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A Critical Review of Peptic Ulcer Disease

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Abstract

Disorders related to the digestive system are on the rise due to the faulty diet and habits. One such condition is Peptic ulcer. Peptic ulcer is an worldwide problem and its prevalence in India particularly south India is quite high, Recent studies suggests approximately 10% of adults at some times of their lives get affected by peptic ulcer. The cardinal feature of Peptic Ulcer Disease is pain during digestion of food which torments the person at every meal time and is a source of constant discomfort. Considering the gravity of the condition, A Critical review of the Peptic Ulcer Disease is made from various sources of available literature.

Key words: Peptic Ulcers, Ulcers, PUD, Peptic Ulcer Disease, H. Pylori.

Introduction

Ulcers are defined as breaks in the mucosal surface >5 mm in size, with depth to the submucosa.^{1,2}

Peptic ulcer is a term used to refer to a group of ulcerative disorders of the gastrointestinal tract, involving principally the most proximal position of duodenum, the stomach, the lower end of the oesophagus, the jejunum after surgical anastomosis to stomach or rarely the ileum adjacent to the Meckel's diverticulum due to ectopic gastric epithelium.

Epidemiology:

Peptic ulcers are remitting relapsing lesions, at one time duodenal ulcers were much more common than gastric ulcer, but their incidence and prevalence are now approaching those of gastric ulcers.

Most often diagnosed in middle aged to older adults, but may first become evidence in young adult life. Male to female ratio for duodenal ulcer is about 3:1 and for gastric ulcers around 1.5:2.1. Women are most often affected at or after the menopause. Genetic influence plays some role in predisposition to both

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forms of ulcers, but more clear cut with the duodenal ulcers. Duodenal ulcers are three times more common in the first-degree relatives of ulcer patients than in general population.

An increased incidence of HLA-B₅ antigen has also been identified in white males with duodenal ulcers. Individuals with blood group 'O' are about 30% more likely to develop duodenal ulcer than those with other blood group.

Role of Helicobacter Pylori Infection in peptic ulcer:

Helicobacter pylori are a small spirally curved, gram negative, microaerophilic rod with multiple polar flagellae. 80–90% of populations are affected with infection of Helicobacter pylori. The incidence of infection within a population increases with age. The possibility of infection is inversely related to socioeconomic group.

Helicobacter pylori infection is the major cause of peptic ulcer not associated with the use of Non **steroidal** anti-inflammatory drugs. Human are the major reservoirs of Helicobacter pylori. The organism colonizes in the stomach, lodging most frequently in the antrum. The route of transmission of Helicobacter pylori

infection is mainly by faeco-oral route and oro-oral route.

Pathogenesis of Duodenal Ulcer due to Helicobacter Pylori:

Helicobacter pylori infection invariably results in chronic gastritis. The clinical result of this infection ranges from asymptomatic gastritis to peptic ulceration and gastric cancer. Helicobacter pylori colonizes in the gastric epithelium causing Type-B gastritis by which it reduces the resistance of gastric mucosa to attack by acid and pepsin resulting in gastric ulcer. Although, Helicobacter pylori normally reside in stomach, it also leads to causation of duodenal ulcers. This can be explained by the fact that antral Helicobacter pylori infection impairs the inhibitory feedback control of acid secretion, thus promoting duodenal ulcerogenesis by increasing duodenal acid load, resulting into duodenitis which leads to local inflammation, mucosal injury and eventually ulcer formation through the following mechanism:

1. By increasing acid secretion: One of the characteristic features of the organism is the production of urease enzymes, which hydrolyzes urea, resulting in production of ammonia, a strong

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alkali. Ammonia generated causes the release of gastrin (hypergastrinaemia) from antral G-Cells, which in turn leads to gastric acid hypersecretion.

2. By disrupting gastric mucous barrier.
3. By secretion of number of enzymes and chemicals, urease, catalase, mucin, lipase, phospholipase, porins, protease's, hemolysins and alkaline phosphatase.
4. By inducing inflammation in gastric epithelium (Wyatt and Dixon): The organism causes inflammation, which causes migration and degeneration of acute inflammatory cells, such as neutrophils and accumulation of chronic inflammatory cells, such as macrophages and lymphocytes.

Helicobacter pylori infection has also been implicated as a risk factor for gastric carcinoma and low-grade gastric lymphoma of mucosa associated with lymphoid tissue (MALT). Now WHO has described *Helicobacter pylori* as Class-I carcinogen.

Pathogenetic Factors Unrelated to *H. pylori* and NSAIDs in Acid Peptic Disease:

- Cigarette smoking
- Genetic predisposition. First-degree relatives of DU patients are three

times as likely to develop an ulcer.

Increased frequency in blood group O.

- Psychological Stress
- Diet
- Specific chronic disorders have been associated with PUD. Those with a strong association are (1) systemic mastocytosis, (2) chronic pulmonary disease, (3) chronic renal failure, (4) cirrhosis, (5) nephrolithiasis, and (6) antitrypsin deficiency.

