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STRESS AS A RISK FACTOR OF ACID PEPTIC DISEASE

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ABSTRACT

Common causes of acid peptic disease include the bacteria Helicobacter pylori and non-steroidal anti-inflammatory drugs (NSAIDs). Other causes include tobacco smoking, stress due to serious illness, Behcet disease, Zollinger-Ellison syndrome, Crohn disease, and liver cirrhosis. This cross-sectional study was conducted in the outdoor department of Nishtar Hospital Multan. The relevant data i.e. name, age, gender, history of the disease, its relation with the stress, and duration of disease were collected on a predefined proforma after informed consent. This data was analyzed with SPSS version 23.0. The qualitative variables were presented as frequency and percentage. The quantitative variables were presented as mean and standard deviation. A total of 100 patients presenting with the symptoms were enrolled in this study. All the other patients were excluded from this study. The mean age of the patients was 32.45±3.89 years. There were 23 males and 73 females in this study. Out of 100 patients, 53 patients commented that the disease aggravates when they are under stress. Out of these 53, fourteen were smokers, two were alcoholics and twelve were also using NSAIDs.

KEYWORDS: Stress, Acid Peptic Disease.

INTRODUCTION

Peptic ulcer disease (PUD) is a break in the inner lining of the stomach, the first part of the small intestine, or sometimes the lower esophagus. An ulcer in the stomach is called a gastric ulcer, while one in the first part of the intestines is a duodenal ulcer. The most common symptoms of a duodenal ulcer are waking at night with upper abdominal pain and upper abdominal pain that improves with eating. With a gastric ulcer, the pain may worsen with eating. The pain is often described as a burning or dull ache. Other symptoms include belching, vomiting, weight loss, or poor appetite. About a third of older people have no symptoms. Complications may include bleeding, perforation, and blockage of the stomach. Bleeding occurs in as many as 15% of cases.

Common causes include the bacteria Helicobacter pylori and non-steroidal anti-inflammatory drugs (NSAIDs). Other, less common causes include tobacco smoking, stress due to serious illness, Behcet disease, Zollinger-Ellison syndrome, Crohn disease, and liver cirrhosis. Older people are more sensitive to the ulcer-causing effects of NSAIDs. The diagnosis is typically suspected due to the presenting symptoms with confirmation by either endoscopy or barium swallow. H. pylori can be diagnosed by testing the blood for antibodies, a urea breath test, testing the stool for signs of the bacteria, or a biopsy of the stomach. Other conditions that produce similar symptoms include stomach cancer, coronary heart disease, and inflammation of the stomach lining or gallbladder inflammation.

Diet does not play an important role in either causing or preventing ulcers. Treatment includes stopping smoking, stopping the use of NSAIDs, stopping alcohol, and taking medications to decrease stomach acid. The medication used to decrease acid is usually either a proton pump inhibitor (PPI) or an H2 blocker, with four weeks of treatment initially recommended. Ulcers due to H. pylori are treated with a combination of medications, such as amoxicillin, clarithromycin, and a PPI. Antibiotic resistance is increasing and thus treatment may not always be effective. Bleeding ulcers may be treated by endoscopy, with open surgery typically only used in cases in which it is not successful.[1-3]

MATERIAL OF METHODS

This cross-sectional study was conducted in the outdoor department of Nishtar Hospital Multan. The relevant data i.e. name, age, gender, history of the disease, its relation with the stress, and duration of disease were collected on a predefined proforma after informed consent. This data was analyzed with SPSS version 23.0. The qualitative variables were presented as frequency and percentage. The quantitative variables were presented as mean and standard deviation.

RESULTS

A total of 100 patients presenting with the symptoms were enrolled in this study. All the other patients were excluded from this study. The mean age of the patients was 32.45±3.89 years. There were 23 males and 73 females in this study. Out of 100 patients, 53 patients commented that the disease aggravates when they are under stress. Out of these 53, fourteen were smokers, two were alcoholics and twelve were also using NSAIDs.

DISCUSSION

Helicobacter pylori is one of the major causative factors of peptic ulcer disease. It secretes urease to create an alkaline environment, which is suitable for its survival. It expresses blood group antigen adhesin (BabA) and outer inflammatory protein adhesin (OipA), which enables it to attach to the gastric epithelium. The bacterium also expresses virulence factors such as CagA and PicB, which cause stomach mucosal inflammation. The VacA gene encodes for vacuolating cytotoxin, but its mechanism of causing peptic ulcers is unclear. Such stomach mucosal inflammation can be associated with hyperchlorhydria (increased stomach acid secretion) or hypochlorhydria (reduced stomach acid secretion). Inflammatory cytokines inhibit the parietal cell acid secretion. H. pylori also secretes certain products that inhibit hydrogen potassium ATPase; activate calcitonin gene-related peptide sensory neurons, which increases somatostatin secretion to inhibit acid production by parietal cells; and inhibit gastrin secretion. This reduction in acid production causes gastric ulcers. On the other hand, increased acid production at the pyloric antrum is associated with duodenal ulcers in 10% to 15% of H. pylori infection cases. In this case, somatostatin production is reduced and gastrin production is increased, leading to increased histamine secretion from the enterochromaffin cells, thus increasing acid production. An acidic environment at the antrum causes metaplasia of the duodenal cells, causing duodenal ulcers.

Human immune response toward the bacteria also determines the emergence of peptic ulcer disease. The human IL1B gene encodes for Interleukin 1 beta, and other genes that encode for tumour necrosis factor (TNF) and Lymphotoxin alpha also play a role in gastric inflammation. Taking nonsteroidal antiinflammatory drugs (NSAIDs) and aspirin can increase the risk of peptic ulcer disease by four times compared to non-users. The risk of getting peptic ulcer is two times for aspirin users. Risk of bleeding increases if NSAIDs are combined with selective serotonin reuptake inhibitor (SSRI), corticosteroids, antimineralocorticoids, and anticoagulants. The gastric mucosa protects itself from gastric acid with a layer of mucus, the secretion of which is stimulated by certain prostaglandins. NSAIDs block the function of cyclooxygenase 1 (COX-1), which is essential for the production of these prostaglandins. Besides this, NSAIDs also inhibit stomach mucosa cells proliferation and mucosal blood flow, reducing bicarbonate and mucus secretion, which reduces the integrity of the mucosa. Another type of NSAIDs, called COX-2 selective antiinflammatory drugs (such as celecoxib), preferentially inhibit COX-2, which is less essential in the gastric mucosa. This reduces the probability of getting peptic ulcers; however, it can still delay ulcer healing for those who already have a peptic ulcer.[4-7]

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