Peptic Ulcer Disease

A Literature Review of Peptic Ulcer Disease

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Peptic Ulcer Disease (PUD) is a disorder of the upper gastrointestinal tract. Ulcers occur when the mucosal lining of the GI tract breaks down, resulting in acute or chronic inflammatory response. Ulcers can develop in the esophagus, stomach, duodenum, or other regions of the GI tract. Based on hospitalization rates, the most common form of PUD is gastric, or stomach ulcers. The rate of PUD hospitalizations was found to be highest in adults > 65 years of age, Caucasians, and males. The prevalence decreased with age. PUD was once thought to be a result of emotional stress and diet. In 2005, Australian researchers Dr. Barry Marshall and Dr. Robin Warren were awarded the Nobel Prize for their 1982 discovery of the *Helicobacter pylori* bacteria (*H. pylori*) and its role in peptic ulcer disease. This revolutionary finding has allowed researchers to determine *H. pylori* as being the primary culprit of PUD, along with the use of non-steroidal anti-inflammatory drugs. Considering there are half a million new cases reported each year in the United States, physicians have developed many different ways to assess, diagnose, and treat common symptoms of PUD. With relatively recent discoveries regarding *H. pylori*, much is to be discovered about the disease and its etiology.

The upper GI tract is dependent upon the equilibrium between hostile factors that damage the mucous lining, like stomach acid, and protective factors, such as prostaglandins and mucus. When the hostile factors outnumber the natural defenses of the mucosa, ulcers form. Pepsin is an enzyme secreted by the mucosa in order to break down protein and hydrochloric acid is produced by parietal cells and released in the digestive process to help break down food. The corrosive actions of both pepsin and hydrochloric acid are significant contributors to ulcer formation. Along with these
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causes there are 2 other major risk factors for Peptic ulcers. These factors are *H. pylori* and the use of non-steroidal anti-inflammatory drugs (NSAIDs).

*H. pylori* is a gram-negative bacteria attached to gastric epithelial cells living within the gastric mucous layer. Transmission of the organism is most likely from person to person, either through oral or fecal contamination. Although the mechanism by which *H. pylori* leads to ulcers is not fully understood, scientists believe an infection may cause malfunction of acid secretion. They also believe the bacteria may cause chronic inflammation of the GI tract, resulting in weakening mucosa and allowing acid to form an ulcer in the mucosal lining. It is estimated that 92% of duodenal ulcers and 70% of gastric ulcers are caused by *H. pylori*. Even though *H. pylori* is a factor in a considerable number of cases, only 15% to 20% of individuals infected with *H. pylori* develop PUD in their lifetime. A study done from 1998 to 2005 showed a significant decline of the overall rate for *H. pylori* diagnosis. This suggests that a decrease in *H. pylori* infections may be partially responsible for the decrease in PUD hospitalizations.

Thanks to the scientific breakthrough by Dr. Marshall and Dr. Warren, PUD is no longer a chronic, disabling condition, but a disease that can be cured by a short regimen of antibiotics.

NSAIDs, short for non-steroidal anti-inflammatory drugs, are medicines that reduce pain, fever, and inflammation. NSAIDs offer many benefits, however, people who regularly take these medicines are 5 times more likely to develop PUD than people who do not take them. Cyclooxygenase-1 (COX-1) and cyclooxygenase-2 (COX-2) are enzymes that produce prostaglandins, which promote pain, inflammation, and fever. NSAIDs work by inhibiting these two enzymes. These medications often cause ulcers
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because COX-1 produces an additional type of prostaglandin that protects the stomach lining from stomach acid. By inhibiting COX-1, NSAIDs increase the risk of ulcers and GI bleeding by making the mucosal cells more vulnerable to hydrochloric acid and pepsin damage. The elderly population often suffers from musculoskeletal and joint disorders, which are commonly treated with NSAIDs. This explains why peptic ulcer bleeding is most common in adults > 65 years of age. Low-dose aspirin is also a cause of drug-induced peptic ulcer bleeding. Aspirin is used for the prevention of cardiovascular incidents. With the continuing rise of coronary and cerebrovascular diseases, the number of low-dose aspirin users may also increase, leading to more cases of PUD.

Lifestyle factors such as consumption of tobacco, alcohol, tea, coffee, and spicy foods are believed to stimulate gastric acid secretion, however, findings of epidemiological studies have been inconsistent. A Japanese study revealed that smokers were at higher risk of gastric and duodenal ulcers, compared to non-smokers. Yet, another study failed to confirm the association between PUD and use of tobacco. The inconsistent results from studies performed on the effect of lifestyle factors with PUD leads the evidence to be inconclusive. Although emotional stress is no longer though to be a cause of PUD, Physical stress may increase the risk of developing complications. People with injuries such as severe burns, spinal injuries, brain damage and people undergoing major surgery often require rigorous treatment to prevent ulcers from developing as a secondary condition.

When assessing a patient, typical symptoms of PUD to watch for include gnawing or burning gastric pain, pain occurring 2-5 hours after meals or on an empty stomach, and
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nocturnal pain. Food intake or antacids may relieve these symptoms. Less common features include indigestion, vomiting, loss of appetite, intolerance of fatty foods, and heartburn. Emergency Symptoms consist of severe stomach pain, bloody or black stools and bloody vomit. These deadly symptoms could be signs of bleeding, hemorrhaging, perforation or obstruction. Anyone younger than 55 years old, diagnosed with PUD, should be tested for \textit{H. pylori}. They should also be advised to discontinue the use of NSAIDs, tobacco, and alcohol. A food recall assessment should focus on the patient’s consumption of food that could potentially increase gastric acidity or foods that the patient cannot tolerate due to pain or gastric aggravation. Although routine laboratory tests usually are not helpful in patients with PUD, lab values to be cognizant of when assessing a patient are CBC (complete blood count), amylase, hemoglobin and lipase. CBC and hemoglobin can help detect anemia, which mandates early endoscopy to prevent GI blood loss.