Pathogenesis of Peptic Ulcer:

All peptic ulceration probably arises because of an imbalance between the aggressive action of acid pepsin secretion and the normal defenses of the gastro-duodenal mucosa.

For duodenal ulcer, the major causal influence appears to be exposure of the duodenal mucosa to excess amount of acid and pepsin.

For gastric ulcer, the major causal influence appears to be some breakdown in the gastric mucosal defenses against acid and pepsin.

The hyper-secretion is related to an abnormally large total mass of parietal cells in the gastric mucosa, perhaps to either increased responsiveness of the parietal cells to secretory stimuli of the parietal cells to

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secretory stimuli or lack of normal regulatory controls.

Increased levels of gastric or unusual sensitivity of the parietal cells to gastrin stimulation may also be involved.

Individual with total achlorhydria never develops a duodenal ulcer.

Defect in the defense mechanism includes deficiencies in mucosal cell removal, in mucous production in elaboration of bicarbonate and in production of prostaglandin.

Gastric Ulcer:³

- Pts. Usually thin and anemic with j-shaped hypotonic stomach
- Periodicity is less marked.
- Epigastric pain –boring / pricking
- Pain occurs almost immediately or any time up to 1and1/2 hrs. after meal as food irritates ulcers.
- Pain not felt in empty stomach.
- Food does not relieve pain (aggravates) pain is not felt in night.
- Vomiting is common after food it relives pain and may be self induced.
- Appetite – good. But patient afraid to eat.
- Patients avoid spicy and fried food.
- Weight loss is usual.
- Hemorrhage is less common.

Duodenal Ulcer:

- Healthy male with steer horn stomach.
- Periodicity is well marked (spring and autumn).
- Pain is more severe and spasmodic. Transpyloric plane about 1 inch to right of the midline. Pain starts 90 min to 3 hours after food.
- Pain is more in empty stomach. Pain that awakes patient from sleep (between midnight and 3 A.M) is the most discriminating symptom.
- Food and antacids relieves pain.
- Vomiting is rare.
- Good appetite eats frequently.
- Not avoid any thing.
- Weight gain.
- Hemorrhage is more common.

Complications of peptic ulcer:⁴

Acute Complication:

- a) Hemorrhage
- b) Perforation

Chronic Complication:

- a) Pyloric stenosis
- b) Teapot deformity
- c) Hour glass contracture of stomach
- d) Penetration into pancreas
- e) Carcinoma of stomach

Differential Diagnosis:

The list of gastrointestinal and non gastrointestinal disorders that can

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mimic ulceration of the stomach or duodenum is quite extensive.

The most commonly encountered diagnosis among patients seen for upper abdominal discomfort is NUD. NUD, also known as *functional dyspepsia* or *essential dyspepsia*, refers to a group of heterogeneous disorders typified by upper abdominal pain without the presence of an ulcer. Up to 60% of patients seeking medical care for dyspepsia have a negative diagnostic evaluation.

Several additional disease processes that may present with "ulcer-like" symptoms include proximal gastrointestinal tumors, Gastritis, gastroesophageal reflux, vascular disease, pancreaticobiliary disease (biliary colic, chronic pancreatitis), and gastroduodenal Crohn's disease.

Diagnostic Evaluation:

Documentation of an ulcer requires either a radiographic (barium study) or an endoscopic procedure.

Barium studies of the proximal gastrointestinal tract are still

commonly used as a first test for documenting an ulcer. The sensitivity of older single-contrast barium meals for detecting a DU is as high as 80%, with a double-contrast study providing detection rates as high as 90%.

Unfortunately, up to 8% of GUs that appear to be benign by radiographic appearance are malignant by endoscopy or surgery. Radiographic studies that show a GU must be followed by endoscopy and biopsy. Endoscopy provides the most sensitive and specific approach for examining the upper gastrointestinal tract. In addition to permitting direct visualization of the mucosa, endoscopy facilitates photographic documentation of a mucosal defect and tissue biopsy to rule out malignancy (GU) or *H. pylori*. Endoscopic examination is particularly helpful in identifying lesions too small to detect by radiographic examination, for evaluation of atypical radiographic abnormalities, or to determine if an ulcer is a source of blood loss

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Table No: 01: Tests for Detection of *H. pylori*:

Test	Sensitivity/Specificity, %	Comments
Invasive (Endoscopy/Biopsy Required)		
Rapid urease	80–95/95–100	Simple, false negative with recent use of PPIs, antibiotics, or bismuth compounds
Histology	80–90/>95	Requires pathology processing and staining; provides histologic information
Culture	—/—	Time-consuming, expensive, dependent on experience; allows determination of antibiotic susceptibility
Non-invasive		
Serology	>80/>90	Inexpensive, convenient; not useful for early follow-up
Urea breath test	>90/>90	Simple, rapid; useful for early follow-up; false negatives with recent therapy (see rapid urease test); exposure to low-dose radiation with ¹⁴ C test
Stool antigen	>90/>90	Inexpensive, convenient; not established for eradication but promising

Peptic Ulcer Disease: Treatment⁵

Before the discovery of *H. pylori*, the therapy of PUD was centered on the old dictum by Schwartz of "no acid, no ulcer." Although acid secretion is still important in the pathogenesis of PUD,

eradication of *H. pylori* and therapy/prevention of NSAID-induced disease is the mainstay of treatment. A summary of commonly used drugs for treatment of acid peptic disorders is shown in Table.

