

Potential Causes of Dyspepsia

Dyspepsia A non specific term, encompasses a number of symptoms attributable to the upper GI tract.

Oesophagitis Gastro oesophageal reflux disease (GORD). Barrett's oesophagus

Gastritis Type: A,B,C

Peptic Ulcers Gastric ulcers. Duodenal ulcers

Zollinger-Ellison syndrome a rare condition caused by tumors (gastrinomas) that produce excessive gastrin, leading to overproduction of stomach acid and peptic ulcers.

Gastric Cancer

3 categories of Gastritis

Type A - AUTOIMMUNE (antibodies against parietal cells). Reduced or no acid secretion and intrinsic factor. Aplastic anaemia due to Vit B12 deficiency.

Type B - BACTERIAL *Helicobacter pylori* infection. Elevated acid secretion

3 categories of Gastritis (cont)

Type C - CHEMICAL Chemicals/drugs eg aspirin. Elevated acid secretion

Diet & Lifestyle & Other drugs

Other causes of Dyspepsia

Caffeine: PDE inhibitor promotes acid secretion

Alcohol: Dissolves mucous layer

Spicy Food: Capsaicin, Activates TRPV1 but may inhibit acid secretion via vagal inactivation.

Concomitant medication (drugs that can relax LOS): PDEV inhibitors eg sildenafil like drugs. Nitrates (relaxes LOS via PDE activation). Theophylline (Relaxes LOS via PDE inhibition). Drugs with antimuscarinic properties (block muscarinic receptors). Ca2+ channel blockers (prevent calcium entry).

Obesity and pregnancy increased intra-abdominal pressure causing reflux.

The Oesophagus

The mucosa of the oesophagus is non-keratinized stratified squamous epithelium

The type of muscle in the muscularis of the oesophagus varies by region the superior 1/3 is skeletal muscle, the intermediate 1/3 is skeletal and smooth muscle, the inferior 1/3 is smooth muscle.

The Oesophagus (cont)

Adventitia replaces serosa. **Serosa** = a slick covering that helps organs move smoothly (like the outer wrap of your intestines). **Adventitia** = a rougher outer layer that holds the organ in place, usually found where organs are attached to other tissues (like parts of the esophagus or rectum). Meaning that part of the organ is not freely moving inside a cavity anymore, but rather fixed or connected to surrounding structures.

Type A Gastritis

Destruction of parietal cells Reduced or absent acid secretion. Vitamin B12 deficiency. Anaemia

Type A Gastritis (cont)		Acid Secretion		Type B Gastritis - Helicobacter pylori (cont)		Eradication of H.pylori (cont)	
Other conditions associated with Type A Gastritis	Autoimmune thyroiditis (Hashimoto's disease). Type I Diabetes. Addison's Disease (Adrenal glands, reduced cortisol & aldosterone). Vitiligo (skin pigmentation disorder) white patches of skin.	M3 and CCK2 (CCKB; gastrin) receptors	GTP-binding protein coupled receptor (GPCR). Linked to Gq (stimulates Phospholipase C). Increases intracellular Ca ²⁺ via PIP2 conversion to DAG & IP3.	Secretes	urea from high urease activity (antral pH raised, gastrin & acid secretion increases). PAF (platelet activating factor).	If allergic to penicillin and previous exposure to clarithromycin and metronidazole ONE WEEK TWICE DAILY	Tetracycline 1g and metronidazole 400 mg. Bismuth subsalicylate and omeprazole 20 mg
Treatment potentially required	Hydroxocobalamin injections	H2 receptors	GTP-binding protein coupled receptor (GPCR) Linked to Gs (stimulates adenylate cyclase) Increases intracellular cAMP	Gram negative	Doesn't retain Crystal Violet stain! - Pink stain!!!	Mucosa aggressors and protectors	
Type C Gastritis - Chemical, Drug and Diet		Inhibit or Reduce acid secretion:	Proglumide, Misoprostol, H2 Blockers, Atropine, Proton Pump Inhibitors.	Eradication of H.pylori		Protective	Mucus, Prostaglandins, Bicarbonate, Mucosal blood flow
SAID's	Steroidal anti-inflammatory drugs (SAID's) inhibit phospholipase A2 by promoting expression of annexin 1 and suppressing expression of COX-2.	Type B Gastritis - Helicobacter pylori		First Line treatment	ONE WEEK TWICE DAILY Amoxicillin 300mg and either Clarithromycin 500 mg or Metronidazole 400 mg and either omeprazole 20 mg or lansoprazole 30 mg.	Aggressive	Acid, Pepsin, NSAID's, H. pylori, Drugs, Diet
NSAID's	Non-steroidal anti-inflammatory drugs inhibit the cyclo-oxygenase enzymes. COX-1 COX-2	Associated with:	80% of gastric ulcers. 95-100% of duodenal ulcers. 100% chronic antral gastritis. gastric cancer (younger infected, greater chance)		Consider lowest acquisition costs and previous exposure to clarithromycin or metronidazole!	Treatment for Dyspepsia	
Barrett's Oesophagus		Gram negative spiral bacterium	colonises mucus in both stomach and duodenum.	If allergic to penicillin ONE WEEK TWICE DAILY	Clarithromycin 500 mg Metronidazole 400 mg and either Omeprazole 20 mg or lansoprazole 30 mg	Surgery (1900--1970's)	Gastric vagotomy & antacids
Long-standing reflux of acid <i>About 1 in 10 people with GORD develop Barrett's oesophagus.</i>						Drugs (1970's onwards)	
Normal stratified squamous epithelium is replaced with simple columnar epithelium with goblet (mucus cells)						Barriers/-Protection:	Alginate and Sucralfate-antacid and barrier
						Muscarinic cholinergic receptor antagonists (M3)	Pirenzepine
						Selective H2 receptor antagonists	Cimetidine, ranitidine, famotidine
						Drugs (1990's onwards)	
						Proton Pump Inhibitors	Omeprazole, Pantoprazole, Lansoprazole, Rabeprazole and Esomeprazole

protecting gastric mucosa when taking NSAID's

Synthetic PGE2 Misoprostol + NSAID. Problem – smooth muscle relaxation – diarrhoea.

Antacids Inhibit acid secretion: H2 antagonists eg: TUMS, famotidine. **Rennie.** **Proton Pump Inhibitors (PPI's)** eg omeprazole, lansoprazole, esomeprazole, pantoprazole.

Selective COX-II inhibitors Diclofenac, refocoxib (Vioxx)

Emerging novel NSAIDS (not clinically used) NO-flurbiprofen (nitric oxide releasing derivatives). H2S releasing NSAID's (currently awaiting MAA)



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