

ENTITLEMENT ELIGIBILITY GUIDELINES

PEPTIC ULCER DISEASE

MPC	00912
ICD-9	533 - Peptic Ulcer - peptic unspecified
	532 - Duodenal Ulcer
	531 - Gastric Ulcer
	E943 - NSAID ulcer

DEFINITION

A peptic ulcer is a nonmalignant, mucosal lesion of the stomach or duodenum in which acid and pepsin play major pathogenic roles. The major forms of peptic ulcer are duodenal ulcer (DU) and gastric ulcer (GU).

For VAC purposes, entitlement for duodenal ulcer, gastric ulcer, or peptic ulcer includes entitlement for any benign ulcer of the stomach and/or duodenum.

DIAGNOSTIC STANDARD

Diagnosis from a qualified medical practitioner is required. Clinical diagnosis must be supported by appropriate history and physical examination data.

Upper gastrointestinal (UGI) x-ray or UGI endoscopy is often useful. However, a negative UGI report does not exclude the existence of an ulcer. The onset of peptic ulcer disease may be established when classical clinical symptoms manifest. A definitive diagnosis can be established by gastroscopic examination at a later date. Results from *Helicobacter pylori* (*H. pylori*) breath test and/or serology, if completed, should be submitted.

ANATOMY AND PHYSIOLOGY

There exists incomplete knowledge regarding the cause of peptic ulcer disease. Available information, however, supports a central role for *H. pylori* and a necessary role for acid and pepsin. Despite the focus on the role of infection with *H. pylori*, an understanding of basic gastric physiology remains central to a consideration of ulcer pathogenesis.

The normal gastric mucosa consists of several different secreting cells: endocrine (enterochromaffin-like [ECL]), gastric, chief, parietal, and mucous neck cells. These cells function together to secrete gastric juices to aid in the digestive process within the stomach. The hormone gastrin is secreted by the gastric cells. Gastrin is responsible for action on the ECL cells, which release histamine, the most important stimulant of gastric acid secretion. The chief cells secrete pepsinogen, which is converted to the active enzyme pepsin by hydrochloric acid secreted by the parietal cells. Mucous neck cells provide a protective barrier for the gastric lining by secreting mucous.

The two types of peptic ulcers discussed are duodenal and gastric, both located in the upper gastrointestinal tract.

Duodenal ulcers account for the majority of peptic ulcers. They develop when there is a disruption in the mucosal defence and the balance between acid-pepsin secretion.

In contrast to those affected by duodenal ulcer, gastric ulcer patients have normal acid secretory rates. The gastric ulcer is deep, penetrating beyond the mucosa of the stomach, and histologically similar to duodenal ulceration. Extensive involvement, however, of the surrounding tissue usually occurs more frequently in gastric than in duodenal ulcers. The majority of benign gastric ulcers are found immediately distal to the junction of the antral mucosa.

Smokers have a higher incidence of peptic ulcer disease. Smoking cessation is associated with recovery of gastric function within hours. Thus, once smoking has ceased, the increased risk of peptic ulcer disease is removed.

CLINICAL FEATURES

Some individuals with active duodenal ulcer have no ulcer symptoms. Thus, there is felt to be an underestimation of duodenal ulcer frequency.

Many signs and symptoms of peptic ulcer disease are obscure and confusing. Persons may complain only of indigestion or other vague dyspeptic symptoms commonly found in other conditions.

Chronic duodenal ulcer frequently presents with epigastric pain, sometimes situated more to the right of the epigastrium or central upper abdomen. The pain varies markedly in nature and intensity from being sharp and burning to aching or gnawing. Characteristically, the pain occurs from 90 minutes to 3 hours after eating and frequently awakens the person at night. The pain is usually relieved in a few minutes by food or antacid. Episodes of pain may persist for periods of several days to weeks or months. Although symptoms tend to be recurrent and episodic, duodenal ulcers often recur in the absence of pain. Periods of remission usually last from weeks to years and are almost always longer than the episodes of pain. In some persons the

disease is more aggressive with frequent and persistent symptoms, or development of complications. Vomiting of blood may occur.

On physical examination, epigastric tenderness is the most frequent finding, usually in the midline and often midway between the umbilicus and the xiphoid process.

