Neurogenic peptic ulcer

- Determining the frequency of Helicobacter pylori in children with chronic kidney failure and its relationship to gastrointestinal symptoms
- A Reappraisal of the Pathophysiology of Cushing Ulcer: A Narrative Review
- CT-Guided Celiac Ganglion Block for Neurogenic Gastrointestinal Dysmotility
- A Systematic Review of the Clinical Use of Curcumin for the Management of Gastrointestinal Diseases
- Evaluation of the Gastroprotective Effect of the Flavonoid Complex from Lychnis chalcedonica L. on the Models of Experimental Ulcerogenesis
- Risks of Sulpiride-Induced Parkinsonism in Peptic Ulcer and Gastroesophageal Reflux Disease Patients in Taiwan: A Nationwide Population-Based Study
- Medicinal Plants in the Treatment of Peptic Ulcer Disease: A Review
- The gastroprotective action of acidic mineral water from the Azhyg-Sug source on the stress-induced injuries to the stomach of white rats

A neurogenic peptic ulcer is a type of peptic ulcer disease that is primarily caused by disturbances in the nervous system. Peptic ulcers are open sores that develop on the lining of the stomach (gastric ulcers) or the upper part of the small intestine (duodenal ulcers). They can be caused by multiple factors, including infection with Helicobacter pylori bacteria, long-term use of nonsteroidal anti-inflammatory drugs (NSAIDs), excessive acid production, and lifestyle factors.

Neurogenic peptic ulcers, however, are specifically associated with disruptions in the normal functioning of the nervous system. The nervous system plays a crucial role in regulating various digestive processes, including the production of stomach acid, the secretion of protective mucus, and the coordination of muscular contractions involved in digestion. Disruptions or abnormalities in the nervous system can lead to imbalances in these processes, making the stomach lining more susceptible to damage and ulcer formation.

Neurogenic peptic ulcers can be caused by several conditions, including:

Brain or spinal cord injuries: Traumatic injuries to the brain or spinal cord can disrupt nerve signals that regulate digestion, leading to an increased risk of developing peptic ulcers.

Central nervous system disorders: Certain central nervous system disorders, such as Parkinson's disease, multiple sclerosis, and autonomic neuropathy, can affect the normal functioning of the digestive system, increasing the likelihood of peptic ulcers.

Stress-related factors: Severe psychological stress, such as in critically ill patients or those with severe burns, can trigger a surge in stress hormones that affect the balance of stomach acid and increase the risk of ulcer formation.

The management of neurogenic peptic ulcers involves addressing the underlying cause, along with standard treatments for peptic ulcers. This typically includes medications to reduce stomach acid production (proton pump inhibitors or H2 receptor blockers), antibiotics to eradicate H. pylori infection (if present), and lifestyle modifications such as avoiding NSAIDs and managing stress levels. Additionally, treating the underlying nervous system disorder or injury is crucial in preventing recurrent ulcers.

It's important to consult with a healthcare professional for an accurate diagnosis and appropriate treatment plan if you suspect you have a peptic ulcer or if you have a known nervous system disorder

that may increase your risk of developing neurogenic peptic ulcers.

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.

Pathophysiology

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-injured 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. Alternations 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. Following the review of the literature, we propose a novel hypothesis that neurogenic peptic ulcer may be associated with alterations in the gut microbiome, resulting in gastrointestinal inflammation leading to ulceration [1)]

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.