Helicobacter Pylori

Opal. L. Caldwell

King University

Abstract

Helicobacter pylori is a very prevalent infection throughout the world affecting approximately fifty- percent of the world's population (CDC, 2009). What is H-pylori? How does it function on a cellular level? What role do inflammation and the immune system have in the H-pylori infection? What are the consequences of H-pylori infection? What risk factors are associated with the H-pylori infection? How is H-pylori infection identified and how are those infected with H-pylori treated? This paper is designed to identify the H-pylori infection, take an in depth look at the infection as a whole, and identify what effects it has on the human body.

Helicobacter Pylori

Helicobacter pylori is a very prevalent infection throughout the world affecting approximately fifty-percent of the world's population (CDC, 2009). It is linked to many upper gastrointestinal conditions such as chronic gastritis and duodenitis, gastric erosions, peptic ulcer disease, and gastric cancer. According to Banning, sources of transmission are overcrowded living conditions, seen in under developed countries, contaminated water supply, poor hygiene, poor waste disposal, dental plaques, and contaminated endoscopy scopes (Banning, 2012). H-pylori is transmitted from feces, salvia, and dental plaque of infected persons (2012). Person to person contact, kissing, and fecal ingestion, with poor hygiene of someone infected with H-pylori, aid with the spread of the infection (2012). H-pylori is usually transmitted in early childhood (McCance, Huether, Brashers, Rote, 2010, p.1465).

Cellular Level

"H-pylori is an enteric pathogen and classified as a unipolar, micro-aerobic, curved, gram negative bacterium" (Banning, 2012). It has four flagella which makes it mobile (2012). It has a helical shape, hence the name helicobacter, which enhances the ability to penetrate the gastric lining (2012). H-pylori produces 4 enzymes that help either protect the bacteria or enable it grow (2012).

The first two enzymes, catalase and urease, help protect the bacteria. Catalase enhances the ability to survive in oxygen- derived free radical environment, which is produced by phagocytes of the human immune system (2012). Urease degrades urea, produced in saliva and gastric mucosa, producing ammonia making the area more alkaline (McCance et al, 2010). The alkaline environment protects the bacteria. Once H-pylori colonizes the stomach it produces protease and phospholipases (Banning, 2012). These enzymes "disrupt the protective mucus

layer of the stomach" (2012, para 2). When the mucus layer of the stomach is disrupted gastric acid, produced in the stomach, is able to break down the epithelium tissue (2012). When the epithelium tissue starts to break down it causes gastric erosions, gastritis, and eventually peptic ulcer disease (2012).

What effects does H-pylori have on human cells? According to Wang et al. "H-pylori inhibits cell proliferation, promotes gastric carcinoma cell invasion, damages cell construction, destroys cell junction, promotes cell invasive ability and might accelerate malignant process and metastasis of gastric cancer" (2012, para. 4).

H-pylori effects the body systemically when it becomes more chronic. When gastritis forms irritating symptoms occur such as; nausea, abdominal pain, and vomiting. Other process related to infection, peptic ulcer disease, show symptoms of abdominal pain, malaise, and can lead to anemia, when ulcers begin to bleed. This will be further discussed under consequences of H-pylori.

Genetics

Genetics does not have a role in contracting H-pylori infection.

Circadian Rhythms.

Circadian rhythms are not affected by the H-pylori infection.

Inflammation.

When H-pylori invade the host, the host response is to "activate T and B lymphocytes with infiltration of neutrophils" (McCance et al., 2010, p.1464). The release of cytokines, tumor necrosis factor a (TNF-a), and interleukins, eight and ten, damage the host gastric epithelium (2010, p.1464). "H-pylori has a gene known as CagA (cytotoxin-associated gene) that produces a vacuolating toxin (VacA) that causes injury and promotes inflammation" (2010, pp.1465). VacA

is a major factor in the H-pylori infection (Delahay, Rugge, 2012). VacA is involved in various mechanisms of programmed cell death, apoptosis, and necrosis (2012). VacA targets host cell mitochondria and induces apoptosis through mitochondrial morphological dynamics (2012). As the inflammatory cells try to engulf the H-pylori bacteria, reactive- oxygen species are released to protect the bacteria and damage surrounding tissue (Banning, 2012). According to Sanchez et al, "The extent of mucosal injury is related to the degree of neutrophil infiltration" (2013, para.5).

