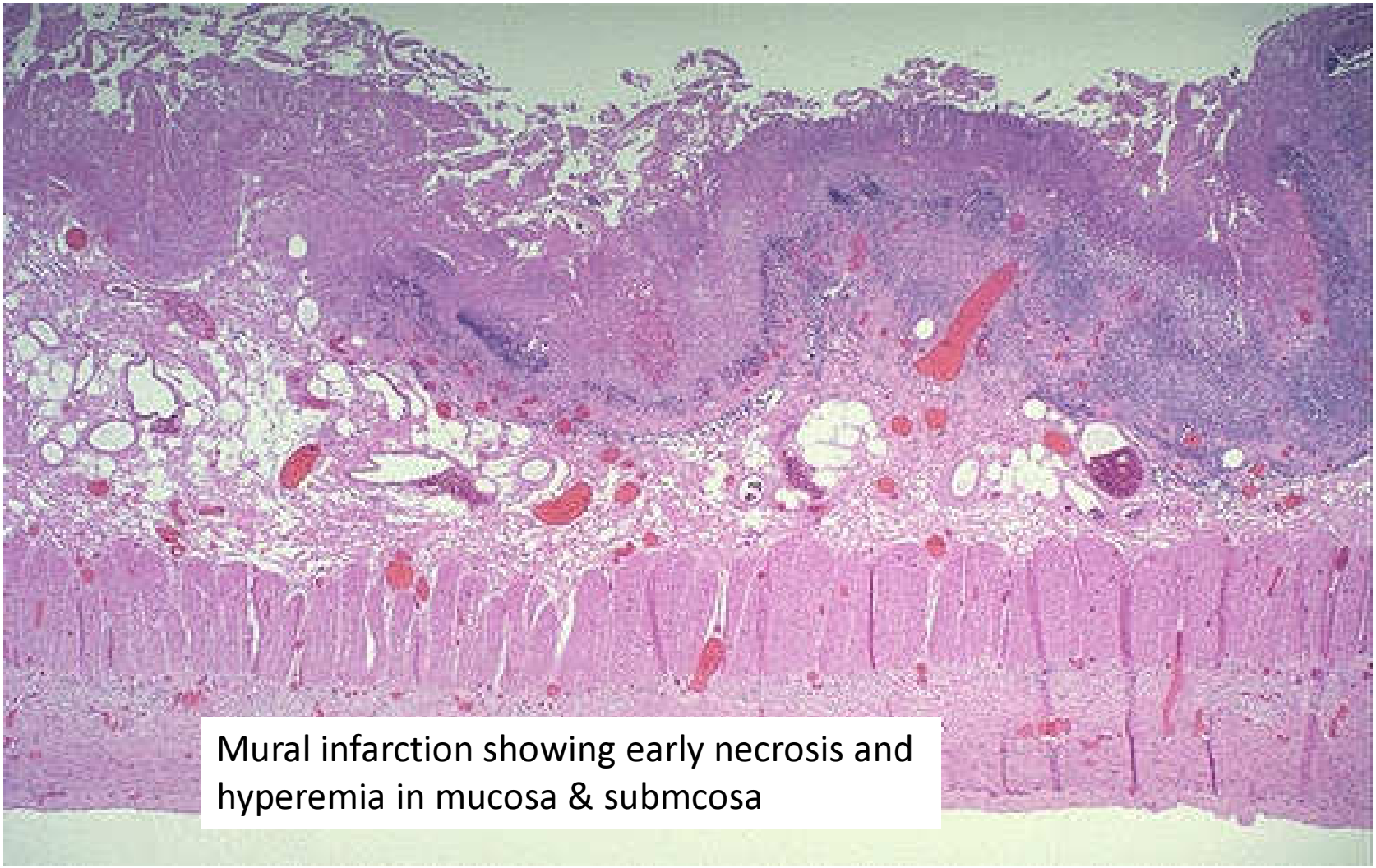
- Lesions maybe multifocal or continuous and widely distributed
- Affected bowel appear dark red or purple (due to accumulated luminal haemorrhage)
- No haemorrhage or inflammatory exudate on serosal surface
- Open bowel will show haemorrhagic edematous thickening of mucosa
- Superficial ulceration maybe 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 superfinfection and formation of enterotoxic bacterial products may induce formation of pseudomembrane inflammation
- This particularly affects colon and can mimic enterocolitis of nonvascular origin

