HELICOBACTER PYLORI

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Helicobacter Pylori

- Affects 50% of the worlds population (Tarkhashvili N, Beriashvili R, Chakvetadze N, Moistsrapishvili M, Chokheli M, Sikharulidze M, 2009)
- Linked to many upper gastrointestinal conditions such as:
  - Gastritis (acute , chronic)
  - Duodenitis
  - Gastric erosions
  - Peptic ulcer disease
  - Gastric cancer

Helicobacter Pylori

- Risk Factors
  - Overcrowded living conditions
  - Under developed countries
  - Contaminated water supply
  - Poor hygiene
  - Poor waste disposal
  - Dental plaques
  - Contaminated endoscopy scopes

Helicobacter Pylori

Transmission

Person to person

- Oral-oral (kissing)

- Fecal-oral (poor hand hygiene)

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).
- Catalase, urease, protease and phospholipases.

Cellular Level

- 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).
- 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).

Inflammation.

- When H-pylori invades the host, the host response is to “activate T and B lymphocytes with infiltration of neutrophils” (McCance et al., 2010, pp.1464).
- The release of cytokines, tumor necrosis factor a (TNF-a), and interleukins, eight and ten, damage the host gastric epithelium (2010, pp.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).

Inflammation.

- 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).

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, pp.1465).

Consequences

- 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, pp.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, pp.1464).

Gastritis

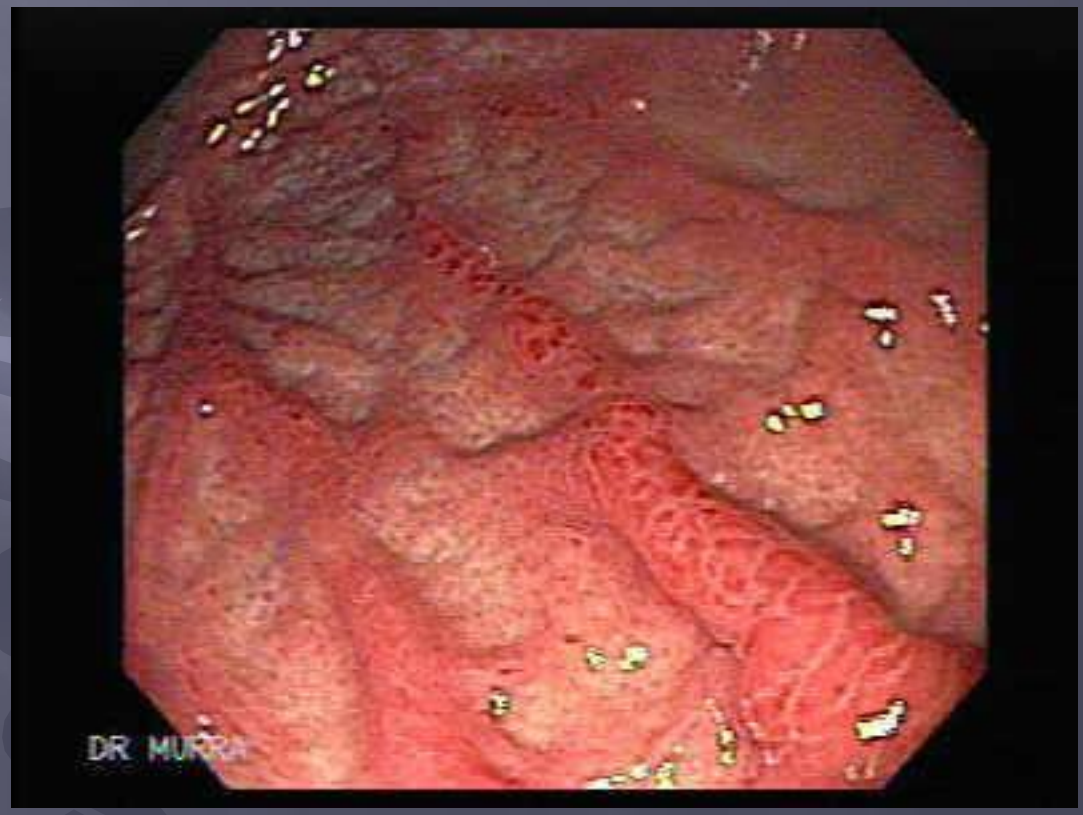
Inflammation of the lining of the stomach.

