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Dr. Bhavik Purohit
Associate Professor, Dept. of Practice of Medicine, JNHMC, Faculty of Homoeopathy, Parul University, Vadodara, Gujarat, India

Dr. Poorav Desai
Professor, Dept. of Anatomy, JNHMC, Parul University, Vadodara, Gujarat, India

Dr. Zankhana Desai
Professor, Dept. of Community Medicine, JNHMC, Parul University, Vadodara, Gujarat, India

Dr. Dhaval Jadav
Professor, Dept. of Obstetrics and Gynecology, JNHMC, Parul University, Vadodara, Gujarat, India

Dr. Ravi Patel
Assistant Professor, Dept. of Surgery, JNHMC, Parul University, Vadodara, Gujarat, India

Corresponding Author:
Dr. Bhavik Purohit
Associate Professor, Dept. of Practice of Medicine, JNHMC, Faculty of Homoeopathy, Parul University, Vadodara, Gujarat, India

A review on peptic ulcer

Dr. Bhavik Purohit, Dr. Poorav Desai, Dr. Zankhana Desai, Dr. Dhaval Jadav and Dr. Ravi Patel

Abstract
Ulcerations (sores) in the lining of upper part of the digestive tract are known as Peptic Ulcers. The ulcers may exist in the lower part of food pipe (esophagus), in the stomach or in the initial part of the intestine (duodenum). About 10% of all adults are affected with Peptic ulcers at some time in their life. The incidence of Peptic ulcers is more common in males as compared to females.

Keywords: Peptic ulcer, gastric ulcer, duodenal ulcer, esophageal ulcer

Introduction
A peptic ulcer is a distinct breach in the mucosal lining of the stomach (gastric ulcer) or the first part of the small intestine (duodenal ulcer), a result of caustic effects of acid and pepsin in the lumen. Histologically, peptic ulcer is identified as necrosis of the mucosa which produces lesions equal to or greater than 0.5 cm. It is the most common ulcer of an area of the gastrointestinal tract that is usually acidic and thus extremely painful. Helicobacter pylori is one of the most common causes of peptic ulcer. Ulcers can also be caused or worsened by drugs such as aspirin, ibuprofen, and other NSAIDs.

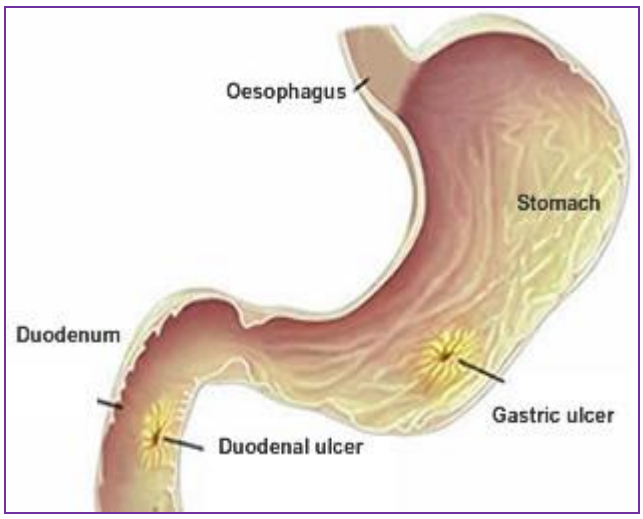

Fig 1: Sites of Peptic ulcer

Four times as many peptic ulcers arise in the duodenum - the first part of the small intestine, just beyond the stomach - as in the stomach itself. About 4% of gastric ulcers are caused by a malignant tumor, so multiple biopsies are needed to exclude cancer. Duodenal ulcers are generally benign.
The appearance of an ulcer can be either the classic erosive, concave, crater-like ulcer (the image held by most patients) or convex, perhaps resembling a colonic polyp. As a generalization, the erosive concave type tends to be located in the stomach proper while the convex type tends to be found in the pylorus/duodenum. These convex growths have an extensive variety of shapes and forms, but in all forms the ulcer projects above the level of the surrounding tissue. For extended periods these growths characteristically lack any surface breaks in the mucosic tissue and also initially lack any visual differentiation from the surrounding tissue even in larger sizes.