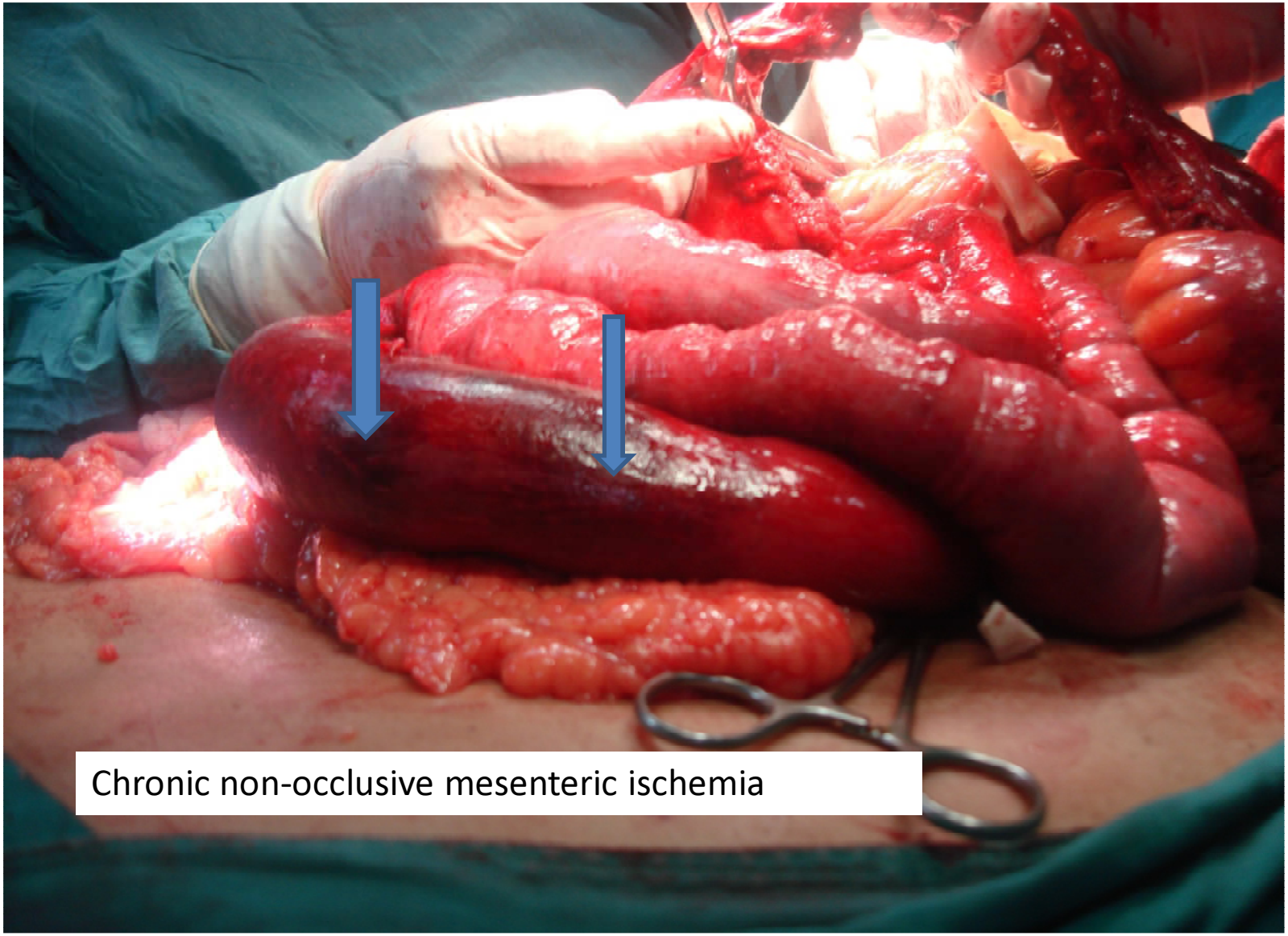


Mural infarction showing early necrosis and hyperemia in mucosa & submcosa

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)



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

END

Reference: Robins Pathological Basis of Diseases

www.patholgyatsmhs.wordpress.com