Symptoms of acute gastritis:

- Abdominal pain and Epigastric tenderness

Symptoms of chronic gastritis

- Anorexia
- Fullness
- Nausea
- Vomiting
- Epigastric pain

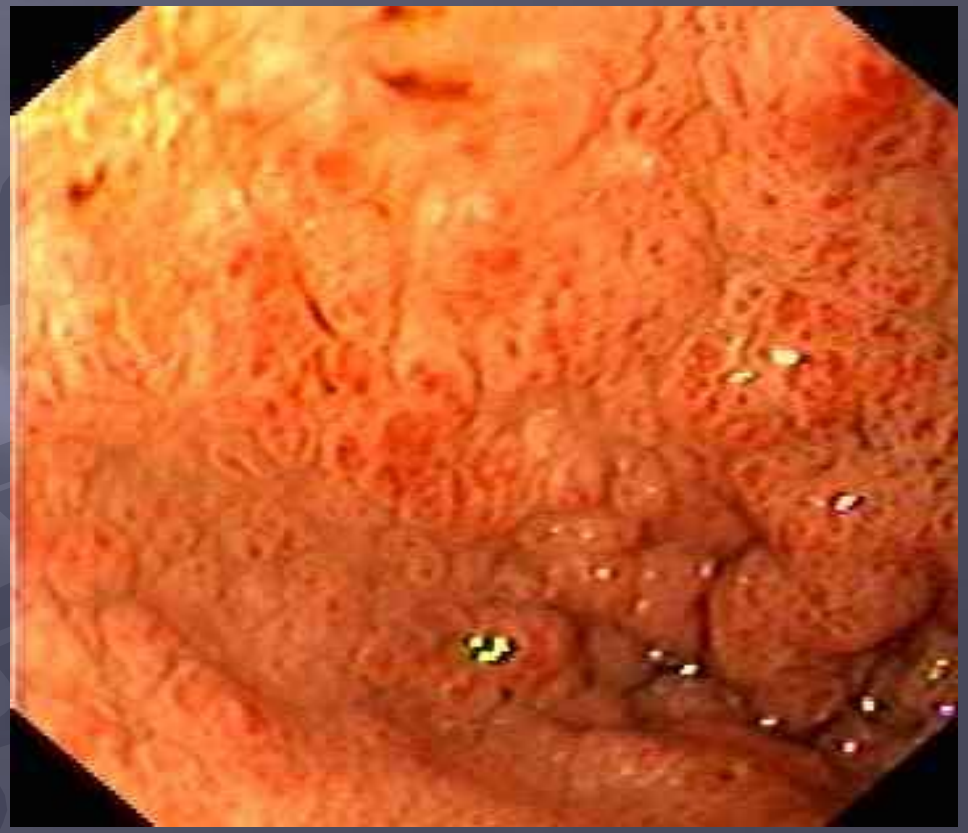


Duodenitis

Inflammation of the duodenal lining.

