Gram Negative Rods

GENUS: *Vibrio*

The medically important vibrios

Organism	Human Disease
Vibrio cholerae serogroups O1 and O139	Epidemic and pandemic cholera
Vibrio cholerae serogroups non-O1/non-O139	Cholera-like diarrhea; mild diarrhea; rarely, extraintestinal infection
Vibrio parahaemolyticus	Gastroenteritis, wound infections, septicemia
Vibrio vulnificus	Gastroenteritis, wound infections, septicemia

- Species of medical importance: Vibrio cholerae-01
- Actively motile, gram-negative curved rods.

- ***Vibrio cholerae* Characteristics:**
- Found in fresh water, shellfish and other sea food.
- Man is the major reservoir of *V. cholerae-01*, which causes epidemic cholera.
- Readily killed by heat and drying; dies in polluted water but may survive in clean stagnant water, or sea water for 1-2 weeks.

- Antigenic structure:
- O antigen . Six major subgroups.
- All strains possess a distinctive O antigen and belong to subgroup I with subdivision into three serotypes; ***Ogawa, Inaba, Hikojima***.
- H antigen .
- Little value in identification

- **Pathogenesis and Clinical features:** Route of infection is fecal-oral route.

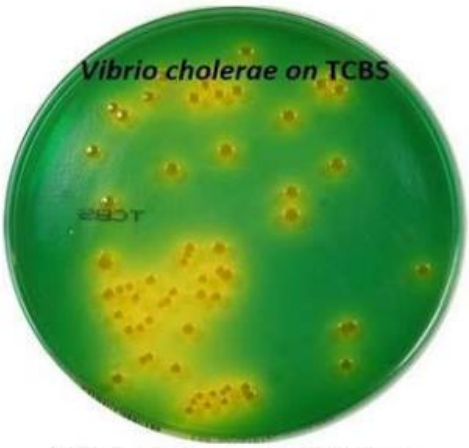
After ingestion of the V.cholerae-01, the bacteria adheres to the intestinal wall without invasion then produces an exotoxin causing excessive fluid secretion and diminished fluid absorption resulting in diarrhea (rice water stool) which is characterized by passage of voluminous watery diarrhea containing vibrios, epithelial cells and mucus; and result in severe dehydration. Cholera toxin is protein bound Gs protein. Gs protein activates adenylate cyclase, which produces elevated levels of intracellular cAMP. This, in turn, causes an outflowing of ions and water to the lumen of the intestine.

- Laboratory diagnosis:

- Specimen: Stool flecks Smear: Gram-negative motile curved rods. Motility of vibrios is best seen using dark-field microscopy.
- Presumptive diagnosis: Inactivation of vibrios in a wet preparation after adding vibrio antiserum.

- Culture:

1. TCBS (thiosulphate citrate bile salt sucrose agar) media. Selective media for primary isolation of V.cholerae. . Observe for large yellow sucrose-fermenting colonies after 18-24 hrs. of incubation.



Vibrio cholerae on TCBS Agar

Vibrio parahaemolyticus on TCBS Agar

2. Alkaline peptone water: Enrichment media for V.cholerae-01 Growth on and just below the surface of peptone water within 4-6 hours at room temperature as well as 37 ºC.

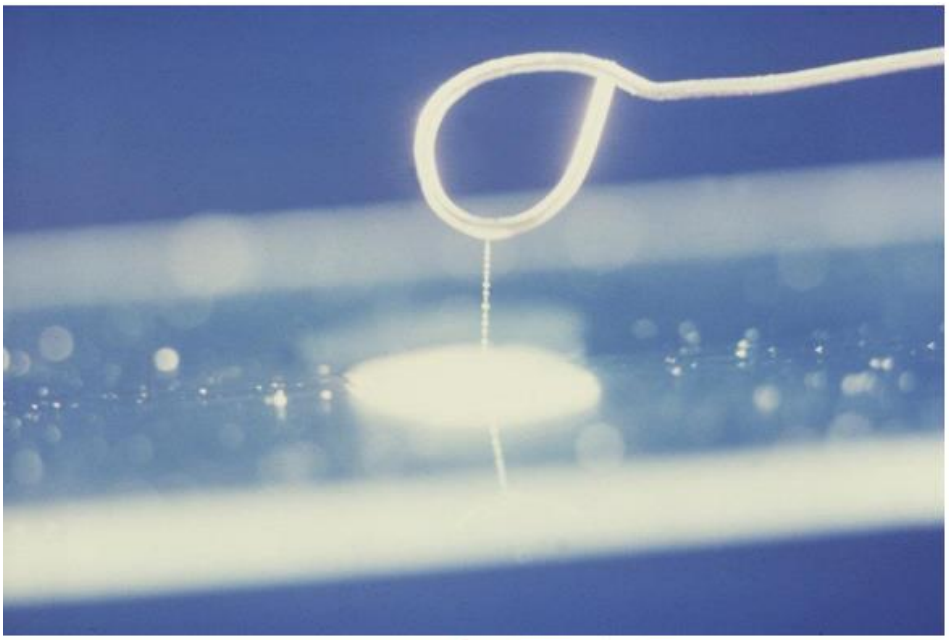
- Biochemical Reaction: . Oxidase-positive. . Ferment sucrose and maltose(acid; no gas). . Do not ferment L-arabinose.

