GASTRO-OESOPHAGEAL REFLUX DISEASE

GOR: symptoms caused by pathological reflux of duodenal/gastric contents into oesophagus
GORD: GOR + mucosal damage NERD: endoscopically normal with GOR symptoms

Epidemiology

Misreporting: normal physiological phenomenon in awake, upright, post-prandial humanoids

Aetiology

Failure of distal oesophageal reflux barrier/increased acid or bile load/downstream resistance
LOS hypotension: (i)DRUGS (CaChB/nitrates/anti-Ach) (ii)SMOKING(iii)EtOH (iv)CAFFEINE (v)PREGNANCY: P_4 (iv) LOS hypotension
Loss of mechanical barrier: (i)HIATUS HERNIA: loss of pinchcock/intra-abdo segment/angle of His$_{(66\% \text{ get GORD})}$ (ii) OBESITY$_{(AoH)}$
Hyperacidity: (i)H. pylori (ii)DIETARY: food/booze (iii)ZES
Intra-abdominal pressure: (i)PREGNANCY (ii)OBESITY
Delayed gastric emptying: (i)DIABETES (ii)HYPOTHYROIDISM (iii)OBESITY

Pathology

TLOSR: LOS relaxes separate from swallow || Pathological TLOSR: 90% of reflux episodes; later disease= LOS hypotension itself; HH eventually
Acid: H^+ enters cytosol, disrupts homeostasis → apoptosis/noci+chemoceptors in intercellular spaces/distal oes spasm

Clinical features = typical vs atypical/PPI responder vs non-responder

1a **TYPICAL:** heartburn/regurgitation$_{(microaspiration \rightarrow cough/asthma)}$/acid brash
1b **ATYPICAL:** non-heartburn pain/cough/hoarseness/other: pharyngitis, tonsillitis, sinusitis
Worse on (i)bending over (ii)supine (iii)night (LOS relaxed/supine) || Better with PPI?
2. **Complications**: oesophagitis/stricture/bleeding/cancer
3. **Cancer** red flag symptoms (age>55/blood/dysphagia/odynophagia/wt loss/mass/<6mths/>5yrs)
EROSIVE: responds to acid suppression NERD: atypical symptoms with variable response BARRETT'S: minimal symptoms

Complications

1. **EROSIVE OESOPHAGITIS/ULCER:** *full dose PPI 8 weeks then maintenance (NICE)*
2. **BENIGN STRICTURE:** (i)"**stretch**": dilatation$_{+/-repeat}$ (ii)**steroid** (iii)**stent** (iv)**surgery** *NICE: long-term PPI after dilatation*
3. **BARRETT'S OESOPHAGUS:** intestinal metaplasia replaces damaged squamous epithelium
4. **MALIGNANT STRICTURE: ADENOCARCINOMA**
5. **BLEEDING**

INVESTIGATIONS

1. OGD: diagnostic$_{(visualise\ oesophagitis\ and\ complications/biopsy)}$ + therapeutic
Savary-Miller: 1=erythema/oedema 2=linear erosion 3=circumferential erosion/superficial ulcer 4=complications eg stricture

2. BARIUM$_{(DCS)}$ (i)**Complications**$_{(stricture/cancer)}$ (ii)**Anatomy**$_{(HH)}$ (iii)**Function**$_{(motility/gastric\ emptying)}$ **poor demo of reflux if intermittent/low volume**

3. pH STUDIES: (i)TYPICALS refractory to treatment (ii)ATYPICALS (iii)suspect motility disorder
Principle(a)Probe: **pH<4** as reflux episode; normal total reflux time **<5%/24hr** (b)Patient: **symptoms/dietary intake/position**
Tekkers: (a)**24Hr Ambulatory** (b)**BRAVO**$_{wireless\ capsule + probe + RF\ transmitter\ 6cm\ above\ GOJ;\ better\ tolerated\ so\ longer\ (>96hrs)\ test}$
DeMeester score: # of episodes/>5min episodes/total reflux time/longest episode/upright+supine time+total time (<14.42 normal)
Limitations: normal study doesn't rule out GORD$_{: non/weakly\ acid\ refluxate}$

