# GI

## Microbiology

LEC no.


Writer: Mahmoud Jaradat - Zade Mansi

Corrector: Omar Smadi

Doctor: Dr. Nader Alarida

# Vibrios, Campylobacters, Helicobacter and Associated Bacteria

ما ينطق به الدكتور سيكون بالاحمر الداكن
الكلام المهم باللون البنفسجي
الكلام الخارجي باللون الأزرق

By: Nader Alaridah MD, PhD

- Vibrios the causative agent of cholera
- Campylobacter is the most common cause of bacterial gastrointeritis in developed countries more than shigella and salmonella especially in children populations,Be aware that the most common cause of gastrointeritis is viral causes.
- Helicobacter pylori the causative agent of peptic ulcer (either gastric or duodenal ulcer )as well as associated with adenocarcinoma of the stomach and MALT lymphoma

All these species are gram-negative bacilli ,they are separated from enterobacteriaceae based on two cardinal features :

1-They are motile , they possess flagella (all of them),vibrio and campylobacter have single polar flagella “monotrichous ” while helicobacter has multiple polar flagella “lophotrichous”.

2- They are oxidase positive .

The natural environment of vibrios ,Aeromonas and Plesiomonas is water so they are (aquatic bacteria)

Vibrios preferred water with slight salinity (it’s a little salty ) “brackish water ,marine and estuarine”

While (Aeromonas and Plesiomonas) are present in fresh water they cause simple gastrointeritis that affected human

Aeromonas causes disease in warm blooded animal and it is Negative DNAase

while plesiomonas in both warm and cold blooded animal including humans and it is positive DNAase

Brackish water is a mixture of fresh and salt water it often found when river meets the sea

# Overview

- These species are gram-negative rods that are all widely distributed in nature.
- Vibrio cholerae produces an enterotoxin that causes cholera, a profuse watery diarrhea that can rapidly lead to dehydration and death.
- Campylobacter jejuni is a common cause of enteritis in humans.
- Less commonly, Aeromonas and, rarely, Plesiomonas have been associated with diarrheal disease in humans.
- Helicobacter pylori has been associated with gastritis and duodenal ulcer disease.

# THE VIBRIOS

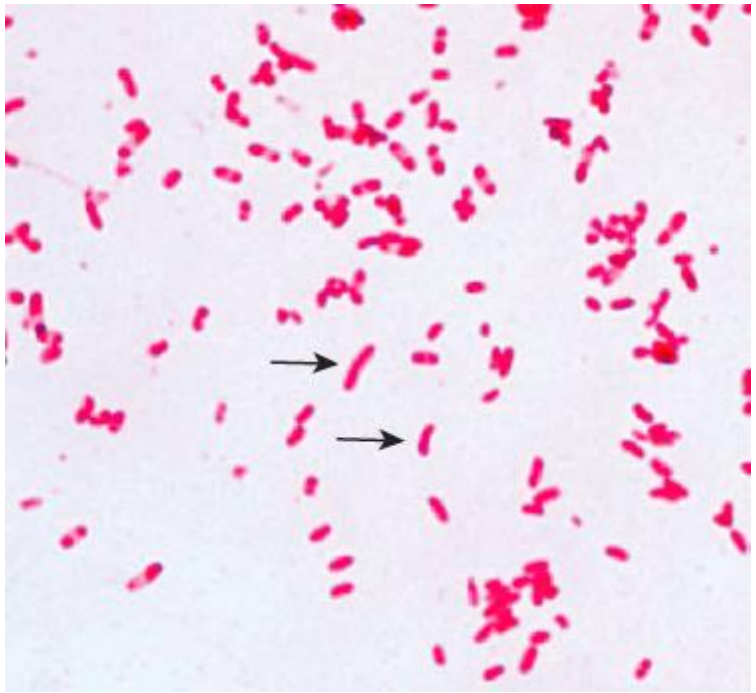
- Vibrios are among the most common bacteria in surface waters worldwide.
- Vibrio cause a number of important infectious syndromes. Classic among them is cholera, a devastating diarrheal disease caused by Vibrio cholerae that has been responsible for seven global pandemics and much suffering over the past two centuries and remains a significant public health concern in the developing world today.
- V cholerae serogroups **O1** and **O139** cause cholera in humans, and other vibrios may cause soft tissue infections ,sepsis or enteritis. Responsible for epidemics and pandmics they cause typical cholera disease .
- Other important Vibrio species that associated primarily with gastrointestinal include V parahaemolyticus, the most common cause of Sea-foodborne (raw fish or shellfish) gastroenteritis in Asia, and V vulnificus (oysters) , a cause of severe sepsis in patients with cirrhosis and primary wound infection (Vulnificus is Latin for “wound maker.”) and V alginolyticus occasionally causes eye, ear, and wound infections.