The surface integrity for this type ulcer enables growth over extended periods without the pain associated with crater ulcers. This site reference contains a series of convex ulcer photos. The images display a range from normal appearing growths to quite abnormal appearances. But notably, until the advanced growth stages non-crater, convex ulcers lack any surface cratering or break in the mucosa.

While these convex growths resemble a tumor to some extent, they are actually abnormal growths of the gastric tissue, and involve the mucosa, sub mucosa, muscularis, and serosa layers, not a pathogenic organism. However, these growths, like crater style ulcers, may metamorphic into pathogenic growths.

A peptic ulcer is a sore in the lining of your stomach or your duodenum, which is the first part of your small intestine. A burning stomach pain is the most common symptom of a peptic ulcer. The pain: - May come and go for a few days or weeks - May bother you more when your stomach is empty - Usually goes away after you eat Peptic ulcers happen when the acids that help you digest food in the stomach damage the walls of the stomach or duodenum. The most common cause is an infection with a bacterium called Helicobacter pylori. Another cause of peptic ulcers is the long-term use of non-steroidal anti-inflammatory medicines (NSAIDs) such as aspirin and ibuprofen. Stress and spicy foods do not cause ulcers, but can make them worse. Peptic ulcers will get worse if not treated. Treatment may include medicines to block stomach acids or antibiotics to kill ulcer-causing bacteria. Not smoking and avoiding alcohol can help.

The incidence of duodenal ulcers has dropped significantly during the last 30 years, while the incidence of gastric ulcers has shown a small increase, mainly caused by the widespread use of NSAIDs. The drop in incidence is considered to be a cohort-phenomenon independent of the progress in treatment of the disease. The cohort-phenomenon is probably explained by improved standards of living which has lowered the incidence of *H. pylori* infections.

Causes

- The main cause of ulcers is infection with bacteria called Helicobacter pylori. This bacterium is found in the saliva of infected people, and may also spread through mouth-to-mouth contact such as kissing or through exposure to the vomit of an infected person. This bacterium weakens the protective mucous coating of the esophagus, stomach and duodenum, which allows acid to get through to the sensitive lining beneath irritating it causing an ulcer.
- A major causative factor is (60% of gastric and up to 90% of duodenal ulcers) chronic inflammation due to Helicobacter pylori that colonizes the antral mucosa. The immune system is unable to clear the infection, despite the appearance of antibodies. Thus, the bacterium can cause a chronic active gastritis (type B gastritis), resulting in a defect in the regulation of gastrin production by that part of the stomach, and gastrin secretion can either be increased, or as in most cases, decreased, resulting in hypo- or achlorhydria. Gastrin stimulates the production of gastric acid by parietal cells. In *H. pylori* colonization responses to increased gastrin, the increase in acid can contribute to the erosion of the mucosa and therefore ulcer formation. Studies in the varying occurrence of ulcers in third world countries despite high *H. pylori* colonization rates suggest dietary factors play a role in the pathogenesis of the disease.
- Another major cause is the use of NSAIDs. The gastric mucosa protects itself from gastric acid with a layer of mucus, the secretion of which is stimulated by certain prostaglandins. NSAIDs block the function of cyclooxygenase 1 (*cox-1*), which is essential for the production of these prostaglandins. COX-2 selective anti-inflammatories (such as celecoxib or the since withdrawn rofecoxib) preferentially inhibit *cox-2*, which is less essential in the gastric mucosa, and roughly halve the risk of NSAID-related gastric ulceration. Abuse of nonsteroidal anti-inflammatory drugs such as aspirin, ibuprofen, etc. make the stomach weak to the harmful effects of acid and pepsin hence resulting in ulcers.
- Everyday life issues such as mental and physical stress, diet, cigarettes, foods and beverages containing caffeine, and alcohol.
- Heredity - patients with peptic ulcer often have a family history of the disease.
- Gastrinomas (Zollinger Ellison syndrome), rare gastrin-secreting tumors, also cause multiple and difficult-to-heal ulcers.