Symptoms

- Abdominal pain

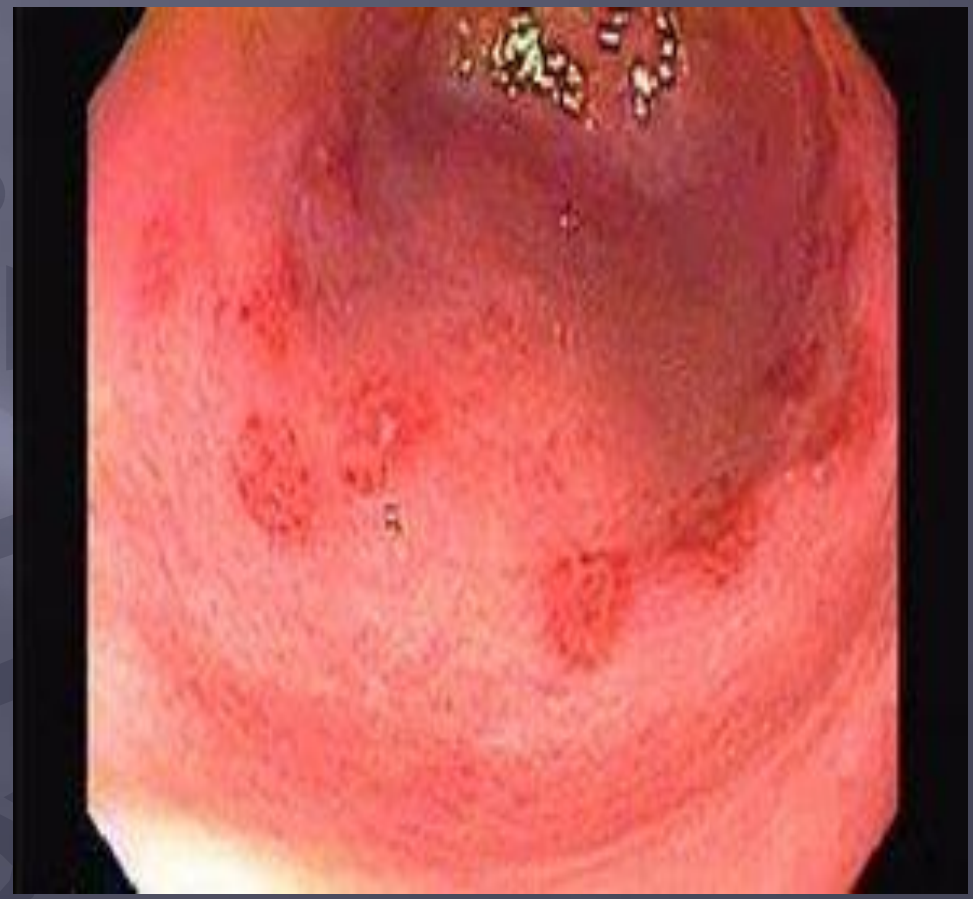


Gastric Erosions

Inflammation of the lining of the gastric mucosa.

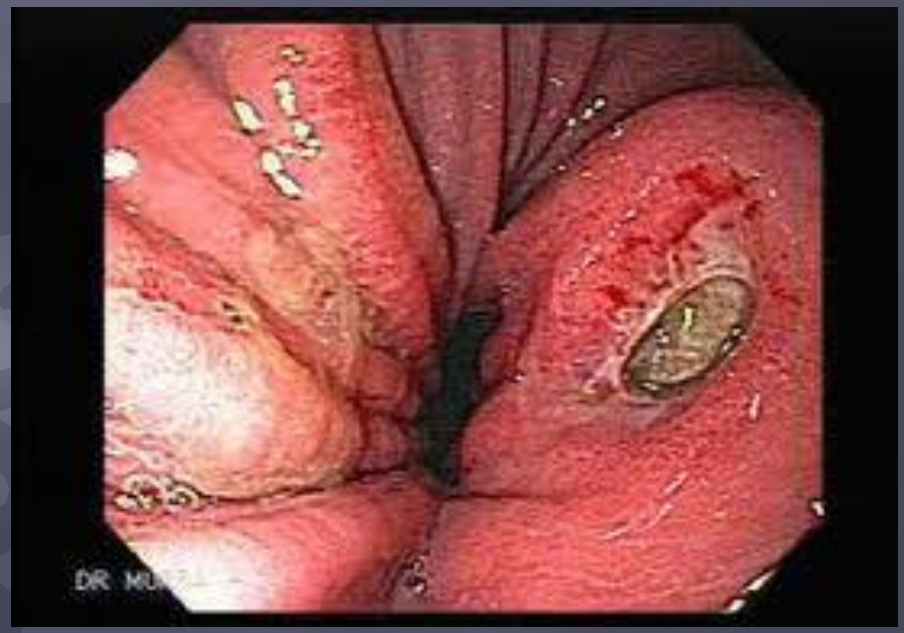
Symptoms (rare)

- Abdominal pain could be present.