- Vibrios are halotolerant they tolerate salt water(certain amount of NaCl)
- non-O1 and non-O139 cause cholera like disease it is mild gastroenteritis self limited

- The vibrios species flourish in water environment where there is shellfish, shrimps and mussels and other invertebrates (invertebrates: don't have vertebral column)
- V vulnificus it is primary causes of cellulitis wound infections(in fisher men) if they have skin abrasion , a common cause of severe sepsis and septicemia in patients with cirrhosis
- V alginolyticus is asociated with localized eye, ear, and wound infections.

# VIBRIO CHOLERAE

- The epidemiology of cholera closely parallels the recognition of V cholerae transmission in water and the development of sanitary water systems.



- V cholerae is a comma-shaped, curved rod 2–4 µm long . It is actively motile by means of a polar flagellum. On prolonged cultivation, vibrios may become straight rods that resemble the gram-negative enteric bacteria.

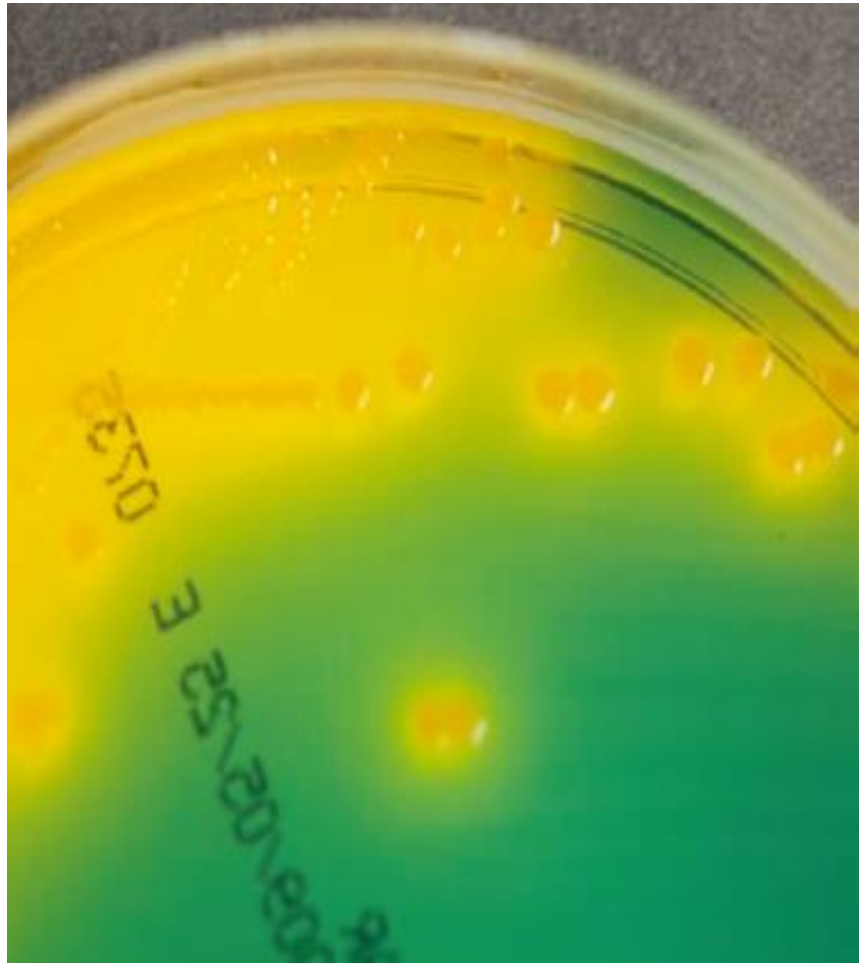
**Vibrio cholera is a gram – ve bacilli slightly curved and that's how we see it under the light microscope (look at the picture)**

**For the enrichment of vibrios, We use peptone alkaline broth Remember that the vibrios are halotolerant, but they are extremely sensitive to the acidity of the stomach, so they need large infectious dose to establish infection with cholera**

- Characteristically, vibrios grow at a very high pH (8.5–9.5) and are rapidly killed by acid.

- V cholerae produces convex, smooth, round colonies that are opaque and granular in transmitted light.

- V cholera grows well on thiosulfate-citrate-bile-sucrose (TCBS) agar, a media selective for vibrios, on which it produces yellow colonies (sucrose fermenters) that are readily visible against the dark-green background of the agar that used for the identification and isolation of vibrios



- A positive oxidase test result is a key step in the preliminary identification of V cholerae and other vibrios.

