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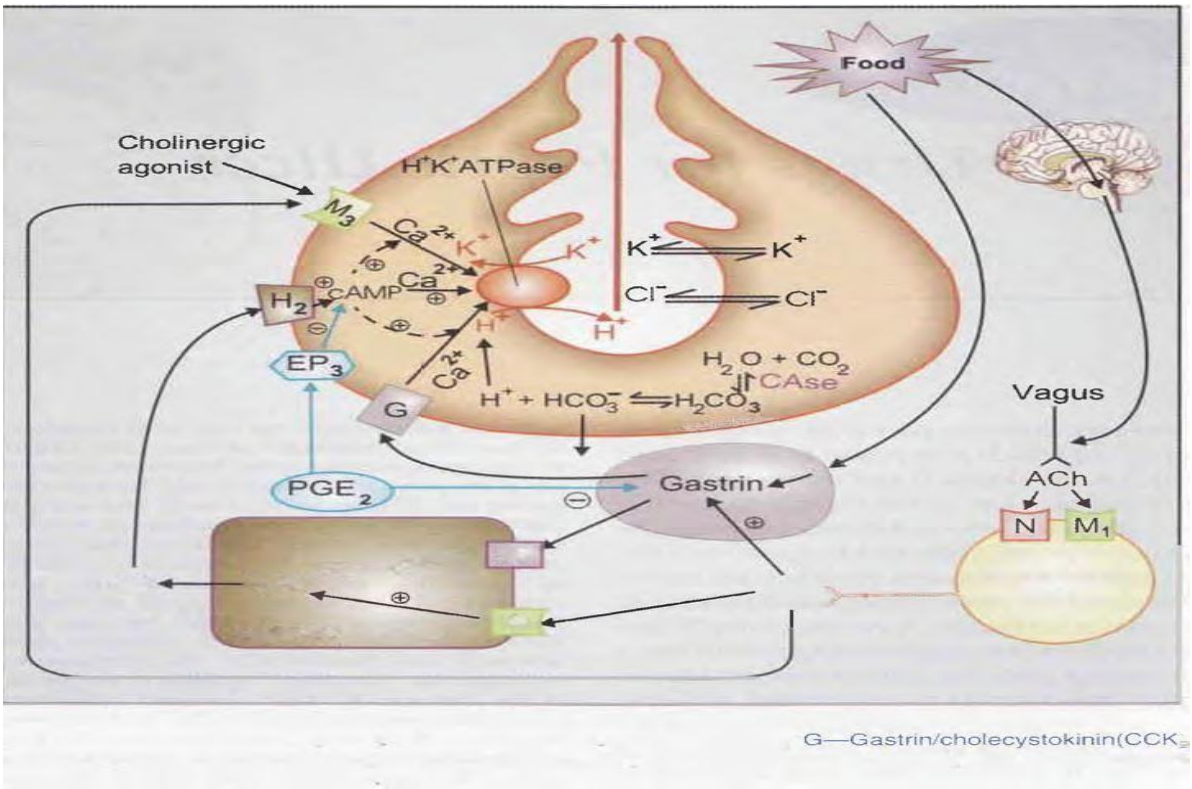
ANTI ULCER DRUGS

Peptic ulcer occurs in that part of the gastrointestinal tract (g.i.t.) which is exposed to gastric acid and pepsin, i.e. the stomach and duodenum. The etiology of peptic ulcer is not clearly known. It results probably due to an imbalance between the aggressive (acid, pepsin, bile and H. pylori) and the defensive (gastric mucus and bicarbonate secretion, prostaglandins, nitric oxide, innate resistance of the mucosal cells) factors.

Regulation of gastric acid secretion



CLASSIFICATION:

1 . Reduction of gastric acid secretion

(a) H2 antihistamines: Cimetidine, Ranitidine, Famotidine, Roxatidine

(b) Proton pump inhibitors: Omeprazole, Lansoprazole, Pantoprazole, Rabeprazole, Esomeprazole

(c) Anticholinergics: Pirenzepine, Propantheline, Oxyphenonium

(d) Prostaglandin analogue: Misoprostol

1. Neutralization of gastric acid (Antacids

(a) Systemic: Sodium bicarbonate, Sodium citrate

(b) Nonsystemic: Magnesium hydroxide, Mag. trisilicate, Aluminium hydroxide gel, Calcium carbonate

3. Ulcer protectives: Sucralfate, bismuth subcitrate (CBS)

4Anti-H. pylori drugs: Amoxicillin, Clarithromycin, Metronidazole, Tinidazole, Tetracyclin

H2 ANTAGONISTS

These are the first class of highly effective drugs for acid-peptic disease. Four H2 antagonists cimetidine, ranitidine, famotidine and roxatidine are available in India; many others are marketed elsewhere. Their interaction with Hz receptors has been found to be competitive in case of cimetidine, ranitidine and roxatidine, but competitive noncompetitive in case of famotidine.

- H2 blockade Cimetidine and all other Hz antagonists block histamine-induced gastric secretion
- Gastric secretion The only significant in vivo action of Hz blockers is marked inhibition of gastric secretion

PROTON PUMP INHIBITORS (PPis)

Omeprazole It is the prototype member of substituted benzimidazoles which inhibit thefinal common step in gastric acid secretion and have overtaken H2 blockers for acid-pepticdisorders. The only significant pharmacological action of omeprazole is dose dependent suppression of gastric acid secretion; without anticholinergic or H2 blocking action. It is a

powerful inhibitor of gastric acid: can totally abolish HCl secretion, both resting as well as that stimulated by food or any of the secretagogues, without much effect on pepsin, intrinsic factor, juice volume and gastric motility.

Zollinger-EIIison syndrome It is a gastric hypersecretory state due to a rare tumour secreting gastrin. H2 blockers in high doses control hyperacidity and symptoms in many patients, but relief is often incomplete and side effects frequent. PPis are the drugs ofchoice.

ANTICHOLINERGICS

A tropinic drugs reduce the volume of gastric juice without raising its pH unless there is food in stomach to dilute the secreted acid. Stimulated gastric secretion is less completelyinhibited. Effective doses (for ulcer healing) of nonselective antimuscarinics (atropine,propantheline, oxyphenonium) invariably produce intolerable side effects.

PROSTAGLANDIN ANALOGUE

PGE2 and PGI2 are produced in the gastric mucosa and appear to serve a protective role by inhibiting acid secretion and promoting mucus secretion. In addition, PGs inhibit gastrinproduction, increase mucosal blood flow and probably have an ill-defined "cytoprotective"action.

ANTI-HELICOBACTER PYLORI DRUGS

H. pylori is a gram negative bacillus uniquely adapted to survival in the hostile environment of stomach. It attaches to the surface epithelium beneath the mucus, has high urease activityproduces ammonia which maintains a neutral microenvironment around the bacteria, andpromotes back diffusion of H + ions