Peptic Ulcer Disease

- chronic mucosal ulceration that penetrate muscularis mucosae (not propria) and is seen in stomach or duodenum.
- associated with
  - H pylori
  - NSAIDs
  - smoking

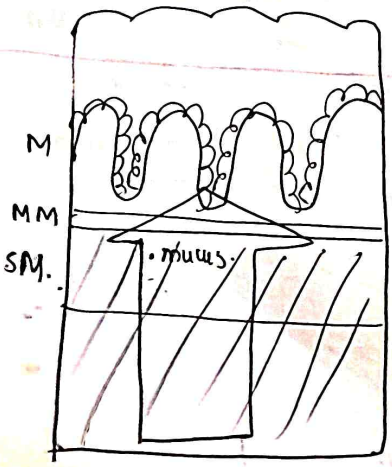
site.
- duodenum → most common site. → 1st 2.5cm.
- Gastric → lesion curvature.

Pathogenesis

FIGURE

- Imbalance between defense factors & damaging factors.

Look at screenshot



Risk factors

1. Helicobacter pylori injection ✓
2. cigarette use ✓
3. NSAIDs ✓
4. Zollinger-Ellison syndrome ✓
5. COPD.
6. alcoholic cirrhosis.
7. psychological stress
8. viral infection?
   CMV., herpes simplex virus

Damaging forces

Just for reading

Normally
- HCl → damages gastric mucosa.
- pepsinogen → pH → Pepsin.

Defensive forces

1. pre-epithelial
2. epithelial
3. Subepithelial elements.

1. pre epithelial → Surface mucus secretion.
   - form unstirred protective layer
   - → Bicarbonates → buffer hydrogen ions entering from luminal aspect

2. Epithelial
   - Restitution → restoration: need blood flow by.
   - epithelial regeneration → PGs, TGFα
   - PGs → maintain mucosal blood flow and epithelial restitution

Pathogenesis

- Increased damaging factors

① H. pylori infection.
{flowchart}

② NSAIDS – ① direct chemical irritation of mucosa
② Supress PGs synthesis
③ ↓ HCO_3^- secretion.

③ Cigarette smoking – Impairs blood flow to mucosa.

④ Alcohol, radiation, chemotherapy → direct injury to mucosal cells

⑤ Ingestion of chemicals – acids, bases.

⑥ Gastric hyperacidity – parietal cell hyperplasia & ZES.

⑦ High dose corticosteroids → ⊖ PGs synthesis.

Impaired defense

- Ischemia : ↓ O_2 delivery
- shock.
- delayed gastric emptying
- Host factor: reduced mucin synthesis in elderly leads to increased susceptibility to gastritis

Clinical feature

- epigastric burning pain
1-3 hours after meals

Complications:

- Bleeding → duodenal ulcer
- malignant transformation
- Iron deficiency anemic.
- Perforation → gastric.

Morphology

- duodenum (more common) at 2.5 cm
- Stomach – lesser curvature near junction of body & antrum.
- gastroesophageal junction of esophagus
- Jejunum – ZES (multiple).
stomach
duodenum.
- heterotropic gastric mucosa in meckel's diverticulum.

Shape: Round / oval.
✓ punched out borders.

✓ base : smooth & clean
- mucosal folds radiating from ulcer.

Microscopy

Four layers : Askanazy zones.

① Necrotic zone : superficial zone – purulent exudate, bacteria & necrotic debris

② Superficial exudative zone – fibrinopurulent exudate with predominantly neutrophilic myeloid cells

③ Granulation tissue zone.

④ zone of cicatrization – contains fibrous tissue or collagenous scar

Figure SG

Q) Barret Esophagitis, Histology of Barrets.

- Intestinal metaplasia within the squamous mucosa of esophagus.

ie,

- replacement of normal stratified squamous epithelium of esophagus with columnar epithelium
- dysplasia, increased risk of Esophageal adenocarcinoma.

CAUSE.

- long standing GERD.

MORPHOLOGY

GROSS tongue like, lesion patches of red-velvety mucosa extending from gastroesophageal junction upto esophagus.
Salmon flesh appearance.

MC.

- Intestinal metaplasia.
  squamous replaced by columnar wine goblet
- Goblet cells with acidic mucin vacuoles – diagnostic.
  stain → ALCIAN BLUE
- dysplasia can occur.

Treatment {reduce acid secretion effect}
✓ Proton pump inhibitors ✓ Antacids.
✓ H2 receptor antagonist

Q) Amoebic Ulcer.

Amoebic dysentery.
↓
due to infection by Entamoeba histolytica.
↓
infection occurs by ingestion of cyst form
↓
cyst walls dissolved in small intestine.
↓
liberated amoeba pass into large intestine.
↓
invade epithelium of mucosa
↓
reach submucosa. {not muscular layer}
↓
produce characteristic flask shaped ulcers

GROSS:

→ early intestinal lesion appears as
↓
small areas of elevation on mucosal surface
→ advanced stages.
↓
typical flask shaped ulcers having narrow neck and broad base are seen.

- most common in caecum, rectum, flexures

MC.

ulcerated area shows chronic inflammatory reaction consisting of.

↓

lymphocytes, plasma cells, macrophages, eosinophils.

- trophozoites are seen in inflammatory exudate and are concentrated at advancing margin of lesion.
- due to ingestion of red cells. they appear as red coloured dots. {trophozoite}.

Ameboma

- rare complication of amoebiasis.
- Intestinal wall show thickening due to inflammation — napkin ring constriction.
- and may resemble — Colon cancer

MC

- granulation tissue
- Inflammatory cells
- fibrosis
- trophozoite clusters.

Amebic liver abscess

- major complication of Intestinal Amoebiasis.
- trophozoite from colon

↓

portal circulation

↓

Liver

↓

To. cell hepatocytes.

↓

produce abscess

↓

Cavity containing dark brown, odourless, semisolid necrotic material.

↓

resembling Anchovy sauce in colour & consistency.

Q) Achalasia Cardia. (UQ)

- It is a neuromuscular dysfunction.
- characterized by triad of

→ Incomplete LES relaxation

→ Increased LES tone

→ oesophageal aperistalsis.

Etiology & pathogenesis

→ Occurs due to loss of. Inhibitory neuron, that release NO & VIP.

→ failure to relax ✓

Etiology

① primary → Idiopathic

② Secondary – * Chaga's disease.
T. cruzi.

* Viral inf like Herpes.

* Diabetes → neurodegeneration disease.

CF

dysphagea to liquids >> solids.

Diagnosis

① Barium study – Bird beak / rat tail

② Esophageal manometry.

Q)