- Vibrio species are susceptible to the compound O/129 (2,4-diamino-6,7-diisopropylpteridine phosphate), which differentiates them from Aeromonas species, which are resistant to O/129.

- Most Vibrio species are halotolerant, and NaCl often stimulates their growth. Some vibrios are halophilic, requiring the presence of NaCl to grow.

**We use this feature to differentiate between vibrios species**
**For example, if we grow all vibrios species at 6%of NACL all vibrio species will grow**
**While at 8-9% NACL (ocean water) only parahaemolyticus and vulnificus will grow, but not cholera.**
**Vibrio cholera is halotolerant not halophilic**

# Antigenic Structure and Biologic Classification

- ❖Many vibrios share a single heat-labile flagellar **H** antigen( remember they are motile). Antibodies to the H antigen are probably not involved in the protection of susceptible hosts.
- ❖V cholerae has **O** lipopolysaccharides (they are gram –ev)( that confer serologic specificity. There are at least 206 O antigen groups.
- V cholerae strains of O group 1 and O group 139 cause classic cholera; occasionally, non-O1/non-O139 V cholerae causes cholera-like disease.(until now there are seven pandemics from the cholera affected the world six of them by O1,so O1 is the biggest cause of epedimics and pandemics).
- Antibodies to the O antigens tend to protect laboratory animals against infections with V cholerae.
- Two biotypes of V. cholerae O1, classical and El Tor, are distinguished. Each biotype is further subdivided into two serotypes, termed Inaba and Ogawa.

- 50% of infected people of the classical biotype of O1 will show symptoms and signs and 50% will show mild disease or be asymptomatic .
- While El Tor biotype is a milder form of cholera infection and 75% of infected people don’t show symptoms and signs
- V cholera O1 doesn’t produce a capsule while O139 produces a capsule.
- O139 is hemolysin positive
- O1 is resistant to polymyxin b while O139 is sensitive .

# Vibrio cholerae Enterotoxin

- Cholera toxin (choleragen)it comes from bacteriophage (CTX bacteriophage), a potent protein enterotoxin elaborated by the organism in the small intestine with a molecular weight (MW) of about 84,000, consisting of subunits A (MW,28,000) and B.

- The genes for V cholerae enterotoxin are on the bacterial **chromosome.**

- Ganglioside GM1 serves as the mucosal receptor for subunit B, which promotes entry of subunit A into the cell. Activation of subunit A1 yields increased levels of intracellular cyclic adenosine monophosphate **(cAMP**) and results in prolonged hypersecretion of water and electrolytes.

**V.Cholera is Non inflammatory diarrhea causing infection and toxin mediated disease**
**So, these patients have profused watery diarrhea (they don't have WBCs and RBCs) v.cholera is considered one of the most dramatic diarrheal diseases that can affect human being ,that lead the patient to lose 1L/h which means 20-30Liter /day**
**Therefore, the management for the cholera is fluid and electrolytes replacement then we use antibiotics**

Vibrio species have double chromosomes, each containing a single circular chromosome. The CTX bacteriophage must infect one of the two chromosomes present in Vibrio species. This chromosome must also carry another virulence factor called toxin coregulated pilus (TCP), which is essential for colonization in the intestine. Once infected, the bacteriophage integrates with the chromosome containing TCP through transduction ( lysogenic conversion). Only then it becomes expressed and elaborated.

The toxin produced by the CTX bacteriophage consists of two subunits: A and B. The B subunit is responsible for binding to its receptor, ganglioside GM1, facilitating internalization. Subunit A then becomes activated, causing ADP ribosylation of the G protein coupled receptor, specifically Gs (G stimulatory protein). This activation leads to a 'locked on mode,' increasing adenylate cyclase activity and subsequently elevating cAMP levels. This mechanism results in hypersecretion of fluid and electrolytes into the intestinal lumen, similar to the mechanism observed in enterotoxigenic E. coli (ETEC).

# Pathogenesis

- Under natural conditions, V cholerae is pathogenic only for humans. A person with normal gastric acidity may have to ingest as many as $10^{10}$ or more V cholerae to become infected when the vehicle is water because the organisms are susceptible to acid.
- When the vehicle is food, as few as $10^{2}$–$10^{4}$ organisms are necessary because of the buffering capacity of food and in patients who had gastrectomy, or pts on acid reducing drugs.
- The toxin-coregulated pilus (TCP), so named because its synthesis is regulated in parallel with that of cholera toxin, is essential for V. cholerae to survive and multiply in (colonize) the small intestine and causes extreme profuse diarrhea “painless diarrhea which no abdominal pain”
- The organisms do not reach the bloodstream but remain within the intestinal tract.
- Virulent V cholerae organisms attach to the microvilli of the brush border of epithelial cells. There they multiply and liberate cholera toxin and perhaps mucinases and endotoxin.