Table No: 02: Drugs Used in the Treatment of Peptic Ulcer Disease:

Drug Type/Mechanism	Examples	Dose
Acid-suppressing drugs Antacids	Mylanta, Maalox, Tums, Gaviscon	100–140 meq/L 1 and 3 h after meals and hs
H ₂ receptor antagonists	Cimetidine Ranitidine Famotidine Nizatidine	400 mg bid 300 mg hs 40 mg hs 300 mg hs

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Proton pump inhibitors	Omeprazole Lansoprazole Rabeprazole Pantoprazole Esomeprazole	20 mg/d 30 mg/d 20 mg/d 40 mg/d 20 mg/d
Mucosal protective agents Sucralfate Prostaglandin analogue Bismuth-containing compounds	Sucralfate Misoprostol Bismuth subsalicylate (BSS)	1 g qid 200 g qid

Table No: 03: Regimens Recommended for Eradication of *H. pylori* Infection:

Drug	Dose
Triple Therapy	
1. Bismuth subsalicylate <i>plus</i>	2 tablets qid
Metronidazole <i>plus</i>	250 mg qid
Tetracycline ^a	500 mg qid
2. Ranitidine bismuth citrate <i>plus</i>	400 mg bid
Tetracycline <i>plus</i>	500 mg bid
Clarithromycin or metronidazole	500 mg bid
3. Omeprazole (lansoprazole) <i>plus</i>	20 mg bid (30 mg bid)
Clarithromycin <i>plus</i>	250 or 500 mg bid
Metronidazole ^b <i>or</i>	500 mg bid
Amoxicillin ^c	1 g bid
Quadruple Therapy	
Omeprazole (lansoprazole)	20 mg (30 mg) daily
Bismuth subsalicylate	2 tablets qid
Metronidazole	250 mg qid
Tetracycline	500 mg qid

Surgical Therapy:

Surgical intervention in PUD can be viewed as being either elective, for

treatment of medically refractory disease, or as urgent/emergent, for the treatment of an ulcer-related

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complication. The development of pharmacologic and endoscopic approaches for the treatment of peptic disease and its complications has led to a substantial decrease in the number of operations needed for this disorder. Refractory ulcers are an exceedingly rare occurrence. Surgery is more often required for treatment of an ulcer-related complication.

Specific Operations for Duodenal Ulcers:

Surgical treatment is designed to decrease gastric acid secretion. Operations most commonly performed include (1) Vagotomy and drainage (by pyloroplasty, gastroduodenostomy, or gastrojejunostomy), (2) highly selective vagotomy (which does not require a drainage procedure), and (3) vagotomy with antrectomy. The specific procedure performed is dictated by the underlying circumstances: elective vs. emergency, the degree and extent of duodenal ulceration, and the expertise of the surgeon. Moreover, the trend has been toward minimally invasive and anatomy-preserving operations.

Specific Operations for Gastric Ulcers:

The location and the presence of a concomitant DU dictate the operative procedure performed for a GU. Antrectomy (including the ulcer) with a Billroth I anastomosis is the treatment of choice for an antral ulcer. Vagotomy is performed only if a DU is present. Although ulcer excision with vagotomy and drainage procedure has been proposed, the higher incidence of ulcer recurrence makes this a less desirable approach. Ulcers located near the esophagogastric junction may require a more radical approach, a subtotal Gastrectomy with a Roux-en-Y esophagogastrorjejunostomy (Csende's procedure). A less aggressive approach, including antrectomy, intraoperative ulcer biopsy, and vagotomy (Kelling-Madlener procedure), may be indicated in fragile patients with a high GU.

Surgery-Related Complications:

Complications seen after surgery for PUD are related primarily to the extent of the anatomical modification performed. In addition to the potential early consequences of any intra abdominal procedure (bleeding, infection, thromboembolism) other complications are

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1. Recurrent Ulceration
2. Afferent Loop Syndromes
3. Dumping Syndrome
4. Postvagotomy Diarrhea
5. Bile Reflux Gastropathy
6. Maldigestion and Malabsorption
7. Decreased serum vitamin B₁₂ levels can be observed after partial gastrectomy.
8. Iron-deficiency anemia
9. Malabsorption of vitamin D and calcium
10. Gastric Adenocarcinoma
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