

# Peptic ulcer disease

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Peptic [ulcer](#) disease is a condition in which there are painful sores or ulcers in the lining of the stomach or the first part of the small intestine (the duodenum). Normally, a thick layer of mucus protects the stomach lining from the effect of its digestive juices.

A duodenal ulcer is a peptic ulcer that develops in the first part of the small intestine (duodenum). An esophageal ulcer occurs in the lower part of your esophagus.

## Etiology

### [Steroid side effects](#)

In 1932, [Harvey Cushing](#) described [peptic ulceration](#) secondary to raised [intracranial pressure](#) and attributed this to vagal overactivity, causing excess gastric acid secretion. Cushing ulcer remains a cause of morbidity in patients, albeit one that is preventable.

[Kumaria et al.](#) evaluate the [evidence](#) pertaining to the [pathophysiology](#) of neurogenic [peptic ulceration](#). A [literature review](#) suggests that the pathophysiology of Cushing ulcer may extend beyond vagal mechanisms for several reasons: (1) clinical and experimental studies have shown only a modest increase in gastric acid secretion in [head injury](#) patients; (2) increased vagal tone is found in only a minority of cases of [intracranial hypertension](#), most of which are related to catastrophic, nonsurvivable [brain injury](#); (3) direct stimulation of the [vagus nerve](#) does not cause [peptic ulceration](#), and; (4) Cushing ulcer can occur after [acute ischemic stroke](#), but only a minority of [strokes](#) are associated with raised [intracranial pressure](#) and/or increased vagal tone. The 2005 Nobel Prize in Medicine honored the discovery that [bacteria](#) play key roles in the pathogenesis of peptic ulcer disease. Brain injury results in widespread changes in the [gut microbiome](#) in addition to gastrointestinal inflammation, including systemic upregulation of proinflammatory cytokines. Alterations in the [gut microbiome](#) in patients with [severe traumatic brain injury](#) include [colonization](#) with commensal [flora](#) associated with peptic ulceration. The brain-gut-microbiome axis integrates the [central nervous system](#), the enteric nervous system, and the [immune system](#).

They propose a novel [hypothesis](#) that [neurogenic peptic ulcer](#) may be associated with alterations in the [gut microbiome](#), resulting in gastrointestinal [inflammation](#) leading to [ulceration](#) <sup>1)</sup>.

## Treatment

**Omeprazole** for Adults with peptic ulcers or gastroesophageal reflux disease (GERD) 20–40 mg PO daily. For Zollinger-Ellison syndrome: 20 mg PO q d to 120 mg PO TID (dose adjusted to keep basal acid output < 60 mEq/hr).

Side effects: N/V, H/A, diarrhea, abdominal pain, or rash in 1–5% of patients. Supplied: 10, 20 & 40 mg delayed-release capsules. Available OTC in 20.6 mg tablets as Prilosec OTC.

**Misoprostol** (Cytotec®), a **prostaglandin**, may be effective in mitigating **NSAID**-induced gastric erosion or **peptic ulcer**. Contraindicated in **pregnancy**. Rx 200 mcg PO QID with food as long as the patient is on NSAIDs. If not tolerated, use 100 mcg. ✖ CAUTION: an abortifacient. Should not be given to pregnant women or women of childbearing potential

1)

Kumaria A, Kirkman MA, Scott RA, Dow GR, Leggate AJ, Macarthur DC, Ingale HA, Smith SJ, Basu S. A Reappraisal of the Pathophysiology of Cushing Ulcer: A Narrative Review. J Neurosurg Anesthesiol. 2023 May 11. doi: 10.1097/ANA.0000000000000918. Epub ahead of print. PMID: 37188653.

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