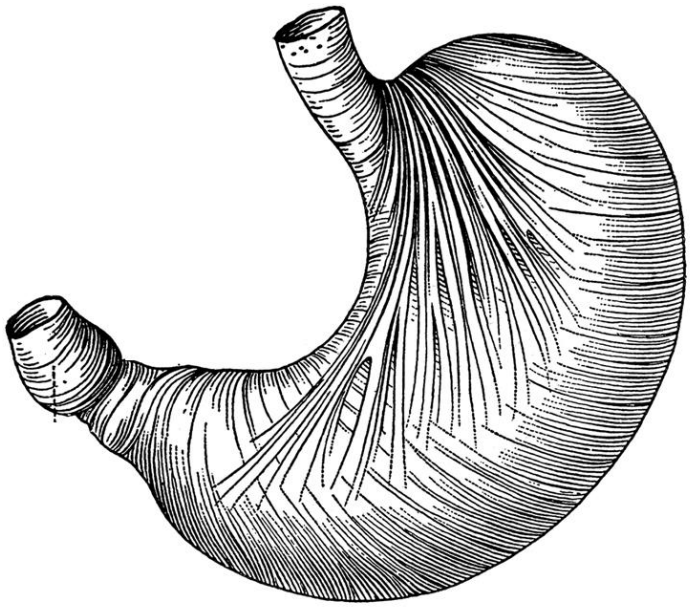
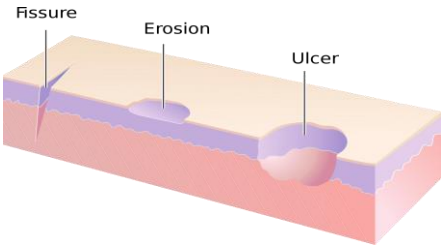
H.Pylori

microBiology 434



- **Important**
- **Hyperlink**

Peptic ulcer disease (PUD)



- **Introduction:**
  - defined as mucosal erosions (≥ 0.5cm) associated with the over usage of NSAIDs.
  - created in an **acidic** area.
  - More Peptic ulcers arise in **duodenum** (generally benign) than the **stomach** (4% of stomach ulcer can turn to be malignant tumor).
  - Multiple biopsies are needed to exclude cancer
- **Signs and symptoms:**
  - Abdominal pain, epigastric with severity relating to **mealtime**.
  - Nausea and vomiting,Loss of appetite and weight loss.
  - Haematemesis (vomiting of blood) due to gastric or esophageal damage.
  - Melena (foul-smelling & dark brown feces due to oxidized hemoglobin iron).

Helicobacter pylori

- Gram-**negative** spiral bacillus(its movement is like a throwing dart)
- is found closely associated with gastric mucosa and causes chronic active gastritis, gastric and duodenal ulcer ,and could develop adenocarcinoma and gastric mucosa-associated lymphoid tissue (MALT) lymphoma.
- H.pylori plays a role in gastric and duodenal ulceration and probably also gastric cancer.
- Over 80% of individuals infected with the bacterium are asymptomatic.
- More than 50% of the world's population harbour H. pylori in their upper gastrointestinal tract.
- Infection is more prevalent in developing countries.

- **route of transmission:** unknown, but individuals typically become infected in childhood.

❖ **Laboratory characteristics:**

➔ **Morphology and staining:** Small , Gram **negative**, spiral rods , motile by polar flagella.

➔ **Culture:** Blood or chocolate agar in a moist microaerophilic atmosphere.Small colonies grow after 5-7 days at 37˚C

➔ **Biochemical reactions:** catalase-positive, oxidase-positive, strongly urease-positive.

➔ **Serology:** IgG and IgM to Cytotoxic Associated Gene A (CagA) and (VacA) for virulence strains.

★ The Hallmark of the species is production of urease enzyme.