Etiology and pathophysiology

Peptic ulcer disease encompasses gastric, duodenal, and esophageal ulcers, with common etiologies of *Helicobacterpylori* infection, NSAID use, and stress-related mucosal damage. Due to the more invasive involvement of the gastric muscularis mucosa, PUD differs from the more superficial acid-related disorders such as erosions and gastritis. Peptic ulcers are typically named for their anatomical location, such as gastric or duodenal ulcers, and increased gastric acid is the primary cause. For the purpose of this article, only etiologies of *H pylori* and NSAID-induced PUD will be discussed. In addition to these etiologies, other controversial environmental risk factors including cigarette smoking, psychological stress, caffeine intake, and alcohol ingestion may increase the risk for the development of PUD.

The disease process of PUD is multifactorial based on etiology and risk factors. Ulcers may occur with hyper secretion of hydrochloric acid and pepsin, causing an imbalance between gastric luminal factors and degradation in the defensive function of the gastric mucosal barrier. Mucosal defenses include mucus, secretion of bicarbonate, mucosal blood flow, and epithelial cell defense. When acid and pepsin invade a weakened area of the mucosal barrier, histamine is released. Histamine will stimulate parietal cells to secrete more acid. With the continuation of this vicious cycle, erosion occurs to form the ulcer.

Although over 50% of the population has chronic *H pylori* infection, only 5% to 10% develop ulcers. *Hpylori* is a pH-sensitive bacterium that can infiltrate the gastric mucosal layer to reside in a neutral-pH environment. Acutely, the infection or colonization may ironically produce a hypochlorhydric environment. It is thought that this protective mechanism for the organism occurs due to the increase of urease, which hydrolyzes urea and converts it to ammonia and carbon dioxide. *H pylori* contribute to mucosal injury by multiple mechanisms.

Ulcers induced by nonselective NSAIDs can occur due to a topical irritation of the gastric epithelial cells and reduced protective prostaglandin synthesis. Due to their pharmacologic properties, many acidic NSAIDs cause alterations in the hydrophobic mucosal gel layer. The topical irritation may be the first insult to injury; however, inhibition of cyclooxygenase (COX) is the greatest concern. NSAIDs inhibit the rate-limiting enzyme in the conversion of arachidonic acid to prostaglandins. COX-2 exists throughout the body, producing prostaglandins associated with inflammation and pain, whereas COX-1 is located in the stomach, kidney, intestines, and platelets. Isoforms COX-1 and COX-2 are inhibited by nonselective NSAIDs. As a result of COX-1 inhibition, adverse effects such as ulcers or GI bleeds may occur.

Types of peptic ulcers

Peptic ulcers usually run a chronic course and complaints tend to be episodic in nature; the patient is usually free from the symptoms in between these episodes.

Classification - By region/area

- Duodenum (called duodenal ulcer)
- Esophagus (called esophageal ulcer)
- Stomach (called gastric ulcer)
- Meckel's diverticulum (called Meckel's diverticulum ulcer; is very tender with palpation)

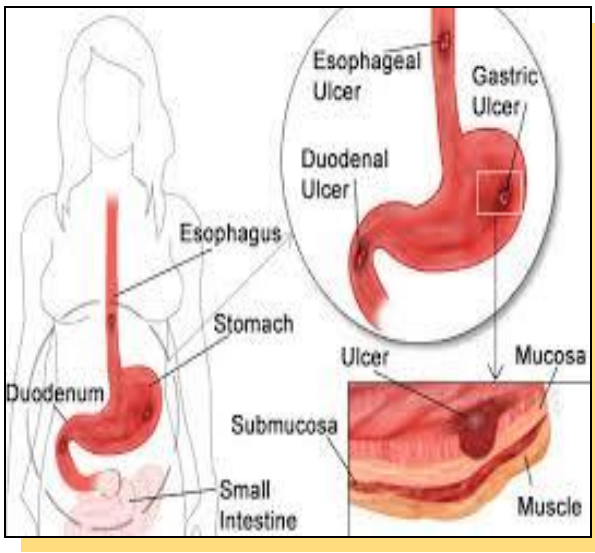

Fig 2: Types of Peptic ulcer

Modified Johnson classification of peptic ulcers

- **Type I:** Ulcer along the body of the stomach, most often along the lesser curve at incisura angularis along the locus minoris resistantiae. Not associated with acid hypersecretion.
- **Type II:** Ulcer in the body in combination with duodenal ulcers. Associated with acid over secretion.
- **Type III:** In the pyloric channel within 3 cm of pylorus. Associated with acid over secretion.
- **Type IV:** Proximal gastroesophageal ulcer
- **Type V:** Can occur throughout the stomach. Associated with chronic use of NSAIDs

