PEPTIC ULCER DISEASE

Definition: Peptic ulcer disease (PUD) refers to a group of ulcerative disorders of the upper GI tract that require acid and pepsin for their formation. Ulcers differ from gastritis and erosions in that they extend deeper into the muscularis mucosa. The three common forms of peptic ulcers include Helicobacter pylori (HP)–associated ulcers, nonsteroidal antiinflammatory drug (NSAID)–inducedulcers, and stress-related mucosal damage (also called stress ulcers.

The most common factor which one could observe in the all the different types of Peptic ulcer is it occur in the presence of acid and pepsin when HP, NSAIDs, or other factors **disrupt normal mucosal defense and healing mechanisms.** Acid is an independent factor that contributes to disruption of mucosal integrity. Increased acid secretion has been observed in patients with DU and may result from HP infection.

Alterations in mucosal defense induced by HP or NSAIDs are the most important cofactors in peptic ulcer formation. Mechanisms include mucus and bicarbonate secretion, intrinsic epithelial cell defense, and mucosal blood flow. Maintenance of mucosal integrity and repair is mediated by endogenous prostaglandin production

H. Pylori induced ulcer

Most non-NSAID ulcers are infected with HP, and HP eradication markedly decreases ulcer recurrence. HP may cause ulcers by direct mucosal damage, altering the immune/inflammatory response, and by hypergastrinemia leading to increased acid secretion.

Nonselective NSAIDs (including aspirin) cause gastric mucosal damage by two mechanisms: (1) a direct or topical irritation of the gastric epithelium, and (2) systemic inhibition of the cyclooxygenase-1 (COX-1) enzyme, which results in decreased synthesis of protective prostaglandins.

Other factors which add on to the pathophysiology is Cigarette Smoking, Cola, Coffee, Beverages

Clinical Presentation: Abdominal pain is the most frequent symptom of PUD. The pain is often epigastric and described as burning but can present as vague discomfort, abdominal fullness, or cramping. A typical nocturnal pain may awaken patients from sleep, especially between 12 AM and 3 AM.

• Pain from DU often occurs 1 to 3 hours after meals and is usually relieved by food, whereas food may precipitate or accentuate ulcer pain in GU. Antacids provide rapid pain relief in most ulcer patients.

• Heartburn, belching, and bloating often accompany the pain. Nausea, vomiting, and anorexia are more common in GU than DU.

• The severity of symptoms varies from patient to patient and may be seasonal, occurring more frequently in the spring or fall.

Pharmacotherapy

First-line eradication therapy is a proton pump inhibitor (PPI)–based, three-drug regimen containing two antibiotics, usually clarithromycin and amoxicillin, reserving metronidazole for back-up therapy (e.g., clarithromycin–metronidazole in penicillin-allergic patients). The PPI should be taken 30 to 60 minutes before a meal along with the two antibiotics. Although an initial 7-day course provides minimally acceptable eradication rates, longer treatment periods (10 to 14 days) are associated with higher eradication rates and less antimicrobial resistance.

First-line treatment with quadruple therapy using a PPI (with bismuth, metronidazole, and tetracycline) achieves similar eradication rates as PPIbased triple therapy and permits a shorter treatment duration (7 days). However, this regimen is often recommended as second-line treatment when a clarithromycin–amoxicillin regimen is used initially. All medications except the PPI should be taken with meals and at bedtime. • If the initial treatment fails to eradicate HP, second-line empiric treatment should: (1) use antibiotics that were not included in the initial regimen; (2) include antibiotics that do not have resistance problems; (3) use a drug that has a topical effect (e.g., bismuth); and (4) be extended to 14 days. Thus, if a PPI–amoxicillin–clarithromycin regimen fails, therapy should be instituted with a PPI, bismuth subsalicylate, metronidazole, and tetracycline for 14 days. • Treatment with a conventional antiulcer drug (e.g., PPI, histamine-2 receptor antagonist [H2RA], or sucralfate alone is an alternative to HP eradication but is discouraged because of the high rate of ulcer recurrence and ulcer-related complications.

For treatment of NSAID-induced ulcers, nonselective NSAIDs should be discontinued (when possible) if an active ulcer is confirmed. Most uncomplicated NSAID-induced ulcers heal with standard regimens of an H2RA, PPI, or sucralfate if the NSAID is discontinued.