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Helicobacter pylori Pathophysiology
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Introduction
Helicobacter pylori is a Gram-negative, microaerophilic bacterium found in the human intestines. Using urease as a pathogen occurs as adolescents by fecal-oral, gastric-oral, and oral-oral transmission. Nearly 50% of the population worldwide carries H. pylori, but only 15% of these individuals develop symptoms (Dionyssios et al. 2015). The pathogen is involved in the causation of gastritis, gastric ulcers, gastric adenocarcinoma, gastric cancer, and symptoms of the gastric lymphoid tissue. Worldwide the third leading cause of cancer deaths is gastric. H. pylori is considered a significant etiologic factor in gastric cancer. Lee et al. (2016) conducted a systematic review and meta-analysis to analyze the relationship between H. pylori infection and the incidence of gastric cancer. Lai et al. (2015) concluded that eradication of H. pylori is associated with better outcomes of gastric cancer.

As a future Nurse Practitioner, screening and prevention is a top priority. Understanding the pathophysiologic changes is crucial to preventing and managing diseases.

Pathophysiology
H. pylori is a helical shaped and has a shielded flagella which allows the bacteria to efficiently travel these tissues (Dionyssios et al. 2015). This pathogen virulence is assisted by secreting venoms VacA and CagA. VacA mediates mucosal cell apoptosis. CagA damages the columnar epithelial of the intestines. Additionally, the exotoxin promotes inflammation by stimulating the production of IL-8 which attracts neutrophils. The inflammatory response results in gastritis. CagA is associated with adenocarcinoma and mucosa-associated lymphoid tissue.

Chronic inflammation or ulcers has the potential to scar, obstructing flow between the duodenum and stomach. Lipopolysaccharides like the exterior, allowing the pathogen to adhere to cells and the intestinal wall (Dionyssios et al., 2015). Urac enzyme located on the xerous portion converts urea to carbon dioxide and ammonia to create a more alkaline environment. Digestion of food is reliant on the acidic environment created by the gastric glands. The alkaline environment, flagella and bile shape allows the pathogens to travel through the gastric layers. It adheres to the gastric epithelial cells, induces inflammation, apoptosis, and erodes the lining. The erosion of the stomach lining and cellular damage leads to ulcerations of the duodenum and stomach. Ulcers have the potential to erode blood vessels or cause a perforation.

Significance of Pathophysiology
H. pylori virulence and chronic inflammation have enormous health consequences. Considering the research, and health complications, providers have room to improve management and surveillance of the pathogens. Park et al. (2016) research aim to provide current evidence concerning the prevalence of H. pylori resistance to clarithromycin in the U.S. This quantitative research indicates >20% of H. pylori have developed resistance to Clarithromycin (MT 23-715 RNA). A mutation for 23S rRNA domain V was observed in 32.3% of cases (Park et al., 2016).

This study reconfirms the importance of adhering to the American College of Gastroenterology guidelines, to test, treat and retest. Markowski et al. (2017) examined physicians management of H. pylori. The research revealed that 59% of physicians confirmed eradication and 29% of physicians seek re-treatment eradication (Markowski et al., 2017). Kim et al. (2014) peer-reviewed retrospective study is to examine the role of H. pylori testing on the hospitalized patient with confirmed bleeding ulcers. U.S. and international guidelines recommend H. pylori testing on patients with bleeding ulcers. Researchers conclude less than half of patients were tested for H. pylori in hospitalized patients with bleeding ulcers. Providers need to increase surveillance to prevent associated disorders and significant health complications for patients.

• Gastritis main causative agent is H. pylori (Eida, H. et al., 2017)
• CagA stimulation of the inflammatory process contributes to gastritis.
• Pepsin Closer is closely associated with H. pylori (Eida, H. et al., 2017).

• Gastric cancer develops from glandular atrophy to chronic gastric disease in adults and children (8th edition) St. Louis, MO: Elsevier.

Helicobacter Pylori

Clinical Manifestations
• Abdominal pain (ache or burning) aggravated by empty stomach
• Fullness
• Weight loss, decreased appetite
• Vomiting
• Heartburn
• Shortness of breath
• Hematemesis
• Hematochezia
• Iron deficiency anemia
• Abdominal distension
• Eructation (burping)

Risk factors
• Living in crowded conditions or a dirty house
• Contaminated water or food sources
• Being in contact with stool, saliva, or blood on an infected person

Case Study
Mr. Smith is a 61-year-old male presents to Urgent Care with non-radiating intermittent epigastric pain, alleviated after eating and aggravated in the morning. nausea, hæmorrhage, recent unintentional weight loss of 8 lbs. in a month, decreased appetite. Patient reports history of duodenal ulcer and currently using with pump injection daily. Family history of gastric cancer.

Abdominal exam yields palpable bladder, active, soft, round, non-tender, no guarding, passing flatus, flat bowel movement today formed. Mr. Smith is initiated on Zofran for nausea, will continue on current PPI, educated to consume 6 small meals daily, add Ensure protein drinks between meals as tolerated, and referred to a Gastro- enterologist for further evaluation.

Evaluation
• Stomach biopsy is the gold standard, however the most invasive for diagnosis of H. pylori.
• Blood antibody test measures H. pylori IgG, specific antigen, with a sensitivity and specificity rage in 80-95%.
• Urea breath test is the least invasive exam, with a specificity of 90% and 95% sensitivity.

Implications for Nursing Care
Recognition, Diagnosis, and Surveillance:
• Advanced practice nurse is responsible for recognizing and diagnosing disease.

Education:
• A diet plan low in sugar, fat, irritating foods, caffeine, and alcohol.
• Adhering to medication
• Referral to local oncologist
• Referrals for eradication of the bacteria
• Symptom requiring further evaluation

Medical Management:
• Asymptomatic
• Clarithromycin
• Proton Pump Inhibitor Antacid

Conclusion
In summary, H. pylori virulence is determined by the immune response, pathogen characteristics, and the host environmental conditions. With the widespread prevalence of this pathogen, providers must be alert to the harmful effects. Providers are responsible for diagnosing disease, managing, and seeking out the source. A variety of disorders have a strong association to this pathogen. Providers must adhere to the American College of Gastroenterologist guidelines to test, treat, and retest. These efforts to evaluate the pathogens will prevent chronic disease, permanent damage, and death.

References