# Clinical Findings

- The burden of disease is often greatest during “cholera seasons” associated with high temperatures, heavy rainfall, and flooding, but cholera can occur year-round.
- The longest epidemic of cholera present in Yemen

- About 50% of infections with classic V cholerae are asymptomatic, as are about 75% of infections with the El Tor biotype.

- The incubation period is 12 hours–3 days for persons who develop symptoms, depending largely on the size of the inoculum ingested.
- The problem with cholera is the rapid loss of fluid 20L/d which lead to acute renal failure ,metabolic acidosis, hypovolemic shock
- There is a sudden onset of nausea and vomiting and profuse diarrhea with abdominal cramps. Stools, which resemble “rice water,” contain mucus, epithelial cells, and large numbers of vibrios.
- One of the presentation of th cholera patients called Barnes diarrhea they don’t have abdominal cramps just you see rice water stool.

- There is rapid loss of fluid and electrolytes, which leads to profound dehydration, circulatory collapse, and anuria. The mortality rate without treatment is between 25% and 50%. (In patients with symptomatic disease)

- The diagnosis of a full blown case of cholera presents no problem in the presence of an epidemic. However, sporadic or mild cases are not readily differentiated from other diarrheal diseases. The El Tor biotype tends to cause milder disease than the classic biotype.

# Diagnostic Laboratory Tests

- A. Specimens
- Specimens for culture consist of mucus flecks from stools.
- B. Smears
- Dark-field or phase contrast microscopy may show the rapidly motile vibrios.(shooting star motility )
- C. Culture
- Growth is rapid in peptone agar, on blood agar with a pH near 9.0, or on TCBS agar, and typical colonies can be picked in 18 hours.
- They are halotolerant so we put them in alkaline peptone water then we isolate them in selective agar TCBS then we see yellow colonies
- D. Specific Tests
- V cholerae organisms are further identified by slide agglutination tests using anti-O group 1 or group 139 antiserum and by biochemical reaction patterns. (So you know the serotype)

**In endemic and pandemic cases, there is no need of laboratory assistance , as diagnosis can often be based on clinical suspicion , especially if characteristic symptoms such as “rice water” stool are present.( This is regard to sporadic cases ,but overall, we need laboratory assistance).**

# Treatment

- The most important part of therapy consists of water and electrolyte replacement to correct the severe dehydration and salt depletion.

- Many antimicrobial agents are effective against V cholerae, but these play a secondary role in patient management. Oral tetracycline and doxycycline tend to reduce stool output in cholera and shorten the period of excretion of vibrios.

- In some endemic areas, tetracycline resistance of V cholerae has emerged; the genes are carried by transmissible plasmids. In children and pregnant women, alternatives to the tetracyclines include erythromycin and furazolidine .

# Prevention

- ❖Provision of safe water and of facilities for sanitary disposal of feces, improved nutrition, and attention to food preparation and storage in the household can significantly reduce the incidence of cholera.(the patients should be Isolated and the contact should follow up )
- ❖Currently, two oral killed cholera vaccines have been prequalified by the WHO and are available internationally :
- WC-rBS (Dukoral.; Crucell, Stockholm, Sweden) contains several biotypes and serotypes of V. cholerae O1 supplemented with recombinant cholera toxin B subunit.
- BivWC (Shanchol™; Shantha Biotechnics–Sanofi Pasteur, Mumbai, India) contains several biotypes and serotypes of V. cholerae O1 and V. cholerae O139 without supplemental cholera toxin B subunit.
- VAXCHORA (live attenuated ) the only one is approved in the United States by the FDA,O1 inaba strain and these three vaccines are taken orally

Chemoprophylaxis For travelers person to cholera endemic areas they take doxycycline usually confer limited protection for short time,

# CAMPYLOBACTER

- Campylobacters are motile, non-spore-forming, curved, gram-negative rods.
- Campylobacters are found in the gastrointestinal tract of many domestic animals used for food (including poultry, cattle, sheep, and swine) and many household pets (including birds, dogs, and cats)
- Campylobacters cause both diarrheal and systemic diseases and are among the most widespread causes of infection in the world.
- The classification of bacteria within the family Campylobacteriaceae has changed frequently. Some species previously classified as campylobacters have been reclassified in the genus Helicobacter. The genus Arcobacter has been created.
- The human pathogens fall into two major groups: those that primarily cause diarrheal disease and those that cause extraintestinal infection
- The most common cause of bacterial gastroenteritis especially in children
- Infection to illness ratio drops significantly after age 2