Peptic Ulcer Disease

An ulceration in the mucosal lining of the stomach or duodenum.



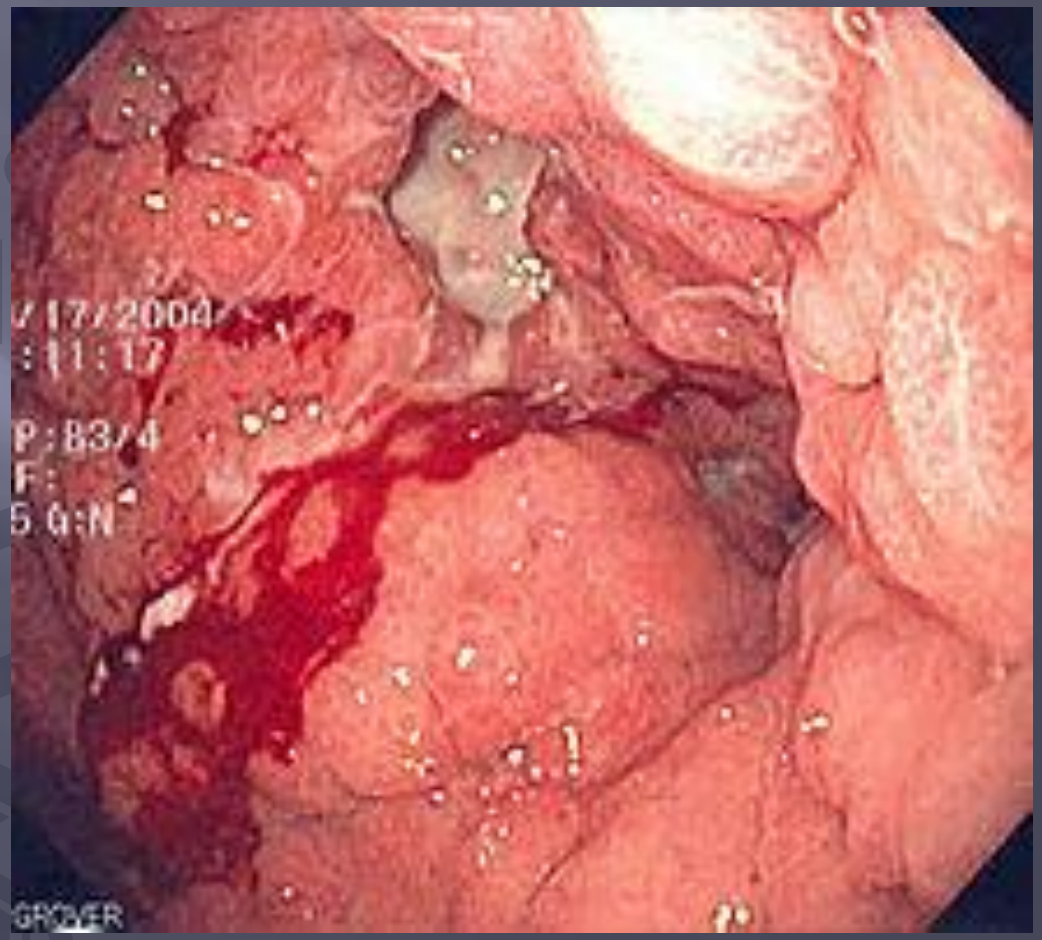
Gastric Cancer

Cancer in the stomach

Symptoms:

- Loss of appetite
- Weakness
- Indigestion

Symptoms are usually vague and prognosis is poor as it is not found until late stages.