According to the Academy of Nutrition and Dietetics’ Nutrition Care Manual, common nutritional diagnoses are Food-and nutrition-related knowledge deficit (NB-1.1), Inadequate oral intake (NI-2.1), Excessive oral intake (NI-2.2), and Undesirable food choices (NB-1.7). Other diagnoses frequently accompanied with PUD are hypertension, acute anemia, iron deficiency, diaphragmatic hernia, and \textit{H. pylori} infection. Diagnostic tests typically performed by a gastroenterologist to test for peptic ulcer include upper gastrointestinal barium x-ray, and endoscopy. Tests to confirm \textit{H. pylori} infection include gastric biopsy, urea breath test, \textit{H. pylori} culture, stool antigen test and a simple blood test.
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PUD is primarily treated using medication intervention.\(^1\) Medical treatment for an *H. pylori* infection includes 1-2 weeks of antibiotics with an antacid. Other medications such as Proton Pump Inhibitors, H2 blockers and mucosal protectants are used to treat PUD. A Swedish study conducted from 1974 to 2002 reported that the increase of proton pump inhibitors (PPI) has reduced the incidence of peptic ulcer complications.\(^12\) Administration of a H2 blocker or PPI for 4 weeks induces healing in most ulcers.\(^1\) PPIs are recommended as initial therapy for most PUD patients because they suppress acid, increase healing rates and relieve most symptoms. A research trial comparing PPIs with H2 blockers revealed that after 4 weeks PPIs provided earlier pain control and better healing rates.\(^18\) Another study revealed that PPIs healed duodenal ulcers in more than 95% of patients in 4 weeks and gastric ulcers in 80-90% of patients in 8 weeks.\(^19\) Based on these results, there is little reason to prescribe PPIs for longer than 4 weeks for duodenal ulcers or longer than 8 weeks for gastric ulcers, unless ulcers are unresponsive to initial treatment. Maintenance therapy with H2 blockers or PPIs prevents PUD recurrence in high-risk patients but is not generally recommended for patients in which *H. pylori* has been eliminated or who are taking short-term NSAIDs.\(^15\)

Surgery is rarely performed to treat PUD, due to the effectiveness of anti-ulcer medications like PPIs, H2 blockers and mucosal protectants. However, some patients do not respond to medication and may require surgery to treat serious complications such as GI hemorrhage, perforation, or gastric obstruction.\(^14\) When patients with duodenal ulcers require surgery, it is usually a vagotomy, vagotomy with antrectomy, or subtotal gastrectomy.\(^2\)
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Even though the evidence of lifestyle factors on PUD remains inconclusive, it is recommended to avoid certain habits such as smoking, alcohol use, and the consumption of high caffeine and spicy foods, in order to achieve optimal health. People who need the benefits of NSAIDs, and continue taking them may take steps to reduce the risk of ulcer occurrence. They can do this by taking the NSAID with a meal, using the lowest effective dose possible, and avoid smoking and alcohol.\textsuperscript{11} Patients suffering from painful PUD symptoms may consider eating smaller, more frequent meals and avoid eating before bedtime.\textsuperscript{1} A study of Traditional Iranian Medicine claims there are several edible fruits and spices used for the management of PUD. They found these remedies were effective in reducing inflammation, discouraging \textit{H. pylori} growth and healing wounds.\textsuperscript{20} Nevertheless, this holistic approach of managing PUD needs pharmaceutical and clinical verification of conclusive results. Goals for patients with PUD are to optimize nutritional intake to meet nutrient needs and implement dietary and lifestyle factors that will reduce symptoms, decrease pain, and promote healing.\textsuperscript{1}

The identification of \textit{H. pylori} as the causative agent in the majority of PUD cases has revolutionized the understanding and management of the GI disease.\textsuperscript{4} The overall incidence of peptic ulcers is declining, perhaps as a result of the increasing use of PPIs and decreasing rates of \textit{H. pylori} infection.\textsuperscript{21} This disease causing bacteria was identified as a major culprit only 20 years ago, which leads much to be discovered about the development of PUD. The use of surgery to treat PUD has also declined thanks to the widespread use of H2 blockers and PPIs.\textsuperscript{2} These highly effective anti-ulcer medications have been some of the most significant advances in the field of gastroenterology in the past 15 years.\textsuperscript{22} Researchers are searching for ways to give people the benefits of
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NSAIDs without the risk of gastrointestinal bleeding. Current research includes studies that; Compare the effectiveness of current medicines used to treat PUD and its complications, Develop new drugs to prevent ulcer development and complications, Identify GI-friendly alternatives to NSAIDs, and Improve understanding of how the mucosal lining can protect itself from stomach-acid erosion. Although PUD is primarily treated using medication intervention, it is important to assess patients thoroughly and treat using proper medical nutrition therapy. Goals for patients with PUD are to optimize nutritional intake to meet nutrient needs and implement dietary and lifestyle factors that will reduce symptoms, decrease pain, and promote healing.
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Citations