- Campylobacter jejuni and coli are the prototype organism in the group and are a very common cause of diarrhea in humans. (Cause gastroenteritis in developed world )

- Campylobacter fetus has two subspecies, fetus and venerealis. C fetus subspecies fetus is an opportunistic pathogen that causes systemic infections especially sepsis in immunocompromised patients. It may occasionally cause diarrhea in immunocompetent pts.
- Campylobacter venerealis the main transmission rote is through venereal rote
- Other organisms that cause diarrheal disease include Campylobacter coli, Campylobacter upsaliensis, Campylobacter lari, Campylobacter hyointestinalis, Campylobacter fetus, Arcobacter butzleri, Arcobacter cryaerophilus, Helicobacter cinaedi, and Helicobacter fennelliae(the habitat of these hilocobacter is intestinal tract causing diarrheal disease while helicobacter pylory in the stomach. (The helicobacter has been separated from campylobacter ,while arcobacter was kept)

# CAMPYLOBACTER JEJUNI AND CAMPYLOBACTER COLI

- C jejuni and Campylobacter coli have emerged as common human pathogens, causing mainly enteritis and occasionally systemic infection. (They cause bloody diarrhea )

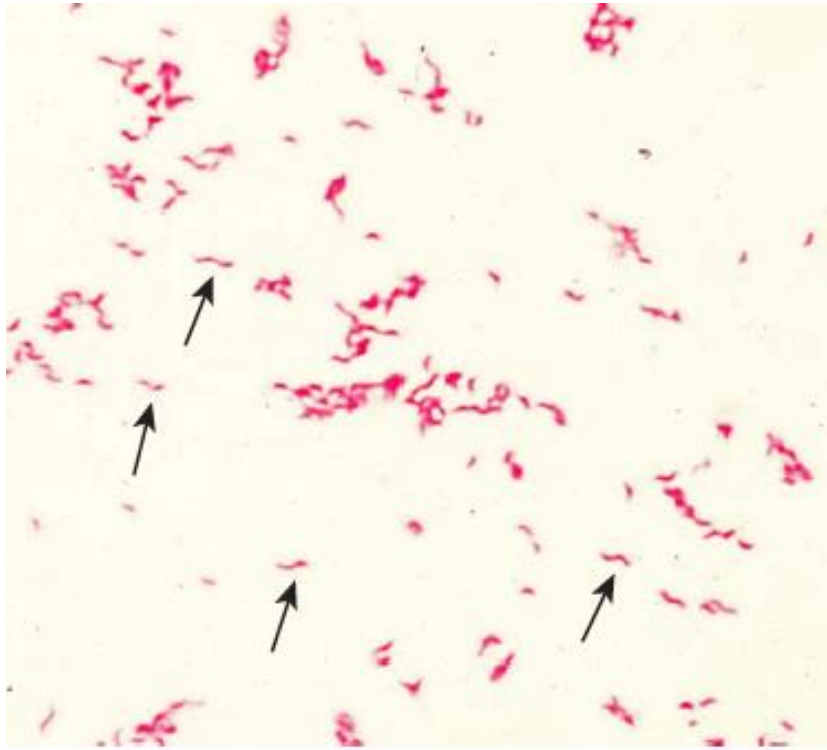
- C jejuni and C coli cause infections that are clinically indistinguishable, and laboratories generally do not differentiate between the two species.

- Between 5% and 10% of infections reported to be caused by C jejuni are probably caused by C coli. These bacteria are at least as common as salmonellae and shigellae as a cause of diarrhea especially in the developed world.

# CAMPYLOBACTER JEJUNI

**These species are microaerophilic ( need low O2 levels = 5% And high CO2 levels )**

- gram-negative rods with comma, S, or "**gull wing**" shapes. They are motile, with a single polar flagellum, and do not form spores.



- Selective media are needed, and incubation must be in an atmosphere with reduced O2 (5% O2) with added CO2 (10% CO2).

- Incubation of primary plates for isolation of C jejuni should be at 42°C. Although C jejuni grows well at 36–37°C, incubation at 42°C prevents growth of most of the other bacteria present in feces, thus simplifying the identification of C jejuni. Several selective media are in widespread use.

**These species are also Thermophilic**

- These species are Thermophilic that can tolerate high temperature conditions.

- Regarding C. Jejuni and C. Coli, they can grow in temperatures that reach 37c and 42c. But growing in temperature like 25c is difficult.

- In contrast; C. Fetus which causes systemic disease, can grow in 25c and 37c conditions, but it is difficult for it to grow in 42c conditions.
- We can use this feature to distinguish between different campylobacter species.

