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Helicobacter pylori Prevalence in Peptic Ulcer Disease

Paulo Mandaric*

Department of Toxicology, Virtual University, Sau Paulo, Brazil

Commentary

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***For Correspondence:**
Paulo Mandaric, Department of Toxicology, Virtual University, Sau Paulo, Brazil

E-mail: Mandaric.paulo@gmail.com

DESCRIPTION

Peptic ulcer illness is now far less common than it was two decades ago because to the rapidly dropping prevalence of *Helicobacter pylori* infection and widespread use of powerful anti-secretory medicines. However, because of the concern of rising antimicrobial resistance worldwide and the widespread use of complicated anti-thrombotic medication in the ageing population, management has become more difficult than ever. Peptic ulcers that are not caused by *Helicobacter pylori* or the use of Non-Steroidal Anti-Inflammatory Medicines are increasingly producing significant diagnostic and treatment problems.

Helicobacter pylori infection is linked to both duodenal and gastric ulcer disease. An infected person's lifetime risk of developing peptic ulcer disease is estimated to be 10%-20%, which is at least 3-4 times higher than that of non-infected people. In 90%-100% of duodenal ulcer patients and 60%-100% of gastric ulcer patients, *H. pylori* infection can be detected. Duodenal ulcers are more likely in those who have been infected with a cytotoxin-producing bacterial strain or one that has the *cagA* gene. The amount of gastric acid produced (which is increased in duodenal ulcer disease and decreased in gastric ulcer disease), the existence of gastric metaplasia in the duodenal bulb, smoking and genetic variables is all factors that may influence peptic ulcer risk in infected patients. The probability of recurrence of ulcer disease is lowered to less than 10% for gastric ulcer disease and to less than 0% for duodenal ulcer disease when the infection is eradicated.

Our understanding of peptic ulcer illness has shifted dramatically since the discovery of *Helicobacter pylori*. Peptic ulcer disease is caused by bacterial, host and environment. Although the number of people with simple peptic

ulcers is decreasing, hospitalizations for ulcer complications caused by non-steroidal anti-inflammatory medicines (NSAIDs) are increasing. The use of highly selective cyclo-oxygenase-2 inhibitors and co-prescription of NSAIDs with powerful antiulcer medicines appears to minimize gastro duodenal ulceration. It remains to be seen whether these therapeutic breakthroughs will result in clinical advantages. One of the most contentious concerns in peptic ulcer disease is the interaction between *H. pylori* and NSAIDs. With the decline in *H. pylori* infection, the fraction of ulcers not caused by this bacterium and caused by NSAIDs has increased affecting peptic ulcer care.

The stomach and proximal duodenum are the most common sites for peptic ulcer disease. Infection with *Helicobacter pylori* and the use of nonsteroidal anti-inflammatory medicines are the most common causes in the United States. Epigastric discomfort (pain eased by food or antacids as well as pain that wakes you up at night or happens between meals), loss of appetite and weight loss are all symptoms of peptic ulcer disease. Patients above the age of 65 as well as those with alarm symptoms indicating a complication or cancer should have an endoscopy as soon as possible.

Patients who are taking Nonsteroidal Anti-Inflammatory Medicines (NSAIDs) should stop taking them. Based on the findings of H, a test-and-treat technique was devised. Pylori testing are indicated for younger individuals with no alarm signs. If *H. pylori* infection is found, it should be treated with antisecretory medication for four weeks (ideally with a proton pump inhibitor). Endoscopy should be recommended for those who have chronic symptoms. If complications arise or the ulcer does not respond to medicines, surgery is recommended. The most common reason for surgery is bleeding. Proton pump inhibitors and endoscopic treatment are used to treat most bleeding. Perforation and blockage of the gastric outlet are uncommon but significant consequences. Peritonitis is a surgical emergency that necessitates patient resuscitation, a laparotomy and peritoneal toilet, the implantation of an omental patch, and in certain cases like surgery.

The gastrointestinal condition of peptic ulcer disease is defined by mucosal damage caused by pepsin and gastric acid production. It is most prevalent in the stomach and proximal duodenum. It is less common in the lower oesophagus, distal duodenum or jejunum as in unopposed hyperse-cretory conditions such Zollinger-Ellison syndrome, hiatal hernias (Cameron ulcers) or ectopic gastric mucosa (e.g., in Meckel's diverticulum).

The discovery of effective and potent acid suppressants as well as the discovery of *F* is both linked to lower rates of peptic ulcer disease. The aetiology, pathology and therapy of peptic ulcer disease have all been rewritten since the discovery of *H pylori* infection. Over the last 25 years, there has been a revolution in the knowledge and therapy of peptic ulcer disease. Despite significant progress, this condition continues to be a significant clinical problem owing to the increased use of Non-Steroidal Anti-Inflammatory Medicines (NSAIDs) and low-dose aspirin.