4. OESOPHAGEAL IMPEDENCE: *resistance to AC to differentiate (i)state of refluxate (ii)direction, velocity, time, extent of flow*
Liquid improves conduction, impedance decreases ||gas impairs conduction, impedance increases
Information: fluid v gas/swallow vs reflux/proximal extent of reflux/velocity/time to clear/re-reflux episodes *NOT VOLUME*
*Combine with ph studies → most sensitive GORD measurement (reflux detection and symptom correlation)
*Especially useful if normal pH (?weakly acidic/duodenogastric)

5.MANOMETRY: *exclude dysmotility/motility disorder (eg achalasia, diffuse oes. spasm) before fundoplication*
(a)**STANDARD STATIC MANOMETRY**: multiple sensors ax peristalsis (normal/non-peristaltic/simultaneous) + LOS (rest/squeeze)
10 wet swallow test: UOS/LOS relax → 1° peristalsis (segmental contraction wave 5cm/s; 180mmHg distally)
3 limitations: test conditions /miss intermittent dysmotility/miss dysmotility between sensors
(b)**HIGH-RESOLUTION MANOMETRY**: more (36) sensors so more closely spaced for more comprehensive assessment

MEDICAL MANAGEMENT

Lifestyle: elevate bed/weight loss/dietary measures to reduce acid and LOS hypotension
LOS: avoid LOS hypotensives (CaCHBs/nitrate/anti-ACh/caffeine/smoking/EtOH) + lose weight
Gastric emptying: (i)treat DM/hypoT4 (ii)weight loss (iii)cisapride
Intra-abdominal pressure: weight loss
Hyperacidity: (i)H. pylori eradication (ii)ZES (iii)PPI (iv)diet
Mechanical barriers: weight loss to restore angle of His/operate on HH

Drugs *(NICE 2014)*

PPI: Full-dose 4-8wks converts acid reflux to non-acid reflux but still have volume reflux (**H2RA** switch if non-response; less effective)
Parietal cell hyperplasia so symptoms recur on cessation + atrophic gastritis with intestinal metaplasia in H. pylori
Cisapride: only prokinetic shown to be better than placebo

SURGICAL MANAGEMENT: FUNDOPLICATION

Principle: (i)create effective barrier to reflux at GOJ (ii)reduce TLOSRs Doesn't reliably lead to regression of Barrett's/adenocarcinoma risk

Indications:

1. Objective evidence of pathological reflux +/- complications
2a Refractory reflux despite adequate pharmacological therapy Poorer outcome (56% success at 11yrs); true non-responders ?alternative diagnosis
2b Medically-responsive reflux but unwilling to take medications for life Better response if symptom relief with acid suppression (77.1% success at 11yrs)

Contraindications (i)Oesophageal Ca (ii)Barrett's HGD (iii)Motility disorder *dysmotility/gastric outflow not absolute contraindications → partial wrap*

Workup: OGD/pH/impedence/manometry **Barium swallow not mandatory unless reoperation*

Procedures

1.Nissen: posterior fundus passed behind oesophagus and fixed to **anterior fundus**
2.Posterior Partial*:* **Toupet posterior 270^0**-fundus passed behind oesophagus + fixed to **lateral oesophageal walls**
3. Anterior Partial:
Dor: fundus passed anteriorly and fixed to **lateral oesophageal walls**
90^o: fundus fixed to **left anterolateral oesophageal wall +posterior oesophagopexy**
120^o: fundus fixed to **right anterolateral oesophageal wall**
180^o: fundus fixed to **right + left hiatal rim +posterior oesophagopexy**

Dysphagia: universal in first few weeks; if persists may be tight hiatus (scarring or overtightening → dilate/cut hiatal rim
Gas-Bloat: one-way valve prevents belch/vomit → flatulence;
Reflux:

Procedure-specific outcomes

Nissen vs posterior: posterior= less gas-bloat; dysphagia similar in Toupet with; reflux higher
Nissen vs anterior: 90^o/180^o= less dysphagia/gas-bloat but more reflux
Wrap length important: 3cm>1.5cm>1cm

Patient-specific Outcomes

1. Responders vs non-responders: 77.1% success at 11yrs in responders vs 56% ?alternative diagnosis in true non-responders
2. Typicals vs atypicals: 85% overall in typicals vs 41% overall in atypicals ?alternative diagnosis in true atypicals