Immune Response.

The body reacts to the presence of H-pylori by initialing an immune response (Banning, 2012). Macrophages and neutrophils respond to the site of the bacteria in the presences of colonization (2012). Macrophages will attempt to engulf and eradicate the bacteria (2012). The body also produces immunoglobulin/antibodies such as IgA and IgG, to neutralize the effects of H-pylori infection (2012). It is believed that the release of IgA neutralizes the effects of H-pylori. That is why 70% of patients are asymptomatic, even when they are positive for the infection (McCance et al., 2010, p.1465).

Stress Response.

Stress response does not have a role in contracting the H-pylori infection.

Neurotransmission problem.

H-pylori does not cause a neurotransmission problem.

Fluid and electrolyte imbalance.

H-pylori does not cause any imbalances with the bodies' fluid and electrolytes.

Consequences and Risk Factors of H-pylori infection.

H-pylori leads to a cascade of consequences when untreated, starting with irritating symptoms, leading to more severe consequences, such as, bleeding ulcers and gastric cancer.

Symptoms associated with acute gastritis can include abdominal pain, and epigastric tenderness (McCance et al., 2010, p.1463). If acute gastritis persist, it turns into chronic gastritis. Symptoms of chronic gastritis may be vague and "include anorexia, fullness, nausea, vomiting, and epigastric pain" (2010, p.1464).

Risk factors associated with H-pylori infection are under- developed countries, children most likely due to poor hand hygiene, infected family members, and poor sanitation (Elfant, 2012). Modes of transmission being person- to- person via kissing or passage of saliva with drinking after an infected person, via oral-oral, fecal-oral, and gastric-oral (2012).

Most patients contract H-pylori as a child. According to Banning, the optimal age for contracting H-pylori is 5 years and older (2012). Close contact at school with other children who are infected is implied, but not stated. Socioeconomics is believed to be the largest risk factor for contacting H-pylori. Developing countries prevalence of H-pylori infection ranges up to 90% (Banning, 2012). This fact is most likely related to contaminated water, poor sanitation, and over crowed living conditions (2012). Infected family members, such as siblings, increase the odds of infection with H-pylori infection by 95% (Elfant, 2012).

Morbidity and Mortality.

H-pylori is associated with many non-malignant and malignant gastrointestinal diseases (Boyanova, Mitov, Vladimirov, 2011). According to the website Cancer.gov. , "in 1994, the International Agency for Research on Cancer classified H-pylori as a cancer causing agent" (2013, para. 2). McCance et al. states "H-pylori is a major causative factor associated with chronic atrophic antral gastritis and peptic ulcer disease" (2010 p.1464).

Duodenal ulcers are also associated with H-pylori infection (2010). Symptoms may include chronic intermittent pain in the epigastria (2010, p.1465). Pain may present 30 minutes to

2 hours after eating (2010, p.1465). Pain may also occur in the middle of the night and resolve in the morning (2010, p.1465). Pain is relieved by eating food or taking antacids (2010, pp.1465).

Untreated gastric and duodenal ulcers may result in damage to muscularis mucosae of the stomach (2010). This can lead to damaged blood vessels, causing hemorrhage, leading to perforation of the gastrointestinal wall (2010). Surgery must be performed to repair the perforation or the patient may die.

H-pylori, especially strains that carries the CagA gene, change cell proliferation pattern in the gastric cell increasing the risk of gastric and duodenal carcinoma (2010). H-pylori is also linked to MALT lymphoma (2010). MALT stands for mucosa-associated lymphoid tissue. According to cancer.gov website, MALT lymphoma is a rare type of non-Hodgkin lymphoma (2013). Normally the stomach lacks lymphoid tissue, but infection of H-pylori, that colonizes the stomach lining, causes the tissue to be present in the stomach (2013).

Prognosis of patients diagnosed with gastric cancer is poor because the cancer usually produces vague symptoms or no symptoms at all (McCance et al, 2010). "Vague symptoms may include loss of appetite, especially for meat, weakness, and indigestion" (2010, p.1499). Cancer is usually not found until it has invaded the muscle layer of the stomach, surround tissues, draining lymph nodes, and veins (2010). Direct visualization through endoscopy is needed to make a definitive diagnosis of cancer.

