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The Major Causes of Peptic Ulcer Disease (PUD) and its Diagnosis

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INTRODUCTION

The digestive system's stomach is the organ that transports food from the oesophagus to the small intestine, where it is further processed. It produces acid and a number of enzymes to break down food into simpler compounds. The interior wall of the stomach is protected from the acid and enzymes by a mucous layer. An imbalance between the digestive juices in the stomach and the various elements that help to protect its lining results in ulcers. One indication of an ulcer may be bleeding. In rare cases, an ulcer can completely erode the stomach wall. One of the main causes of stomach ulcers is the bacteria *Helicobacter pylori*. For ulcers brought on by this bacterium, treatment approaches frequently include antibiotics to eradicate the infection and drugs to reduce stomach acid.

DESCRIPTION

The first layer of the inner lining is where most ulcers occur. An opening in the stomach or duodenum is known as a hole. The stomach infection caused by the bacteria *Helicobacter pylori* (*H. pylori*) is the most common cause of ulcers. These bacteria are present in the digestive tract of the majority of people with peptic ulcers. However, many people whose stomachs contain these bacteria do not develop an ulcer. For low-risk cardiovascular patients, NSAIDs can be used with a Proton Pump Inhibitor (PPI), an H2 antagonist, or misoprostol to prevent peptic ulcer disease. The prevalence of ulcers may be decreased with NSAIDs of the COX-2 inhibitor type in comparison to non-selective NSAIDs. PPI is the most often employed medication for peptic ulcer prevention. Unfortunately, there is no proof that H2 antagonists can prevent gastrointestinal bleeding in NSAID users. While being helpful in avoiding peptic ulcers, misoprostol's use is constrained due to its propensity to promote abortion and cause gastrointestinal pain. For those who have a high risk of developing cardiovascular disease, naproxen plus PPI can be a helpful alternative. Other alternatives include low-dose aspirin, PPI, and celecoxib.

Helicobacter pylori are one of the primary causes of peptic ulcer disease. By secreting urease, it creates an alkaline environment that is beneficial for its existence. Because it expresses the blood group antigen-binding adhesin (BabA) and the outer inflammatory protein adhesin, it can adhere to the stomach epithelium (OipA). The bacterium also possesses virulence factors like CagA and PicB that help to cause stomach mucosal inflammation. The vacuolating cytotoxin produced by the VacA gene is not known to have a peptic ulcer-causing mechanism. Both hyper and hypochlorhydria have been associated to inflammation of the stomach mucosa. Inflammatory cytokines prevent the parietal cell from secreting acid. Additionally, *H. pylori* secretes substances that inhibit hydrogen potassium ATPase, start calcitonin quality related peptide tactile neurons, increase somatostatin emission to inhibit the production of corrosive by parietal cells, and stop the secretion of gastrin. This decrease in acid production leads to gastric ulcers. On the other hand, in 10% to 15% of *H. pylori* infections, duodenal ulcers are associated with an increase in acid production at the pyloric antrum. In this case, somatostatin production is reduced but gastrin production is increased, resulting in increased receptor discharge from the enterochromaffin cells and hence increased corrosive production [1-4].

CONCLUSION

The diagnosis is mostly made using the standard symptoms. The initial symptom of a peptic ulcer is typically stomach ache. Without completing particular tests, doctors can treat ulcers and watch to see whether symptoms get better, proving their initial diagnosis was accurate. Peptic ulcers specifically destroy the muscularis mucosae, typically to the level of the submucosa.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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