

ULCERS

(PEPTIC, GASTRIC, DUODENAL)

AN OVERVIEW

Compiled by

Campbell M Gold

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IMPORTANT

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General

Ulcers are 'sores' that frequently affect the stomach and the first part of the small intestine (duodenum).

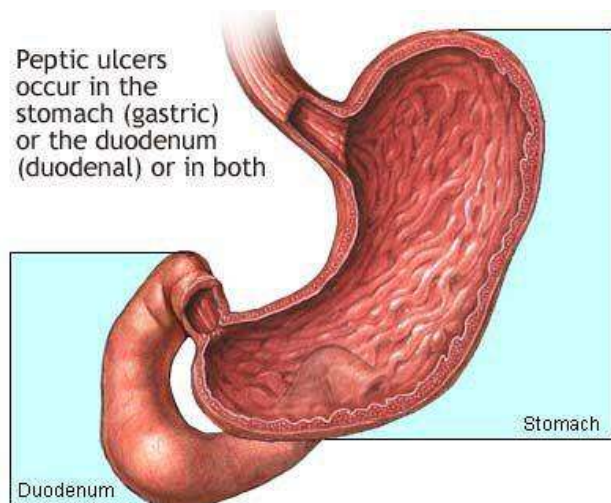
Bacterial infection is the most common cause of duodenal ulcers.

Stomach ulcers are often a side effect of pain killers and anti-inflammatory drugs used primarily to treat arthritis.

Alcohol ingestion, cigarette smoking, and emotional stress may also influence the development of an ulcer or may interfere with its healing.

Upper abdominal pain is the most common symptom of ulcers, but many ulcers cause no symptoms at all.

Ulcers may haemorrhage into the gastrointestinal tract; thus resulting in the passage of black (tarry) stool. Very serious ulcer disease may also cause a blockage between the stomach and small intestine, with this complication resulting in persistent vomiting. Severe pain



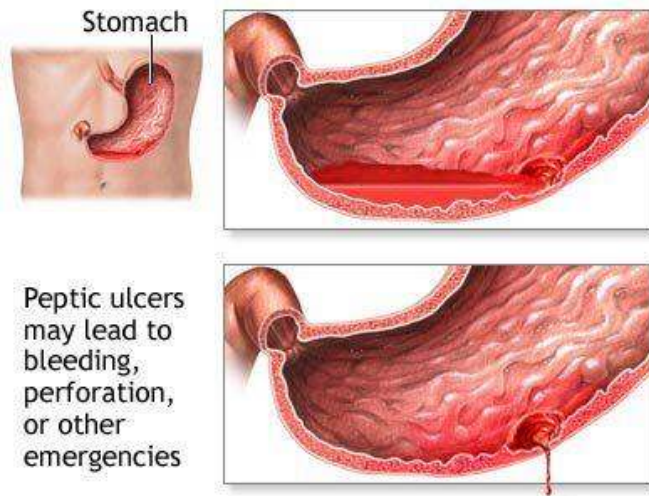
results from the most urgent complication of ulcers - peritonitis caused by a tear through the wall of the stomach or duodenum.

Almost all ulcers can be treated successfully, usually without surgery.

Ulcer treatments include antibiotics, agents that neutralise gastric acid or reduce its secretion, and drugs that strengthen the resistance of the stomach and duodenum.

Definition

Ulcer - a circumscribed excavation of the gastric or duodenal mucosal wall that penetrates the muscularis mucosae and exposes it to acid and pepsin.

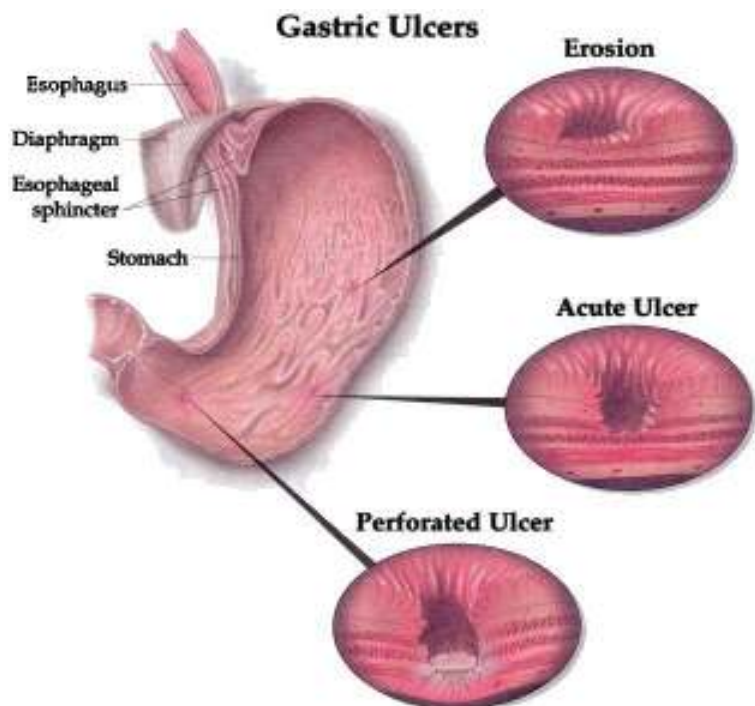


Peptic ulcers may lead to bleeding, perforation, or other emergencies

Causes and Incidence

The precise etiologic mechanisms of peptic ulcer formation are unclear. However, recent research points to infection by *Helicobacter pylori* bacteria as the major factor in ulcer formation. Other factors that have been implicated include use of certain drugs (e.g., aspirin and other nonsteroidal antiinflammatory drugs (NSAIDs – e.g. ibuprofen); use of alcohol, cigarettes, and caffeine; and a familial history of ulcers.