Complications

General: DVTPE/infection: pericardial, mediastinal, peritoneal, LRTI/bleeding/
Dissection: pneumothorax/pneumomediastinum/perforation of oesophagus, stomach, duodenum/liver injury
Mechanical: sliding or migrating wrap/hiatal stenosis (scarring, overtightening)/bilobed stomach

Failure after surgery

1. Recurrent reflux: wrap disruption/slippage/migration$_{\text{esp if large HH defect}}$ → repeat physiology ?true reflux
2. Persistent pain: original diagnosis of pain wrong → review physiology studies
3. Gas-bloat: usually air-swallowers or FGID but vagotomy can be cause → pyloric dilatation/roux-en-Y
4. Dysphagia: (i)persistent after op= motility disorder missed/tight wrap/tight hiatus (ii)after normal swallowing period= wrap related/hiatal scarring/cancer
Ix: OGD: de novo path/Ca/wrap/hiatus ||Barium: wrap/hiatus/gastric emptying||CT: HH||Mano: missed motility disorder/dysmotility ||pH studies: other dx

BARRETT'S OESOPAGHUS

Definition: Any portion of normal distal oesopagheal squamous epithlieum with metaplastic columnar epithelium
Must be (a) endoscopically **visible** (1cm above true GOJ) and (b)confirmed by **biopsy**

Aetiology: (i)Reflux (12% of GORD) and (ii) non-reflux
Pathogenesis: adaptive to acid/alkaline reflux causing squamous cell damage; *p53/p16 (TS)* induced by → clonal expansion

Diagnosis

Endoscopy: **Prague** (CxMx) and **Paris** (lesions); **2cm quadrantic biopsies;** any noduler lesion is cancer until disproven
Histo: (i)**Metaplasia** (i)**LGD**: loss of goblets/differentiation (ii)**HGD**: pleomorphism/crypt loss (iii)**IMN**: invades lamina propria (T1)
Genetics: ***p53*** testing
Risk of cancer progression **<1%/yr**= depends on (i)**length** (ii)**H/LGD (iii)intestinal/cardiac/fundic metaplasia**

Screening

Not feasible in general population (Barrett's common with low risk of progression)
Indicated if GOR with risk factors: **3+ of >50/male/white/obese**

1. Surveillance of intestinal metaplasia *(RCT data lacking but recommended if intestinal metaplasia)*

(i)<3cm without IM/dysplasia → **rpt** → **discharge** if normal (ii)<3cm with IM → **3-5yrly** scope (iii)>3cm with IM →**2-3yrly** scope

2. Management of dysplasia

(i)INDEFINITE FOR DYSPLASIA: PPI + repeat 6mths→if normal repeat 6mths then treat as non-dysplastic (2-3/3-5yrly)
(ii)LGD: PPI + repeat 3mths→ 6mthly scopes if persistent *EMR for visible lesions
→ 2yrly scopes if regresses *RFA for flat lesions

(iii)HGD: 2nd GI pathologist confirmation/quadrantic 1cm biopsies/staging including diagnostic EMR/MDT
EMR visible lesions (95% remission; 14% metachronous lesion at 12mth) || **RFA** dysplasia without visible lesion (81% remission)
Oesophagectomy: M&M less than with invasive cancer; optimal staging; complete removal → no surveillance

Follow-up

Lifelong endoscopic surveillance if endotherapy (not if oesophagectomy

RFA:
Reduces progression of HGD to cancer
Ablate entire Barrett's segment (metachroncous recurrence 20% at 2yrs) not just dysplasia
Risks: stricture/bleeding/perforation + treatment failure + recurrence

GORD HISTORY
Pc: Symptoms
HPc (i)GOR (typical/atypical/PPI response (ii)Complications (stricture, bleeding, ulcer, cancer) (iii)Cancer (iv)motility/dysmotility
PMHx: Barrett's/cancer treatment + DM/thyroid for delayed gastric emptying
MedX: acid ie NSAIDS/steroids/EtOH/smoking; LOS relaxant ie caffeine/smoking/EtOH/P4
FHx: Barrett's/oes cancer/HH
SHx: occupation and ethnicity for oes ca risk; diet

GOR + GOR complications + cancer symptoms
Medical causes of GOR
drug causes of GOR and treatment response to PPI
FHX of cancer
SHx for ca risk