# Pathogenesis

- The infection is acquired by the oral route from food, drink, or contact with infected animals or animal products, especially poultry.
- C jejuni is susceptible to gastric acid, and ingestion of about $10^4$ organisms is usually necessary to produce infection.
- Both the motility of the strain and its capacity to adhere to host tissues appear to favor disease, but classic enterotoxins and cytotoxins (cytolethal distending toxin, or CDT) appear not to play substantial roles in tissue injury or disease production.
- The organisms multiply in the small intestine, invade the epithelium (mucosa) and submucosa, and produce inflammation that results in the appearance of red and white blood cells in the stools. Occasionally, the bloodstream is invaded, and a clinical picture similar to enteric fever develops. Localized tissue invasion coupled with the toxic activity appears to be responsible for the enteritis.

- **Site of colonization: Small and large intestine.**
- **Incubation period: < 2 days**
- **Clinical presentation usually starts with watery diarrhea.**
- **Then, they invade mucosa and submucosa causing BLOODY DIARRHEA. Also, they might reach systemic circulation causing fever.**
- **In some cases, they might develop to cause HEMOLYTIC UREMIC SYNDROM ( HUS syndrome ).**

# Clinical Findings

- A prodrome of fever, headache, myalgia, and/or malaise often occurs 12–48 h before the onset of diarrheal symptoms. profuse diarrhea that may be grossly bloody.
- Usually, the illness is self-limited to a period of 5–8 days, but occasionally it continues longer.
- Most cases resolve without antimicrobial therapy; however, in about 5–10% of patients, symptoms may recur.
- Local suppurative complications of infection include cholecystitis, pancreatitis, and cystitis; distant complications include meningitis, endocarditis, arthritis, peritonitis, cellulitis, and septic abortion. All these complications are rare, except in immunocompromised hosts
- Hepatitis, interstitial nephritis, and the hemolytic-uremic syndrome occasionally complicate acute infection
- Certain serotypes of C jejuni have been associated with post-diarrheal Guillain-Barré syndrome, a form of ascending paralytic disease. Reactive arthritis and Reiter's syndrome may also follow acute campylobacter diarrhea.

**Reiter's syndrome: Autoimmune disease – molecular mimicry + Triad of (conjunctivitis+arthritis+urethritis)**

# Diagnostic Laboratory Tests

**The sample must be put in a Microaerophilic and thermophilic conditions.**

- A. Specimens
- Diarrheal stool is the usual specimen. C jejuni , C fetus , and other campylobacters may occasionally be recovered from blood cultures usually from immunocompromised or elderly patients.
- B. Smears
- Gram-stained smears of stool may show the typical "**gull wing**"–shaped rods. Dark-field or phase contrast microscopy may show the typical **darting motility** of the organisms.
- C. Culture
- Culture on the selective media (Skirrow's, Butzler's, Blaser's, Campy-BAP and Preston media ) is the definitive test to diagnose C jejuni enteritis. If another species of Campylobacter is suspected, medium without a cephalosporin should be used and incubated at 36–37° C.

**Enzyme Immunoassay test is used in USA.**
**Weak point in EIA: Low sensitivity.**

# Treatment

**For systemic infection caused by C. Fetus, Gentamycin and Chloramphenicol must be used.**

- Fluid and electrolyte replacement is central to the treatment of diarrheal illnesses.
- Even among patients presenting for medical attention with Campylobacter enteritis, not all clearly benefit from specific antimicrobial therapy. Indications for therapy include high fever, bloody diarrhea, severe diarrhea, persistence for >1 week, and worsening of symptoms. A 5- to 7-day course of erythromycin is the regimen of choice.
- An alternative regimen for adults is ciprofloxacin or another fluoroquinolone for 5–7 days.
- For systemic infections, treatment with gentamicin or imipenem or chloramphenicol should be started empirically, but susceptibility testing should then be performed

# HELICOBACTER PYLORI

**H. Pylori is part of microbiome in stomach.**

**H. Pyolri is Oxidase +, Catalase +, Urese +**

- H pylori is **a spiral-shaped** gram-negative rod.
- It has **multiple flagella** at one pole and is actively motile.
- The organism has several acid-resistance mechanisms, most notably a highly expressed **urease** that catalyzes urea hydrolysis to produce buffering ammonia. H. pylori is microaerophilic (i.e., requires low levels of oxygen), is oxidase positive and catalase positive is slow-growing, and requires complex growth media in vitro.
- H pylori is associated with antral gastritis, duodenal (peptic) ulcer disease, gastric ulcers, gastric adenocarcinoma and gastric mucosa-associated lymphoid tissue (MALT) lymphomas. It may be one initial precipitant of pernicious anemia and also may predispose some patients to iron deficiency through occult blood loss and/or hypochlorhydria and reduced iron absorption.

