Peptic Ulcer Disease

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- **<u>Peptic Ulcer Disease</u> :**
- Break in mucosa of the stomach and small Intestine principally the duodenum produced by the action of gastric secretion affect 10% of population that extends through the muscularis mucosa, submucosa or deeper.
- Although P.U.D. is one of the most common disorder in human, it is still unknown why ulcers develop and why they heal.

P.U. can occur as high as Barrette's esophagus and as low as Meckel Diverticulum i.e. it is chronic, most other solitary lesion that occur in any portion of the G.I.T. exposed to the aggressive action of acid/peptic juices located in the following site with decreasing frequency.

1. Duodenum 1st part.
2. Stomach – antrum.
3. GE junction in the setting of gastroesophageal reflux and Barrett esophagus.
4. Within the margin of a gastrojejunectomy.
5. In duodenum, stomach and jejunum in patient with ZE sample.
6. Within or adjacent to an ileal Meckel diverticulum that contain ectopic gastric mucosa.

- **<u>Epidemiology</u> :**
- It was uncommon during 19th century and that occurred was gastric ulcer after World War I. The occurrence of D.U. changed from rare event to common one while G.U. decreased in incidence and become a disorder of elderly.
- P.U. has progressively increased in the past 50 years with peak incidence at the age 30-60 M > F in 20th century and at 19th F > M.
- It is rare in black African but in USA both B and W have the same incidence.

PATHOGENESIS

- Peptic ulcers are produced by an imbalance between gastroduodenal mucosal defense mechanimized the damaging forces particularly gastric acid and pepsin. However, hyperacidity is not prerequest except for few cores of duodenal ulcer but less in gastric ulcer.

Environment factor:

- No evidence of relationship of spicy food and alcohol with P.U.
- However alcohol -→ cirrhosis --→ ↑ incidence of P.U.
- Drugs as Aspirin and other non-sterioidal anti inflammatory drugs have been incriminated in the production of peptic ulcer.
- Cigarette smoking is a definite risk factor for duodenal and peptic ulcer.

Genetic factor:

Families, twins, blood group O.

- Pepsinogen I is usually secreted by the chief and mucous cells. Patient with high level of pepsinogen have high risk of developing G and D.Us.
- It is autosomal dominant familial hyper functioning of gastric secreting cells is also being demonstrated to contribute to P.U.

Psychological Factor:

Stress

Hydrochloric Acid:

- HCL ↑ contribute to duodenal ulcer more than gastric ulcer.
- ↑ Pepsinogen → ↑ HCL
- Stomach
- **Gastritis** → Acute
- Chronic

Acute gastritis : irritants as drugs or alcohol.

- Vasodilatation of oedema → Erosion and haemorrhage which can be small and multiple. Transient with rapid healing. Haemorrhage can be life threading.

Chronic gastritis

- **ill defined**
- Type A-Chronic gastritis (Auto-immune) Ab in the serum are directed antipartial cells.
- Variable degree of hypochlorohydria, macrocytic anaemia due to vit B12 defining.
- Body of the stomach is affected with marked loss of glandular epithelium.
- Lamina propria is infiltrated by lymphocytes and plasma cells with presence of intestinal metaplasia.
- Neutral secreting mucus cell will be replaced by goblets cells contain acidic glycoprotein typical of intestine which is recognized as pre-malignant condition although a few case develop malignancy.
- Presence of intestinal metaplasia in malignant tumour does not imply its origin from metaplasia.

(2) Type B- (Bacterial Ch. Gastritis).