Symptoms of peptic ulcer disease

- Pain in the epigastrium (upper part of abdomen). Abdominal pain, classically epigastric strongly correlated to mealtimes. In case of duodenal ulcers the pain appears about three hours after taking a meal
- Hunger pain- worsening of pain when the stomach is empty
- Night pain- wakes the patient from sleep
- Bloating and abdominal fullness
- Heartburn
- Water brash (rush of saliva after an episode of regurgitation to dilute the acid in esophagus - although this is more associated with gastro esophageal reflux disease)
- Nausea, and copious vomiting;
- Loss of appetite and weight loss
- Hematemesis (vomiting of blood); this can occur due to bleeding directly from a gastric ulcer, or from damage to the esophagus from severe/ continuing vomiting.
- Melena (tarry, foul-smelling feces due to presence of oxidized iron from hemoglobin)

Rarely, an ulcer can lead to a gastric or duodenal perforation, which leads to acute peritonitis, extreme, stabbing pain, and requires immediate surgery.

A history of heartburn, gastroesophageal reflux disease (GERD) and use of certain forms of medication can raise the suspicion for peptic ulcer. Medicines associated with peptic ulcer include NSAIDs (non-steroid anti-inflammatory drugs) that inhibit cyclooxygenase, most glucocorticoids

In patients over 45 with more than two weeks of the above symptoms, the odds for peptic ulceration are high enough to warrant rapid investigation by esophagogastroduodenoscopy.

Diagnosis of peptic ulcers is confirmed by

- Endoscopy to evaluate ulcers
- Biopsy may be required
- Barium meal (double contrast)
- Stool examination
- Complete blood count

The diagnosis is mainly established based on the characteristic symptoms. Stomach pain is usually the first signal of a peptic ulcer. In some cases, doctors may treat ulcers without diagnosing them with specific tests and observe whether the symptoms resolve, thus indicating that their primary diagnosis was accurate.

Confirmation of the diagnosis is made with the help of tests such as endoscopies or barium contrast x-rays. The tests are typically ordered if the symptoms do not resolve after a few weeks of treatment, or when they first appear in a person who is over age 45 or who has other symptoms such as weight loss, because stomach cancer can cause similar symptoms. Also, when severe ulcers resist treatment, particularly if a person has several ulcers or the ulcers are in unusual places, a doctor may suspect an underlying condition that causes the stomach to overproduce acid.

An esophagogastroduodenoscopy (EGD), a form of endoscopy, also known as a gastroscopy, is carried out on patients in whom a peptic ulcer is suspected. By direct visual identification, the location and severity of an ulcer can be described. Moreover, if no ulcer is present, EGD can often provide an alternative diagnosis.

One of the reasons that blood tests are not reliable for accurate peptic ulcer diagnosis on their own is their inability to differentiate between past exposure to the bacteria and

current infection. Additionally, a false negative result is possible with a blood test if the patient has recently been taking certain drugs, such as antibiotics or proton pump inhibitors.

The diagnosis of Helicobacter pylori can be made by

- Urea breath test (noninvasive and does not require EGD)
- Direct culture from an EGD biopsy specimen; this is difficult to do, and can be expensive. Most labs are not set up to perform H. pylori cultures
- Direct detection of urease activity in a biopsy specimen by rapid urease test
- Measurement of antibody levels in blood
- Stool antigen test
- Histological examination and staining of an EGD biopsy.

The breath test uses radioactive carbon atom to detect H. pylori. To perform this exam the patient will be asked to drink a tasteless liquid which contains the carbon as part of the substance that the bacteria breaks down. After an hour, the patient will be asked to blow into a bag that is sealed. If the patient is infected with H. pylori, the breath sample will contain radioactive carbon dioxide. This test provides the advantage of being able to monitor the response to treatment used to kill the bacteria.