# Epidemiology

**More advanced clinical presentations are associated with strains with: Type 4 secreting system, pathogenicity islands, CAG A gene, VACA**

- Helicobacter pylori colonizes the stomach in ~50% of the world's human population, essentially for life unless eradicated by antibiotic treatment.

**There is a tendency for casing clustering:**
**بنفس العيلة ينصاب أكتر من حدا**

- Humans are the only important reservoir of H. pylori. Children may acquire the organism from their parents (most often the primary caregiver) or from other children.

- Most H. pylori–colonized persons do not develop clinical sequelae. That some persons develop overt disease whereas others do not is related to a combination of factors: bacterial strain differences (cag-positive , type IV secretion system, the vacuolating cytotoxin VacA), host susceptibility to disease, and environmental factors (the interleukin 1 gene polymorphisms, and smoking).

**Route of transmission: person-person , feco-oral, oral-oral (saliva)**

# Pathogenesis

**Multiplication and colonization occur in stomach and duodenum but not other parts in small intestine.**

- H pylori is found deep in the mucous layer near the epithelial surface where physiologic pH is present.
- H pylori is quite motile, even in mucus, and is able to find its way to the epithelial surface. H pylori overlies gastric-type but not intestinal- type epithelial cells.
- H pylori also produces a protease that modifies the gastric mucus and further reduces the ability of acid to diffuse through the mucus.
- H pylori produces potent urease activity, which yields production of ammonia and further buffering of acid.

**Multipolar flagella helps in movement along mucus of stomach**

- The mechanisms by which H pylori causes mucosal inflammation and damage are not well defined but probably involve both bacterial and host factors. The bacteria invade the epithelial cell surface to a limited degree. Toxins and lipopolysaccharide may damage the mucosal cells, and the ammonia produced by the urease activity may also directly damage the cells.
- Polymorphonuclear and mononuclear cell infiltrates are seen within the epithelium and lamina propria. Vacuoles within cells are often pronounced. Destruction of the epithelium is common, and glandular atrophy may occur. H pylori thus is a major risk factor for gastric cancer.
- H. pylori colonization induces chronic superficial gastritis, a tissue response in the stomach that includes infiltration of the mucosa by both mononuclear and polymorphonuclear cells.