Diagnosis and Treatment options

Diagnosis of H-pylori is obtained through invasive and non-invasive testing (Lopes, 2010). Non-invasive test include urea breath test, ELISA stool samples, and serology (2010). Invasive test include biopsies of the gastric mucosa (2010).

ELISA, enzyme-linked immunosorbent assay, is a "stool antigen test with a monoclonal antibody to H-pylori" (2010). ELISA has a sensitivity up to 100% making it a valuable test in diagnosing H-pylori. However titers will be positive if the patient has a history of H-pylori, indicating it cannot be used to check once treatment is complete (2010). Serologic test detect IgG antibodies to H-pylori, but like ELISA it cannot be used to assess for H-pylori after treatment is complete.

Treatment for H-pylori infection consist of a proton pump inhibitor, PPI, which reduces gastric acid secretions, and two antibiotics to eradicate the infection (Lopes, 2010). Proton pump inhibitors, used in treatment of H-pylori infection, are lanzoprazole, omeprazole, and pantoprazole (2010). Antibiotics used to eradicate H-pylori are amoxicillin, clarithromycin, metronidazole, and tetracycline. 'The addition of a bismuth-containing medication" (2010) along with the antibiotics and PPI is first-line therapy for H-pylori treatment (2010).

Duration of treatment for H-pylori varies, but is usually 7 days to 2 weeks (2010). There are different combinations of PPI's, antibiotics, and bismuth-containing medications used to treat H-pylori. If the PPI is used twice a day, the treatment is more effective than if the PPI is used once a day (2010). Success of treatment with triple therapy, PPI and two antibiotics, ranges from 60-85% depending on antibiotic resistance (2010). Failure rates increase for patients who smoke, older patients, and patients who are not compliant with treatment (2010).

Once treatment is complete the patient would need to be tested for the H-pylori to see if the treatment eradicated the infection. The test used is a urea breath test, UBT. This test must be performed with in one month after treatment of infection. The UBT, consist of the patient ingesting either C13, non-radioactive urea, used in pediatric patients and pregnant women, or C14 radioactive isotope (Boyanova et al, 2011). The patient ingests the urea with citric acid

solution (2011). The citric acid solution help reduce gastric emptying (2011). If the patient has H-pylori, urease released by the infection will hydrolyzed urea and form CO2 (2011). The CO2 will be released by the stomach to the blood stream and then to the lungs (2011). If the patient is positive another treatment therapy is prescribed. If the patient is negative no further treatment is needed. Follow up would be recommended, by the physician, if symptoms re-occur.

Summary of the disease condition and the impact on the body.

H-pylori is a prevalent infection affecting approximately fifty-percent of the world's population (CDC, 2009). It has been linked to many chronic gastrointestinal disease and conditions such as chronic gastritis, peptic ulcer disease, and malignancy, depending on the strain and location of the infection. Under developed countries are at greater risk for H-pylori infection due to sanitation problems, over crowed living conditions, and poor hygiene.

There are non-invasive and invasive test to diagnose H-pylori infection. Non-invasive test would be the urea breath test, the ELISA stool test, and serologic test to detect IgG antibodies to H-pylori (Lopes, 2010). The invasive procedure used to diagnose H-pylori infection is an upper endoscopy. A physician runs an endoscopy scope to the patient's stomach and obtains tissue biopsies of the antrum and body of the stomach. Those samples are sent to the lab for testing for the presence of the H-pylori infection. There are many treatment regimens used to eradicate H-pylori. The use of PPI's, antibiotics, and bismuth containing medications are the first line treatments for the infection.

The impact of H-pylori on the human body depends on the disease process caused by the infection. Chronic gastritis can cause symptoms such as abdominal pain, nausea, and vomiting. Chronic gastritis leads to peptic ulcer disease. Peptic ulcer disease causes ulcerations in the stomach lining causing symptoms like abdominal pain, weight loss, due to poor appetite, and

anemia, if the ulcers begins to bleed. Ulcers can worsen and lead to perforation of the gastric mucosa. Perforation is a life threating event that requires immediate surgical intervention. When untreated H-pylori can lead to gastric cancer. Cancer would have the greatest effect on the body causing symptoms of malaise, poor appetite, weight loss, and ultimately leading to death.

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