If a peptic ulcer perforates, air will leak from the inside of the gastrointestinal tract (which always contains some air) to the peritoneal cavity (which normally never contains air). This leads to "free gas" within the peritoneal cavity. If the patient stands erect, as when having a chest X-ray, the gas will float to a position underneath the diaphragm. Therefore, gas in the peritoneal cavity, shown on an erect chest X-ray or supine lateral abdominal X-ray, is an omen of perforated peptic ulcer disease.

Macroscopic appearance

Gastric ulcers are most often localized on the lesser curvature of the stomach. The ulcer is a round to oval parietal defect ("hole"), 2 to 4 cm diameter, with a smooth base and perpendicular borders. These borders are not elevated or irregular in the acute form of peptic ulcer, regular but with elevated borders and inflammatory surrounding in the chronic form. In the ulcerative form of gastric cancer the borders are irregular. Surrounding mucosa may present radial folds, as a consequence of the parietal scarring.

Microscopic appearance

A gastric peptic ulcer is a mucosal defect which penetrates the muscularis mucosae and lamina propria, produced by acid-pepsin aggression. Ulcer margins are perpendicular and present chronic gastritis. During the active phase, the base of the ulcer shows 4 zones: inflammatory exudate, fibrinoid necrosis, granulation tissue and fibrous tissue. The fibrous base of the ulcer may contain vessels with thickened wall or with thrombosis.

Differential diagnosis

- Gastritis
- Stomach cancer
- Gastroesophageal reflux disease
- Pancreatitis
- Hepatic congestion
- Cholecystitis
- Biliary colic
- Inferior myocardial infarction
- Referred pain (pleurisy, pericarditis)
- Superior mesenteric artery syndrome

Complications

- Hemorrhage - Gastrointestinal bleeding is the most common complication. Sudden large bleeding can be life-threatening. It occurs when the ulcer erodes one of the blood vessels, such as the gastroduodenal artery.
- Perforation of ulcer - Perforation (a hole in the wall of the gastrointestinal tract) often leads to catastrophic consequences if left untreated. Erosion of the gastro-intestinal wall by the ulcer leads to spillage of stomach or intestinal content into the abdominal cavity. Perforation at the anterior surface of the stomach leads to acute peritonitis, initially chemical and later bacterial peritonitis. The first sign is often sudden intense abdominal pain; an example is Valentino's syndrome, named after the silent-film actor who experienced this pain before his death. Posterior wall perforation leads to bleeding due to involvement of gastroduodenal artery that lies posterior to the 1st part of duodenum.
- Perforation and penetration are when the ulcer continues into adjacent organs such as the liver and pancreas.
- Gastric outlet obstruction is the narrowing of pyloric canal by scarring and swelling of gastric antrum and duodenum due to peptic ulcers. Patient often presents with severe vomiting without bile.
- Cancer is included in the differential diagnosis (elucidated by biopsy), *Helicobacter pylori* as the etiological factor making it 3 to 6 times more likely to develop stomach cancer from the ulcer.

Prevention

Proper lifestyle modifications, diet and regimen measures shall help in prevention peptic ulcer formation. It also helps in speedy recovery and arrests the further progression of complications. Dietary and regimen measures that should help are as follows:

DO's

- The food should be masticated well and swallowed.
- The food should be taken in a relaxed manner, without any hurry.

To take

- Easily digestible, oil free diet so that the work load of stomach and intestine can be reduced.
- Frequent small meals should be encouraged to utilize the acid load in the stomach.
- Water is to be taken before and after meals.
- Nutritious diet should be taken at regular intervals or at regular time.
- Diet rich in fiber, like green vegetables and fruits which are good for digestion and also for the general health should be consumed regularly.
- Foods containing flavonoids, like apples, celery, cranberries (including cranberry juice), onions, and tea may inhibit the growth of H. pylori.

To avoid

- Stressful and hurried life.
- Habits of smoking, drinking alcohol, chewing tobacco and overeating which increases acid secretion of the stomach.
- Hunger - which denotes acid load, avoid it by taking small meals often.
- Full meal, oily diet, pickles and fatty snacks before bed time.
- Oily foods
- Food containing high spices, chilies and pepper.
- Dry bread, cakes and cookies.
- High fat content nuts and biscuits.
- Aerated drinks, coffee and tea.
- Milk products.
- Stress by the regular use of relaxation techniques such as yoga, tai chi, or meditation.
- Cabbage, onions, garlic, cauliflower, tomatoes - if not tolerated
- Drugs like NSAIDs - pain killers (for example. - aspirin)

Hence, through Homoeopathic treatment permanent relief from peptic ulcer can be achieved. Ulcers take time to heal, so medicines should be continued for long time, even if the pain goes away. Homoeopathy aids in eradicating the ulcer even without the need of surgery.