# Clinical Findings

**H. Pylory induces two types of peptic ulcer: Duodenal and Gastric**

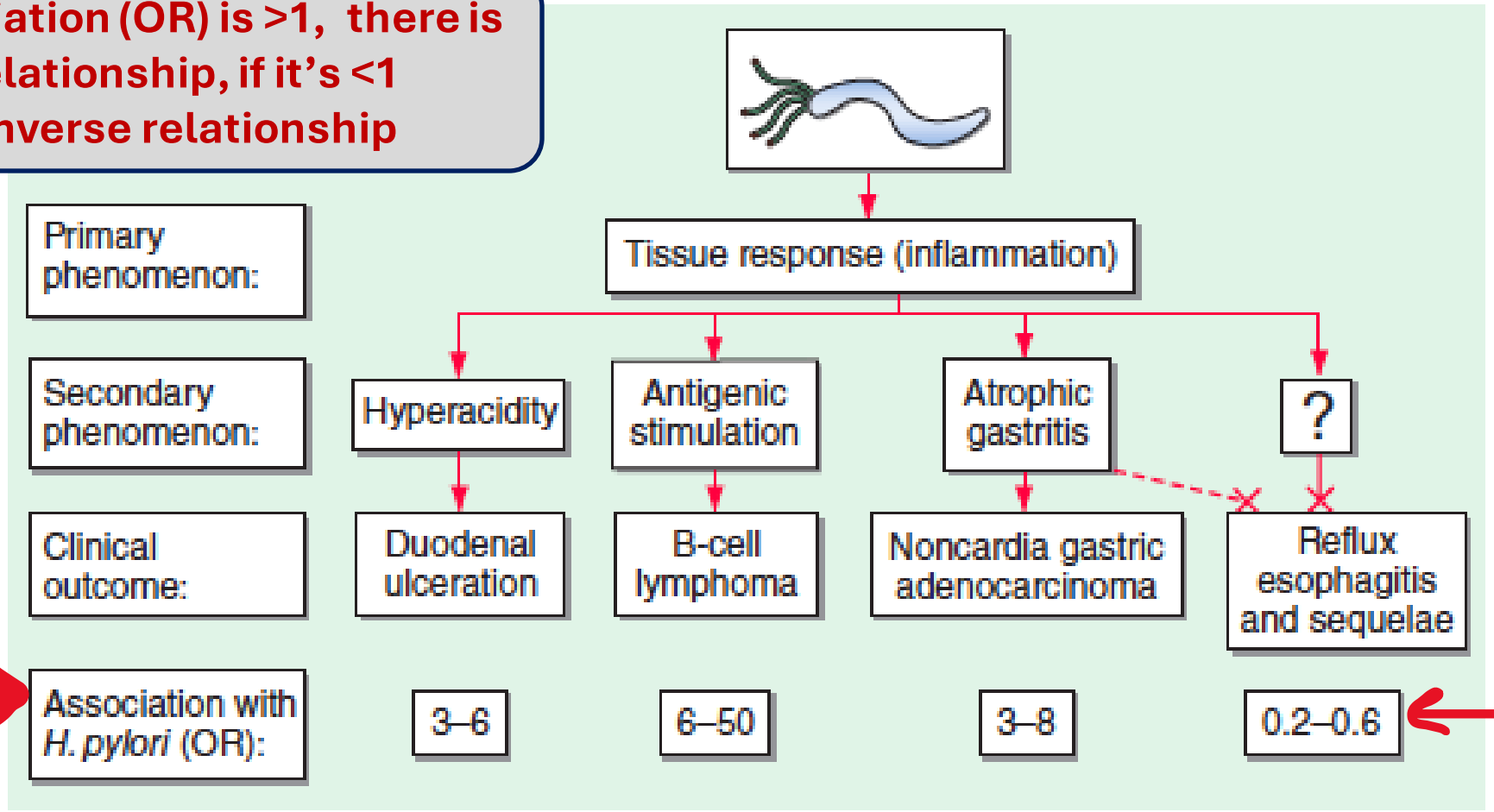
- Acute infection can yield an upper gastrointestinal illness with nausea and pain; vomiting and fever may also be present. The acute symptoms may last for less than 1 week or as long as 2 weeks.

- After colonization, the H pylori infection persists for years and perhaps decades or even a lifetime. About 90% of patients with duodenal ulcers and 50–80% of those with gastric ulcers have H pylori infection. Recent studies confirm that H pylori also is a risk factor for gastric carcinoma and lymphoma.

**One way to know if its gastric or duodenal ulcer is to ask the pt if the pain is relieved when eating (duodenal), or if it exacerbates when eating (gastric).**

# Relationships between colonization with Helicobacter pylori and diseases of the upper gastrointestinal tract.

**if the association (OR) is >1, there is a positive relationship, if it's <1 there is an inverse relationship**




**Infection with h.pylori is protective to GERD and Barret esophagus.**

# Diagnostic Laboratory Tests

**Culturing of H. Pylori is very difficult**

❖Smears

- The diagnosis of gastritis and H pylori infection can be made histologically. A gastroscopy procedure with biopsy is required. Routine stains demonstrate gastritis, and Giemsa or special silver stains can show the curved or spiral-shaped organisms.

❖Culture

- .Culture is performed when patients are not responding to treatment, and there is a need to assess susceptibility patterns.

**There are two types of tests:**
**1.Invasive 2.Noninvasive ( urea breath test )**

❖Special Tests

- Rapid tests to detect urease activity in vitro are widely used for presumptive identification of H pylori in specimens.
- In vivo tests for urease activity can be done also. In urea breath tests, 13 C- or 14 C-labeled urea is ingested by the patient. If H pylori is present, the urease activity generates labeled $CO_2$ that can be detected in the patient's exhaled breath.
- Detection of H pylori antigen in stool specimens is appropriate as a test of cure for patients with known H pylori infection who have been treated.

# Treatment

- Triple therapy with (1)metronidazole and (2)either bismuth subsalicylate or bismuth subcitrate plus (3)either amoxicillin or tetracycline for 14 days eradicates H pylori infection in 70–95% of patients.

- An acid-suppressing agent given for 4 o 6 weeks enhances ulcer healing. Proton pump inhibitors (PPIs) directly inhibit H pylori and appear to be potent urease inhibitors.

- The preferred initial therapy is 7–10 days of a PPI plus amoxicillin and clarithromycin or a quadruple regimen of a (1)PPI (2)metronidazole, (3)tetracycline, and (4)bismuth for 10 days.

نرجوا منكم يا أفاضل بعد الدعاء لنا أن تدخلوا لهذا الرابط
وتقوموا بتزويدنا بتغذية راجعة عن الشيتات والموديفايدز

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The End