In gastric ulcer, epigastric pain is the most common symptom, but the pattern is less characteristic than with duodenal ulcer. The pain may be precipitated or aggravated by food, and may or may not respond to antacids. Ulcers may heal and then recur. Vomiting of blood may occur. While nausea and vomiting almost always indicate gastric outlet obstruction in duodenal ulcer, these symptoms in a gastric ulcer may occur in the absence of mechanical obstruction. Gastric ulcers are associated with weight loss due to anorexia or aversion to food developing from the discomfort produced by eating.

H. pylori infection is associated with 90-95% of duodenal ulcers and 60-80% of gastric ulcers. Recognition of the role of *H. pylori* in the development of peptic ulcer disease has brought about substantial changes to the clinical treatment of people with peptic ulcer disease and has allowed a re-evaluation of the clinical approach to dyspepsia depending on whether or not a person has *H. pylori* in the upper GI tract.

H. pylori is associated with a greatly increased risk of duodenal and gastric ulceration. It is important to note, however, that while the infection is extremely common throughout the world and that approximately 50% of adults in developed countries are colonized by the age of 60 years, not all of these persons will develop or have had peptic ulcer disease. A number of studies have demonstrated that it is present in areas of overcrowding or highly infective areas.

A negative UGI x-ray report does not exclude the existence of an ulcer. If scarring or deformity is present on x-ray or gastroscopic findings, it is considered that peptic ulcer disease has been present for at least 3 to 4 years. A positive diagnosis can usually be established when classical symptoms are described.

NOTE:

It is well-recognized in the medical literature that early gastric carcinoma and gastric ulcer disease are difficult to differentiate. As a result, an initial diagnosis of gastric ulcer may be changed to gastric carcinoma, the cause of which differs from gastric ulcer disease.

PENSION CONSIDERATIONS

A. CAUSES AND/OR AGGRAVATION

THE TIMELINES CITED BELOW ARE NOT BINDING. EACH CASE SHOULD BE ADJUDICATED ON THE EVIDENCE PROVIDED AND ITS OWN MERITS.

1. *Helicobacter pylori* (H. pylori) prior to onset or aggravation

Objective confirmation of *Helicobacter pylori* is required.

2. Ingestion of non-steroidal anti-inflammatory drugs (NSAIDs) at time of clinical onset or aggravation

For NSAIDs to cause or aggravate peptic ulcer disease, signs/symptoms of peptic ulcer disease should develop during the NSAID therapy or within 30 days of cessation of the therapy.

Ingestion of NSAIDs is associated with a higher incidence of peptic ulcer disease.

3. Ingestion of oral corticosteroids at time of clinical onset or aggravation

For oral corticosteroids to cause or aggravate peptic ulcer disease, signs/symptoms of peptic ulcer disease should develop during the corticosteroid therapy or within 30 days of cessation of the therapy.

Minimum dosage levels and duration of treatment which can cause and/or aggravate ulcers varies among individuals, and according to their disease and other medications ingested. The risk of developing peptic ulcer disease while ingesting oral corticosteroids may be increased even further when NSAIDs are used concurrently.

4. Hepatic cirrhosis prior to clinical onset or aggravation

Persons with hepatic cirrhosis have an increased incidence of peptic ulcer disease.

5. Stress: aggravation only

For stress to aggravate peptic ulcer disease, increased signs/symptoms of peptic ulcer disease should develop during the period of stress and persist, on a recurrent or continuous basis, for a period of at least 6 months.

There is considerable medical research available on the impact of prolonged psychologic stress on visceral function and organic disease. It has not been possible to establish an association between stress and ulcer formation in controlled studies. Peptic ulcer disease is, however, often exacerbated, i.e. *temporarily worsened*, during or shortly after stressful life events such as occupational, financial and educational problems, divorce or marital separation, death, and family illness. The role of stress in aggravation, i.e. *permanent worsening*, is not settled; any association between psychologic factors and ulcer disease is likely to be complex and multifactorial. An individual's reaction to stress may determine whether a *permanent worsening* occurs. Aggravation, or permanent worsening, may be demonstrated by, but not limited to, the following:

1. Requirement for ulcer-specific medication;
2. Progression in frequency of use of ulcer-specific medication;
3. Requirement for surgery for peptic ulcer disease;
4. Development of complications, e.g. bleeding, outlet obstruction, dumping syndrome.