❖ **Diagnosis:** Checking for dyspeptic patients for H pylori:

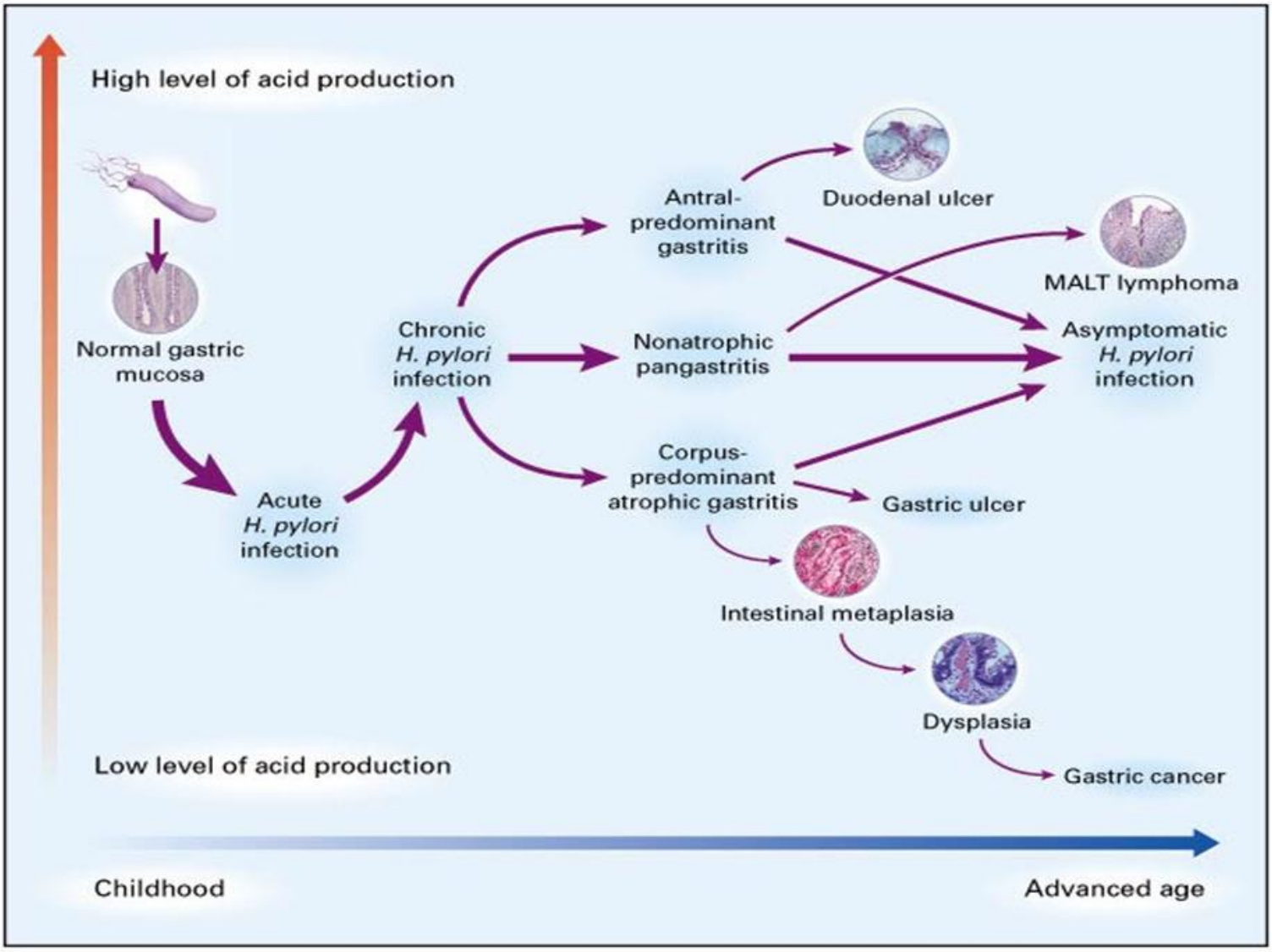
Non-invasive methods	Invasive methods (most reliable) Endoscopy
Blood antibody test (IgG, IgM or IgA)	
Stool antigen test	Histological examination.
Polymerase chain reaction (PCR)	culturing the bacteria.
Carbon urea breath test (C^{14} or C^{13}).	

❖ **Genome:** not important

❖ **Pathophysiology:**

•To **colonize** the stomach, *H pylori* must survive acidity. It uses flagella*, H pylori* moves through stomach lumen and drill into the mucoid lining of stomach.