Those at risk of a duodenal ulcer have increased acid production and type O blood. Risk factors for a gastric ulcer include type A blood and underlying disease processes such as pancreatitis, gastritis, and hepatic disorders. About 80% of all peptic ulcers are duodenal in origin, and the remaining 20% are gastric. Gastric ulcers strike men and women equally, with the peak incidence occurring between ages 55 and 65. There are 40,000 to 80,000 cases reported annually. Duodenal ulcers occur in men two to three times as often as in women, and the incidence increases with age. The annual incidence is 200,000 to 300,000, but it has been steadily decreasing since the 1950s.



(Picture right – Benign gastric ulcer)

Disease Process

The pathology is unclear, but it is hypothesized that *H. pylori* or other factors may upset the balance between ulcer-promoting factors, such



as secretion of acid and pepsin and factors that serve as protectors of the mucosal lining, such as mucus production and replacement of damaged mucosal cells. This sets up an inflammatory process, with resultant ulceration, thrombosis, fibrosis, and scarring of the muscularis mucosae layer of the stomach or duodenum.

Symptoms

Manifestations vary with location, and ulcers are often asymptomatic or associated with vague symptoms. Only about 50% of individuals have a characteristic pattern of symptoms. The characteristic pain is described as burning, gnawing, or aching and is located in a well-circumscribed epigastric area. In duodenal ulcers the pain usually appears midmorning, is relieved by food, and then reappears 2 to 3 hours after eating. It also awakens the individual 2 to 3 hours after falling asleep. It occurs daily for 1 week or longer and may then disappear without treatment. With a gastric ulcer the pain usually occurs after eating food, is located in the left midgastric area, and often radiates to the back. Epigastric pain occurs with an empty stomach. Pain in both instances is typically relieved by antacids or milk.

Potential Complications

Complications include hemorrhage and perforation of the stomach or duodenum, with resulting peritonitis and obstruction of the pylorus or gastric outlet.

(Picture right – Bleeding gastric ulcer)



Diagnostic Tests

Endoscopy/biopsy

To establish presence of ulcer and determine whether malignancy is present

Upper gastrointestinal series

May reveal ulcers overlooked on endoscopy

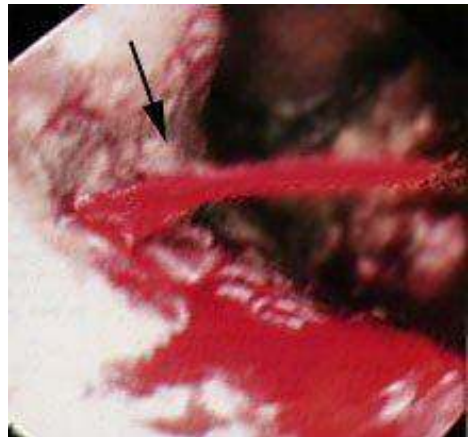
Gastric analysis

Increased output with duodenal ulcer; decreased or normal output with gastric ulcer

Carbon 13 urea breath test

Low levels of ^{13}C in exhaled breath indicative of *H. pylori* infection

(Picture right – Ulcer spurting blood)

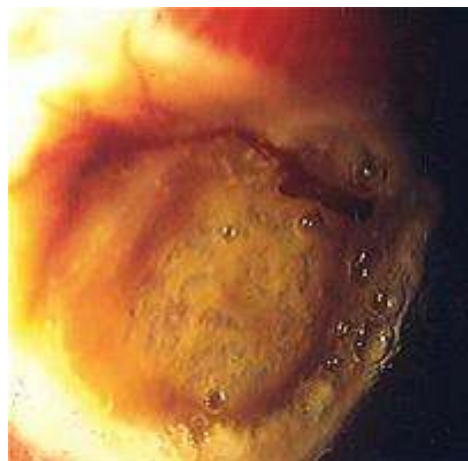


Treatments

Surgery - Gastrectomy, gastroduodenostomy, gastrojejunostomy to remove gastrin-producing portion of stomach in intractable cases with complications; fundic vagotomy with chronic duodenal ulcer disease

(Picture right – Stomach ulcer)

Drugs - Histamine receptor antagonists to block gastric acid output; antacids to reduce pain; cytoprotectives (Sucralfate) to form a protective coating in the base of the ulcer; bismuth preparations in combination with anti-infective drugs for *H. pylori* (not yet approved in all countries); omeprazole is a proton pump inhibitor in clinical trials in the United States;



prostaglandins are in clinical trials for treatment associated with NSAID use.

General - Avoidance of alcohol and tobacco products; avoidance of pepper, coffee (caffeinated and decaffeinated), and foods that cause epigastric distress; avoidance of Nonsteroidal AntiInflammatory Drugs (NSAIDs) (ibuprofen, etc).

End

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