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Risk Factors for Developing Peptic Ulcer Disease

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ABSTRACT

Peptic ulcer disease (PUD) is predicted to affect between 0.1 and 0.3 percent of the population each year. PUD develops at a rate of roughly 1% per year in Helicobacter pylori (H. pylori) infected persons, which is 6 to 10 times higher than in uninfected people. The prevalence of PUD increases with age in both duodenal and stomach ulcers. Two factors contribute to peptic ulcer disease: H. pylori infection and the use of nonsteroidal anti-inflammatory drugs (NSAIDs). NSAIDs have also been related to an increased risk of complications from peptic ulcer disease, such as gastrointestinal bleeding, perforation, and gastric outlet obstruction.

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INTRODUCTION

Peptic ulcers are flaws in the mucosa of the stomach or duodenum that extend through the muscularis mucosae. They form and remain in response to the acid-peptic action of gastric juice. Peptic ulcer disease is still a significant source of morbidity and health-care expenses. [1]

The natural course of a peptic ulcer ranges from no therapy to the development of complications with severe morbidity and death, including as bleeding and perforation.[2]

RISK FACTORS

The presence of Helicobacter pylori (H. pylori) or the use of nonsteroidal anti-inflammatory medicines (NSAIDs) is unlikely to be sufficient to cause ulcer development. Although H. pylori infection continuously causes gastritis and NSAIDs consistently block prostaglandin synthesis in the mucosa, the yearly incidence of clinical ulcer illness among at-risk people is only approximately 1% for both types of ulcer. Ulcer disease is caused by a variety of risk factors. [3]

The presence of Helicobacter pylori (H. pylori) or the use of nonsteroidal anti-inflammatory medicines (NSAIDs) is unlikely to be sufficient to cause ulcer development. Smoking is a risk factor for both symptomatic and asymptomatic peptic ulcer disease (PUD). The risk of PUD rises in direct proportion to the number of pack-years smoked. According to a population-based study, the prevalence of ulcer disease in smokers and former smokers is about double that of nonsmokers. When compared to never smoking, smoking more than 15 cigarettes per day raised the chance of a permeable ulcer by more than twofold. Furthermore, ulcers in smokers tend to be more difficult to cure and may have a greater recurrence rate. Ulcer disease is caused by a variety of risk factors. Tobacco use may be hazardous because it alters the balance of aggressive and defensive elements in the mucosa. However, after H. pylori has been eliminated, smoking does not appear to be a risk factor for ulcer recurrence. [4]

Host genetic variables appear to have a role in the susceptibility to H. pylori infection as well as the outcomes of illnesses such as duodenal ulcers (UD) and gastric cancer. Individual responses to H. pylori infection are accounted for by genes coding for cytokines. PUD is related with genetic differences in proinflammatory cytokines (e.g., IL-1B, IL-6, IL-8, and tumor necrosis factor (TNF)-alpha) and anti-inflammatory cytokines (IL-10). 5 A genome-wide association research conducted in Japan discovered that particular genetic polymorphisms in prostate stem cell antigen are linked to gastric ulcer, duodenal ulcer, and gastric cancer. EU in children has been connected to a genetic variation related to synergy between host (TNF-alpha promoter) and bacterial factors (triggered by interaction with epithelium or the iceA1 gene). [6]

There is also evidence of a genetic propensity to PUD that is unrelated to H. pylori infection. An example study indicated that monozygotic twins had a higher concordance of a self-reported ulcer history than dizygotic twins, regardless of whether they were reared together or apart. Correlations between twins and cross-traits in monozygotic and dizygotic twins revealed that genetic influences for peptic ulcer illness were independent of H. pylori genetic influences. Hyperpepsinogenemia with higher blood pepsinogen levels has also been seen in group A andn individuals with family

clustering of PUD. The genetic predisposition caused by a cytochrome P450 2C9 polymorphism can delay the metabolism of certain NSAIDs, resulting in a longer duration of the drug's impact and an increase in ulcerogenicity.[7]

Peptic ulcers have been linked to blood types O and A, the Lewis Le genotype (a + b-), and non-ABH secretors in particular. Other research, however, have found no link between blood type O and H. pylori infection or peptic ulcer. According to research, H. pylori strains from different parts of the world may have varying binding affinities for stomach epithelium, which explains the seemingly contradicting findings in blood group investigations from different nations. [8]

