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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. Exposure to the pathogen often occurs as adolescents by fecal-oral, gastric-oral, and oral-oral transmission. Nearly 50% of the population worldwide carry the pathogen, only 30% of those develop symptoms (Dionyssios et al, 2015). The pathogen virulence is determined by the strain and characteristics. This microorganism stimulates an immune response leading to chronic inflammation. Potentially causing acute or chronic gastritis, peptic ulcers, gastric adenocarcinoma, gastric cancer, and lymphomas 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 examine the relationship between *H. pylori* eradication and the incidence of gastric cancer. Lee et al. (2016) concluded that eradication of *H. pylori* is associated with a lower incidence of gastric cancer.

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

Pathophysiology

H. pylori is helical shaped and has a sheathed flagella which allows the bacteria to efficiently travel thru the intestines (Dionyssios et al, 2015). This pathogen virulence is assisted by secreting exotoxins VacA and CagA.

VacA induces mucosal cell apoptosis. CagA damages the columnar epithelia 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 edema has the potential to scar, obstructing flow between the duodenum and stomach.

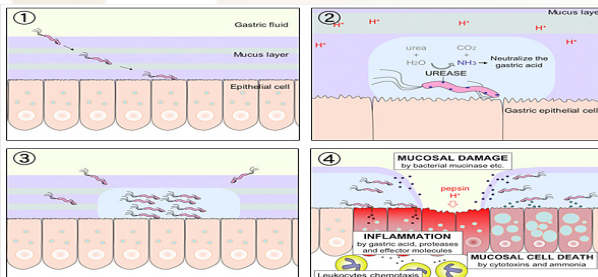
Lipopolysaccharides line the exterior, allowing the pathogen to adhere to cells and the intestinal wall (Dionyssios et al, 2015). Urease enzyme located on the pathogen's surface converts urea to carbon dioxide and ammonia to create a more alkaline environment.

Digestion of food is reliant on the acidic environment created by the goblet cells. The alkaline environment, flagella, and helical shape allow the pathogen to travel thru 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 pathogen.

Park et al. (2016) research aim to provide current evidence of 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 (MUT 23 rRNA). A mutation for 23S rRNA domain V was observed in 32.3% of cases (Park et al, 2016).



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This study reconfirms the importance of adhering to the American College of Gastroenterology guidelines, to test, treat and retest. Murakami et al. (2017) examined physicians management of *H. pylori*. The research revealed that 58% of physicians confirmed eradication and 26% "sometimes" confirm post-treatment eradication (Murkami et al, 2017).

Kim et al. (2014) peer-reviewed retrospective study is to examine the rate 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* (Eda, H. et al, 2017). CagA stimulation of the inflammatory process contributes to gastritis.
- Peptic Ulcer is closely associated with *H. pylori* (Eda, H. et al, 2017). The erosion of the mucosa layers and inflammatory process contribute to ulcer formation.
- Gastric cancer develops from glandular atrophy to chronic gastritis. *H. pylori* have a direct involvement and is recognized as the leading cause of gastric cancer (Ki et al, 2014).

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

Abdominal exam yields palpable bladder, active, soft, round, non-tender, no guarding, passing flatus, last 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 Gastroenterologist for further evaluation.

Case Study

Helicobacter Pylori



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Clinical

Manifestations

- Abdominal pain (ache or burning) aggravated by empty stomach
- Fullness or bloating
- Weight loss, decreased appetite
- Nausea
- Vomiting
- Heartburn
- Shortness of breath
- Hematochezia
- Hematemesis
- Iron deficiency anemia
- Abdominal distention
- Eructation (burping)

Risk factors

- Living in crowded conditions or a developing country
- Contaminated water or food sources
- Being in contact with stool, saliva, or blood on an infected person

Evaluation

- **Stomach biopsy** is considered the gold standard, however the most invasive for diagnosis of *H. pylori*.
- **Blood antibody test** measure *H. pylori* IgG specific antigens, with a sensitivity and specificity range is 80-95%.
- **Urea breath test** is the least invasive exam, with a specificity of 98% and a sensitivity of 97%.
- **Stool antigen test** is a noninvasive method for HpSA using an enzyme immunoassay.

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
 - Retesting for eradication of the bacterium
 - Symptoms requiring further evaluation
- **Medical Management:**
 - Amoxicillin
 - Clarithromycin
 - Proton Pump inhibitor
 - Antiemetic

Conclusion

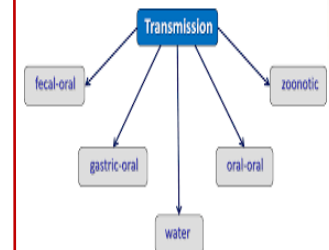
In summary, *H. pylori* virulence is determined by the immune response, pathogen characteristics, and the host environmental conditions. With the widespread prevalence of this pathogen, providers must be alert to the harmful effects.

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 eradicate the pathogen will prevent chronic disease, permanent damage, and death.

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Transmission of H. pylori Infection



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