McCance, (McCance, Huether, Brashers, Rote, 2010, pp.1464)

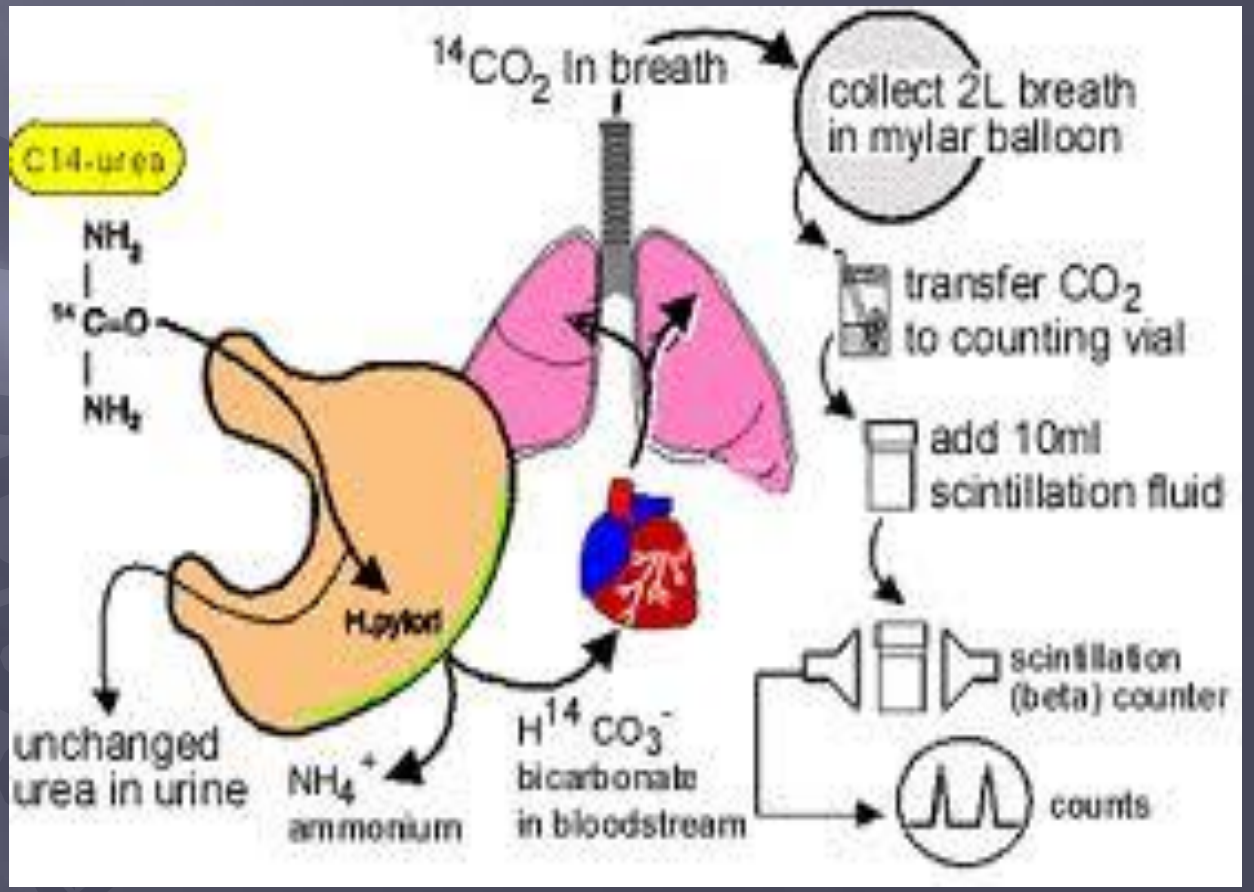
Diagnosis

Non-invasive:

ELISA stool test- Stool antigen test with antibody to H-pylori (cannot be used to check for H-pylori after treatment)

Serology- blood test to check for antibodies build up to H-pylori (cannot be used to check for H-pylori after treatment)

Urea Breath Test- Can be used to test for H-pylori before and after treatment.



INVASIVE PROCEDURE ESPHOGOGASTRODUODENOSCOPY

http://www.youtube.com/watch?v=ZvudWuvMjtA

Treatment

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).

Reference

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