- **Helicobacter pylori – g-ve in area when PH close to neutrality. 90% of Bx. with chronic gastritis in type B but uncommon in type A – found only in gastric endothelium and not duodenum.**
- **Acute gastritis by Helicobacter pylori is mediated by complement → Neutrophils infiltrate → proteases production → glandular destruction.**
- **Anti Helicobacter pylori IgA, IgG, IgM Abs are produced by plasma cells affect antrum initially but in long standing → all the stomach is affected.**
- **(3) Type C: Reflux gastritis:**
- **Regurgitated bile + alkaline duodenal juice → epithelial esquamation → proliferative hyperplasia + oedema + vasodilatation.**
- **Seen in**
- **(a) Post – Operative stomach.**
- **(b) Pyloric incompetence.**
- **(4) Other forms of gastritis:**
- **Lymphocytic gastritis:**
- **Eosinophilic gastritis**
- **Granulomatous – Crohn's or T.B.**

- **PEPTIC ULCER**
- **Sites** : 1st part of the duodenum Junction of antral and body mucosa
- Distal oesophagus
- Gastroenterostomy stroma

Etiology : Hyperacidity, Helicobacter pylori, duodenal reflux. NSAIDs, smoking and genetic factors.

- **Gastric Ulcer** **Duodenal Ulcer**
- **1** : **3**
  - Incidence
- Increase with Age :7 upto 35 years - Age
- A O - Blood group
- Normal or low : ↑↑
- - Acid level 70% :
  95-100% - H. Pylori
- **Peptic Ulcer can be acute or chronic**
- **Acute** : Can develop through
- - As part of an acute gastritis
- - Complication of severe stress
- - Extreme hyperacidity

- Extension of ulcer can result from NSAIDs and alcohol overdosage. Stress is the common cause as severe burns (Curling's ulcer)
- Major trauma or cerebrovascular accident probably due to mucosal ischaemia.
- Extreme hyperacidity as Z-E-Syndrome → acute ulcer of antrum and duodenum and Jejunum.
- **Chronic Ulcer**
- Occur most commonly at mucosal junction i.e. ulcers are found in the antral body mucosa type. Duodenum with pylorus, oesophagus at cardio-oesophageal.
- **Pathogenesis** : ↑ Acid production is thought to be the cause for many years where people with ulcer are found to have low or normal acid production. So an-ulcer could represent an adverse outcome of conflict between aggressive forces in the stomach or duodenum with the defense mechanism. In which normal acid/pepsin attack is balanced by mucus barrier and other defense mechanisms.
- ↑ attack by hyperacidity or NSAID → ulceration of the mucosa. ↓ mucosal defense due to Helicobacter pylori → ulceration in the presence of normal or reduced acid level.

Ulcers can be

Gastric : PH- is acidic under fasting condition in which unprotected mucosa would undergo auto digestion.

Mucosal defence

- Mucus – bicarbonate
- Surface epithelium.
- Ulceration can occur when either of them are damaged.

Mucus can be damaged due to

- duodeno-gastric reflux

Epithelium can be damage by

- NSAID will block PGs or due to Helicobacter pylori infection.

2. Duodenal Ulcer :

50% can show ↑ acid secretion.

Other factors

- genetic predisportion
- Blood group O
- HLA B5 – AG
- Autosomal dominant hyperpepsinogenomia, smoking

- **<u>Pathology</u> :**
- Chronic PU usually < 20mm but may be larger upto 100mm.
- Edge are clear-cut
- Microscopically : Necrotic base
- Granulation tissue
- Arteries with extreme narrowing pylori sterosis.
- **Complication :**
- Perforation
- Penetration
- Hemorrhage
- - Stress, Anxiety, Fatigue, intake the unbuffered
- aspirin smoking, carticosteroids
- - Helicobacter pylori's role ???

Gastric Ulcer :

- Patients usually have low or normal level of gastric acid but never Achlorohydria. Therefore all views that there is primary defect in gastric mucosa resistance. → ↑ tendency to back diffusion of H + → disagreement in gastric mucosal barried.
- Frequent association with chronic antral gastritis with ulcer in 60 – 80% gastritis are found with ulceration mainly in antrum but can be seen in the body gastritis can persist despite ulcer healing explaining that gastritis primary for ulceration is secondary.
- Gastritis can be caused by reflux of bild due to Iavered Sphincter incompetence.
- Also Sphincter response does not ↑ in response to Secretin, Cholecystokinin or intraduodenal amino acid or fat.
- Sphinecter presence ↓↓ = smoking
- Exogenous exposure to damaging agent as aspirin, alcohol, non-steriodal anti-inflammatory drugs. They are usually habitant user of these analgesic.