Homeopathic approach to peptic ulcers

Homeopathy considers peptic ulcers as a malfunction of one or more of our body systems. This is a result of a slow degenerative process due to the lack of adequate bodily supplies of the elements necessary for normal function and rejuvenation of affected organs. Homeopathic medicines have a remarkable healing property on the esophageal, gastric or duodenal mucosa, by counteracting the H. Pylori infection, reducing edema and the amount of acid that our stomach makes.

Homeopathic treatment is safe and gentle, without side-effects. Homoeopathy medicines are prescribed on the basis of physical, emotional, and genetic makeup that individualizes a person. This constitutional approach surrounding mind and body works at the root-level. Homoeopathy is very effective in managing all the symptoms of peptic ulcers and also plays an important role in preventing relapse of the condition and improving the general health of the person. Another outstanding thing about homeopathy is that people on multiple medications can safely take. Homeopathy offers some very good medicines for peptic ulcer disease. But for a homeopath, the symptoms of the disease are much more important than the ulcer itself. This is because to select the right medicine a homeopaths needs to differentiate between the finer presentations of a disease which, vary from person to person. A homeopath not only tries to heal the ulcer but also tries to remove the general predisposition to acquire it. Homeopathic remedies for peptic ulcers not only provide permanent relief from pain and other related symptoms, they also treat body's inability to fight with helicobacter pylori. Thus once treated with homeopathic remedies, peptic ulcers will be gone for good.

Homoeopathic treatment

Homoeopathy offers long-term cure instead of temporary relief as the treatment targets the root cause. Treatment is at the deeper level of immunity and it enhances the healing capacity of the body. Homoeopathy also helps prevent complications of Peptic ulcers. They can be treated successfully in Homeopathy without any side-effects. Homoeopathy improves the general resistance and also avoids recurrences.

Some of the commonly used medicines for gastritis and duodenal ulcers are argentum-nit, arsenic-alb, atropine, geranium, hydrastis, kali-bichrom, merc-cor, ornithogalum, phosphorus, uranium-nit, terebintha, lycopodium, pulsatilla, graphites, natrum-phos, medorrhinum etc.

There are a wide variety of homeopathic Medicines for peptic Ulcers, ranging from Uranium Nitricum, Kali Bich and Graphites to Crotalus Horridus, Geranium and Graphites.

Uranium Nitricum to be the ideal homeopathic remedy for gastric and duodenal ulcers characterized by boring pain in the pyloric region, a bloated abdomen, and a ravenous appetite followed by flatulence.

Kali Bich is best suited for round ulcers occurring in the stomach that lead to vomiting of bright yellow water and a load in the stomach immediately after eating, giving the patient the feeling that digestion has come to a standstill.

Homeopathic medicine Crotalus Horridus is helpful in cases of ulceration in the stomach due to chronic alcoholism. Patients who are unable to retain anything, exhibit violent vomiting or vomit blood benefit from this medicine.

Homeopathic remedy Graphites is good for treating duodenal ulcers marked by a burning in the stomach and a feeling of hunger.

Homoeopathic medicines commonly used for peptic ulcers are - Belladonna, Borax, Colocynth, Bryonia, Dulcamara, Nux Vom, Symphytum, Hydrastis, Arg Nit, Acid nit, Aethusa, Ars Alb., Geranium, Hydratis, Merc cor., Ornithogalum, Phos., Uranium nit., Terebintha, Graph., Pulsatilla, Sulphur, Kali Bich, Lycopodium, Carbo Veg, Antim crud, Calc carb, Med., Nat mur, Nat phos, China, etc. These medicines should be taken under the advice and diagnosis of a qualified Homeopath. Details of a few of the medicines are explained below and the remaining drugs too should be studied in a similar manner.

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