- Produces adhesions that binds to the epithelial cells.
- Produces large amounts of *urease* enzyme that break down urea into co2 +ammonia and this in-turn **neutalizes** gastric acid.
- **Ammonia** is toxic to epithelial cells along with ***proteases***, *vacA* protein and ***phospholipases*** produced by *H pylori* and could **damage** epithelial cells.
- Colonization of stomach or duodenum results in chronic gastritis (inflammation of stomach lining) >> this inflammation stimulate **more production of gastric acid** >> This leads to gastric and duodenal **ulcers, atrophy and later cancer**.



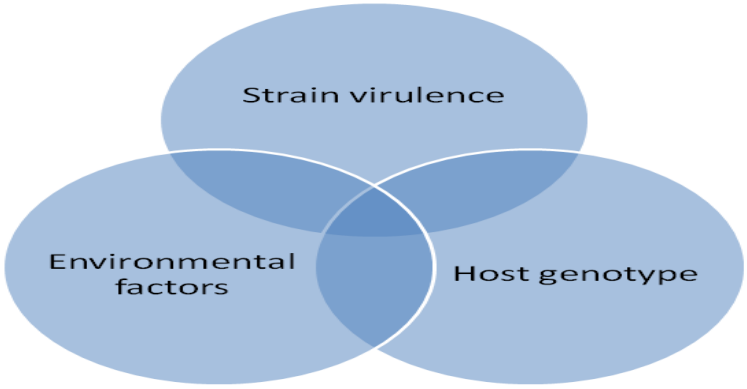
❖ **Prevention:**

- **Eradication of infection will improve symptoms** such as (dyspepsia, gastritis, peptic ulcer and cancer).
- **Vaccination:** Promising results with studying adjuvant, antigens and determining route of immunization (oral or intramuscular).
- **Dietary and drinking methods:** (eating broccoli, cabbage, honey, and drinking green tea).

❖ **Epidemiology:**

- **Around 50%** of world's population harbor *H pylori*.
- Third world has more rate of infection and infections are usually **acquired at childhood.**

❖ **The outcome of infection by *H. pylori* reflects an interaction between:**



❖ **Treatment (*VERY IMPORTANT)***

- **In vitro** (in the lab, outside the body) *H.pylori* is sensitive to amoxycillin, tetracycline, metronidazole, macrolides (clarithromycin).
- However, **in vivo** (inside the body) their efficacy is often poor due to the low pH of the stomach, their failure to penetrate the gastric mucus and the low concentration of antibiotic obtained in the mucosa of the stomach.
- Recently , Metronidazole in "developing countries" is becoming resistance (80-90%).

•**First line therapy**

- PPI b.d. (twice a day)+ clarithromycin 500mg b.d. + amoxicillin 1000mg b.d. **or** metrodiazole 500md (you have to choose one of them after doing the resistance test) (twice a day) minimum of 7 days

- **In case of failure we will go to the second line therapy:**
  - PPI b.d. + bismuth subsalicylate/subcitrate 120mg QDS (four times a day) + metronidazole 500mg t.d.s. (3 times a day)+ tetracycline 500mg q.d.s. for a minimum of 7 days. If bismuth is not available, PPI based triple therapies should be used 7days.
  - Regimen of combination of Amoxycillin , metronidazole, Ranitidine Bismuth Citrate and proton pump inhibitor (Omeprazole) have shown to increase the eradication rate up to 98%, Unfortunately it was followed by side effects such as vaginal candidiasis in 10% of women and Pseudomembranous colitis in 11% of patients.
  - With less than 40% Metronidazole resistance prevalence (over all the world) PPI-Clarithromycin-metronidazole is used.
  - Bismuth-based quadruple theapies remain best second choice treatment if available.

MCQ

1-The first line therapy for a patient with PUD :

a)Omeprazole + Clarithromycin +Tinidazole

b) PPI + clarithromycin + amoxicillin

c) Omeprazole + Amoxycillin + metronidazole + (PPI)

2- H.Pylori produce urease enzyme that breaks down urea into:

a)$NH_3 + CO_2$

b)$NH_4 + CO_2$

c)$NH_3 + O_2$

3-H.Pylori culture in:

a)Aerobic

b)Anaerobic

c)microaerophilic

ANSWERS: 1)B 2) a 3)C

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