It has been proposed that dietary considerations explain part of the geographical diversity in ulcer disease, potentially due to toxins produced by food storage or the protective benefits of specific foods. [9]

There is little evidence to support the use of a bland diet or dietary restrictions to prevent PUD. Although some foods, drinks, and spices can cause dyspepsia, there is no conclusive evidence that they can create, maintain, or reactivate peptic ulcers. There is no evidence that coffee intake increases the risk of ulcer illness, while increased consumption may be linked to a higher likelihood of H. pylori infection. Coffee enhanced dyspepsia in participants with non-ulcer dyspepsia but not in those with ED when compared to controls in one study. [10]

Evidence points to a link between psychological variables and ulcer etiology. First, poorly tolerated stress or depression symptoms at baseline increase the chance of ulcer formation over the next 9 to 15 years, according to prospective research. Other psychological variables, such as job-related stress, social issues, and post-traumatic stress disorder, are also associated with later ulcer illness. Second, multiple studies have found that peptic ulcer complications are substantially more common during times of natural or societal calamity.[11]

The pathophysiological mechanisms behind the impact of stress on ulcer development are unknown. These effects might be mediated by changes in both behavior and physiology. A prospective population-based research of 3379 persons in Denmark found that psychological stress increased the incidence of peptic ulcer disease in part via altering health risk behaviors. Stress had the same effect on ulcers caused by H. pylori infection as it did on ulcers not caused by H. pylori or NSAID usage. Stress also raises acid secretion, although the effects are more pronounced in DU patients than in controls. As a result, not only the stressor, but also the individual's physiological and psychological reaction to stress, should be considered; harmful effects may be observed only in a subgroup of susceptible individuals. Endoscopic healing has also been shown to be affected by stress, worry, and depression, as well as the return of endoscopically identified ulcers. Stress appears to be reversible; patients who get an ulcer after painful life experiences but are psychologically stable likely to be well when the stress has subsided. Finding a link between psychological variables and ulcer illness, on the other hand, does not prove causality. Anxiety and neuroticism were elevated in a group of patients with early ED, but normalized in patients without relapse during a 10-year follow-up period, according to one research. In certain circumstances, psychological features may be the outcome of the illness process rather than the cause.[12]

Sleep problems may be a risk factor for ad ulcer disease, peptic ulcer complications, and ulcer recurrence. In large retrospective research that included 7,096 patients with sleep apnea and 28,384 age- and sex-matched controls, patients with sleep apnea were twice as likely as controls to suffer gastrointestinal bleeding over a four-year follow-up period, according to a rigorous analysis. In another study, women who reported sleeping more than nine hours per day had a lower incidence of ulcer illness than women who reported sleeping fewer than nine hours. Men showed a similar non-significant trend.[13]

Gastrointestinal bleeding is prevalent in COVID-19 patients, and the most common cause of gastrointestinal gas hemorrhage is peptic ulcer disease. Individuals' clinical presentations are identical to those of patients who do not have COVID-19 infections, and ulcers are detected in half of the patients. Overall mortality was high in a meta-analysis, at 19.1% (95% CI, 12.7% to 27.6%), while total death owing to gastrointestinal bleeding was 3.5% (95% CI, 1.3% to 9.1%). The probability of rebleeding was substantial, at 11.3% (95% confidence interval: 6.8%-18.4%). The majority of patients may be handled conservatively, with just one-third of those who had upper endoscopy requiring therapeutic intervention to control bleeding. In this cohort, mortality from gastrointestinal bleeding was modest, and the majority of deaths were caused by other complications of the illness rather than haemorrhage. [14]

Immune checkpoint inhibitors are becoming more used in cancer, however they have been linked to severe gastritis, gastric ulcers, and gastrointestinal bleeding. These ulcers and gastritis may not respond to proton pump inhibitor medication, and in severe instances, corticosteroid therapy is required. The incidence and risk factors are yet unknown.[15]

CONCLUSION

It is vital to identify individuals at risk of developing peptic ulcer disease in order to make prompt changes and create suitable care for each patient's particular situation.

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