Peptic Ulcer Disease (PUD)

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Definition

Gastric & duodenal ulcers. Dyspepsia ["indigestion" - discomfort or heartburn or nausea] is common (40% population annually). But only 1% (rising) have an endoscopy - 40% of these show non-ulcer dyspepsia, 40% have GORD, ~15% have PUD, and <5% have oesophageal or gastric Ca.

Aetiology

- Helicobacter pylori [duodenal (90%) & gastric (70%) ulcers]
- NSAID
- Other: EtOH, steroids, stress, smoking, Zollinger-Ellison, family history

Epidemiology

Incidence: Duodenal ulcer > gastric ulcer. Decreasing with H. pylori eradication. Sex: 3M:2F

Prevalence: 0.1-0.15%

Presentation

Symptoms: Epigastric pain (usually 1-3hrs post-prandial, maybe relieved by food) \pm heartburn, nausea, aerophagy (\rightarrow burping, bloating, distension) & intolerance of fatty food (DDx gallstones). A posterior ulcer may \rightarrow pain radiating to back esp if penetrating. Antacids may have helped. Patients may present with acute life-threatening Cx: haemorrhage or perforation. Signs: Often epigastric tenderness. If gastric emptying slow, there may be a succussion splash.

Differential diagnosis

- Gastro-oesophageal reflux
- Gastric carcinoma
- Gallstones
- Pancreatitis
- Crohn's disease
- Irritable bowel syndrome
- Drug-induced dyspepsia
- Hepatitis
- ACS

Investigations

Bloods: FBC (occ iron-def anaemia), amylase/Lipase, LFT, ±G&H, ±Cardiac markers if IHD RF.

ECG: DDx of ACS

Radiology: CXR/AXR - perforation.

Helicobacter pylori detection:

- Serology ELISA (85% sens, 80% spec) may be +ve post-eradication.
- Urease detection breath/blood/endoscopic(CLOtest) tests
 - o Bacterial urease cleaves ¹³C- or ¹⁴C-labelled urea \rightarrow NH₄ & HCO₃⁻ \rightarrow exhaled labelled CO₂ \rightarrow active H.pylori (Sens>90%). False negs: bismuth, PPI, ABx.
- Faecal antigen test
- Endoscopic biopsy
- NB. if H.pylori not detected & not on NSAIDs → low likelihood PUD

Endoscopy: (Largely has replaced barium studies)

• Advantages vs Barium: Direct vision of ulcer, gastritis, oesophagitis, biopsy, H.pylori detection, LXR, and can treat bleeding ulcers with injection, ligation, heat, laser.

Management

Modification of behaviour:

- ↓EtOH, ↓smoking, ↓stress. Possibly ↓coffee (unproven),
- Drugs Cease, change or comply more fully with recommendations for possible culprits.
- E.g. taking NSAIDs including aspirin after food or changing to new COX2 inhibitors.

Pharmacotherapy: For symptomatic relief & ulcer healing.

- Antacids Al, Ca, Mg, alginate & LAs Mild relief only, don't heal. SE: constip/diarrhoea
- Proton-pump inhibitors for acid suppression: full dose PPI for 1-2mo. usually heals ulcer. E.g.: Omeprazole, pantoprazole, esomeprazole, rabeprazole all 40mg/day PO. SE: GI upset, dry mouth, headache
- H₂ antagonists, reduce acid production, are less effective than PPIs.
 E.g. ranitidine 300mg or cimetidine 800mg/day in single/split doses. Cimetidine has sig.
 SE: ↑PRL, gynaecomastia, impotence, CYTP450 inhibition & so ↑warfarin, phenytoin etc.
- PGE₁ analogue misoprostol 800mg daily inhibits H2-dependent gastric acid secretion & is cytoprotective but \text{leffective than PPI at healing. May be better at prevention in smokers & NSAID related ulcers. SE: GI upset, facial flushing, tremor.
- Cytoprotectants: These chelate to proteins at the base of the ulcer. Bismuth also suppresses H.pylori but relapse rates high.
 bismuth SE: NH₃-taste, dark tongue, black stools, long term Rx can → renal toxicity. sucralfate (Al salt of sucrose, requires low pH). SE: constip, bioavail phenytoin.

H.pylori eradication:

- If H.Pylori +ve. Also if DU or on NSAIDs (\precurrence).
- Triple therapy PPI + dual ABx or bismuth. Number of regimes suggested.
- E.g. amoxicillin 1g+clarithromycin 500mg+omeprazole 20mg PO bd x 7d. Cont PPI x 4-8wks.
- Or pantoprazole $40mg+amoxicillin 1g bd \times 5d$ then pantoprazole $40mg+clarithromycin 500mg+tinidazole 500mg bd \times 5d$

Management of recurrence:

- For gastric ulcer with H pylori infection → eradication therapy followed by proof of eradication and repeat endoscopy (proof of ulcer healing & not malignant).
- If failure of eradication \rightarrow repeat with different antibiotics.

Surgery

- Profuse bleeding/exsanguinating
- Perforation
- Rebleeds in hospital
- Failed endoscopic Mx
- Consider if giant GU or posterior DU

Complications

- Haemorrhage (10-20%) → [see Gastrointestinal Haemorrhage]
- $\bullet \quad \text{Perforation (5\%)} \rightarrow \text{acute abdomen} \rightarrow \text{ABC, NGT, ABx, \pm surgery}$
- Penetration through to other viscera e.g. pancreas
- \bullet $\;$ Scarring of the duodenum may \rightarrow pyloric stenosis with vomiting and weight loss (rare)
- Malignancy (GU>>>DU)

Prognosis

Prognosis is excellent if the underlying cause such as H pylori infection or drugs addressed. Annual recurrence <2% if treated GU>DU. Higher if smoking, EtOH, NSAIDs. Mortality <10%.