6. Zollinger-Ellison Syndrome

Zollinger-Ellison syndrome (gastronomas) can produce peptic ulcer disease through hypersecretion of gastric acid within the upper gastrointestinal tract, thereby disturbing the delicate acid-pepsin balance. **If peptic ulcer disease is caused by Zollinger-Ellison syndrome, entitlement should be sought for Zollinger-Ellison syndrome.**

7. Inability to obtain appropriate clinical management

B. MEDICAL CONDITIONS WHICH ARE TO BE INCLUDED IN ENTITLEMENT/ASSESSMENT

- Dumping syndrome (early or late)
Dumping syndrome is a complication of total or partial gastrectomy for peptic ulcer disease. The common form is early dumping syndrome. It is characterized by epigastric fullness or pain, cramps, nausea, palpitations, vertigo, extreme weakness, hot sensations, and cold sweats occurring immediately after eating. The late dumping syndrome is characterized by weakness, sweating, dizziness, fainting and mental confusion occurring one to three hours after eating. Both types may occur in the same person. Symptoms may be mild to incapacitating.
- Postvagotomy syndrome
Vagotomy with such drainage procedures as gastroenterostomy, pyloroplasty, or partial gastrectomy may give rise to abdominal distention, belching of foul smelling gas, diarrhea, nausea, vomiting, and evidence of gastric stasis. Though symptoms may disappear with time, gastric emptying time may be prolonged indefinitely.
- Bile reflux gastritis
Bile reflux gastritis is a complication of partial gastrectomy for peptic ulcer surgery. The reflux of bile from the small intestine into the stomach remnant results in nausea, bilious vomiting, and epigastric pain. The mere presence of bile is not evidence of bile gastritis. There must be objective evidence of bile reflux and of gastric inflammation, proven by endoscopy, along with significant symptomatology, to support a diagnosis of bile reflux gastritis.
- Hemorrhage
- Perforation
- Pyloric outlet obstruction

C. COMMON MEDICAL CONDITIONS WHICH MAY RESULT IN WHOLE OR IN PART FROM PEPTIC ULCER DISEASE AND/OR ITS TREATMENT

- Intestinal obstructions secondary to adhesions
- Hiatus hernia
Antrectomy and/or partial gastrectomy are surgical procedures for duodenal ulcer. In the course of these procedures the esophageal hiatus has to be dissected free in order to expose the vagus nerves. The interference with the esophageal hiatus may contribute to the development of hiatus hernia. Vagotomy, if involving esophageal dissection, may also contribute to the development of hiatal hernia. The time interval following surgery and the development of the hernia, and age, are factors in consideration of the degree of consequentiality. No specific recommendations in respect of the time interval can be made, but the hiatal hernia would generally occur within a few months of the surgery. There is an increased incidence of hiatus hernia with age.
- Gastroesophageal reflux disease
Interference with the esophageal hiatus during surgical procedures such as vagotomy may predispose to reflux esophagitis. The time interval between an operation and the development of esophageal reflux is a consideration in the degree of consequentiality. No specific recommendation in respect of the time interval can be made.
- Cholelithiasis
Physiological changes which occur in the gall bladder following truncal vagotomy for peptic ulcer disease may contribute to the formation of gall stones.
- Anemia if caused by peptic ulcer disease.
- Incisional hernias
- Osteoporosis
Total or partial gastrectomy may result in faulty absorption of essential food stuffs such as vitamins and mineral salts.

- Carcinoma of the stomach (in association with *H. Pylori* infection)
There is some epidemiologic evidence supporting an association between chronic *H. pylori* infection and gastric cancer (gastric adenocarcinoma and mucosa-associated lymphoid tissue (MALT) lymphoma). The International Agency for Research on Cancer has classified *H. pylori* as a Group I (definite) carcinogen in humans. However, the development of gastric cancer is felt to be multifactorial.
- Carcinoma of the stomach (in association with surgical treatment)
Persons who have undergone gastrectomy for benign disease are at increased risk of developing adenocarcinoma fifteen or more years after gastrectomy. This finding is particularly relevant in cases of subtotal or near total gastrectomies.

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