- String test:

• A single colony or A loopful of vibrio liquid the growth is mixed with a drop of 0.5 % sodium deoxycholate or sodium taurocholate on a slide or petri dish.

• If the test is positive, the sodium deoxycholate or sodium taurocholate will lysis the bacteria cells, leading to release the DNA from the sells making the suspension mucoid and form a 'string' when taken by loop.

• This test differentiate V. cholera (string +ve) from other vibrio species (string –ve).



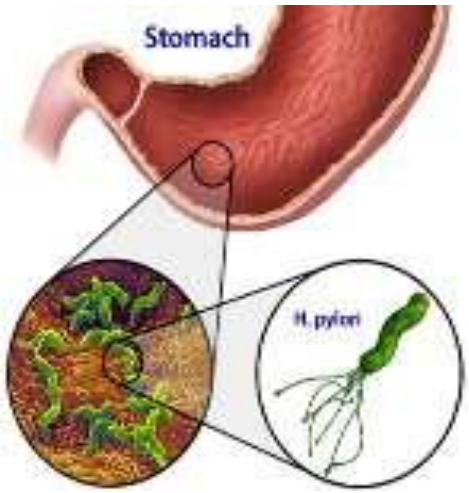
String test of *Vibrio cholerae*

- Treatment: Sensitive to tetracycline and chloramphenicol. Fluid and electrolyte replacement are the first line of management for cholera.

GENUS : *Helicobacter pylori*

- **General characteristics:**

-Spiral-shaped gram negative, microaerophilic, motile rods with polar flagella.
-Antigenic structure: Pili, Protease and Urease.



-Pathogenesis and clinical features:

-Route of entry: Ingestion of contaminated food and drinks Familial clustering of H. pylori infection occurs .

- Type B chronic gastritis.
- Peptic ulcer disease (gastric and duodenal ulcer).
- Gastric carcinoma.
- Gastric lymphoma.
- Transmission of H. pylori is thought to be from person to person; the organism has not been isolated from food or water. Untreated, infections tend to be chronic, even lifelong. H. pylori colonizes gastric mucosal (epithelial) cells in the stomach, and duodenum or esophagus only. The organism survives in the mucous layer that coats the epithelium, and causes chronic inflammation of the mucosa. the organism is non-invasive. Initial infection with H. pylori causes acute gastritis. Urease released by H. pylori produces ammonia ions that neutralize stomach acid in the vicinity of the organism, favoring bacterial multiplication.

- Virulence Factors:

- Urease (major virulence factor), Urease produces ammonia that damages gastric mucosa, ammonia also neutralizes acid pH; which allows the organism to live in the stomach.
- Flagella for adhesions.
- Protease.
- Exotoxins (inhibit stomach acid production).
- LPS (damage mucosal cells)

- **Lab. Diagnosis:**
- **Specimen:** Gastric biopsy, serum Smear: Giemsa or silver stain.
- **Culture:** Skirrow's media Translucent colonies after 7 days of incubation.
- **Biochemical reaction:**
- Catalase positive .
- Oxidase positive .
- Urease positive.

- **Serology:** Detection of antibodies in the serum specific for H. pylori .
- Detection of H. pylori antigen in stool specimen.

- **Urea breath test**
- **Treatment:** Triple or quadruple therapy: . Amoxicillin + clarithromycin/ metronidazole + Proton pump inhibitors (PPI (Omeprazole or lansoprazole)) or .Metronidazole + Bismuth subsalicylate/ Bismuth subcitrate + Amoxicillin / Tetracycline + PPI.
- **Prevention and control:** Improving sanitary hygiene.