










Cholera

-  A life-threatening secretory diarrhea induced by enterotoxin secreted by *V. cholerae*
-  Water-borne illness caused by ingesting water/food contaminated by copepods infected by *V. cholerae*
-  An enterotoxic enteropathy (a non-invasive diarrheal disease)
-  A major epidemic disease

V. cholerae






-  Transmitted by fecal-oral route
-  Endemic in areas of poor sanitation (India and Bangladesh)
-  May persist in shellfish or plankton
-  7 pandemics since 1817 – first 6 from Classical strains, 7th from El Tor
-  1993: Cholera in Bengal caused by O139 – may be cause of 8th pandemic

Profile of *vibrio cholerae*

- Gram-negative
- Highly motile; polar flagellum
- Brackish rivers, coastal waters
 - Associate with plankton and algae
- Proliferate in summers
- Produce Cholera toxin
- Pathogenic and nonpathogenic strains
 - 206 serogroups



Transmission

-  **Contaminated food or water**
-  **Inadequate sewage treatment**
-  **Lack of water treatment**
-  **Improperly cooked shellfish**
-  **Transmission by casual contact unlikely**

People Most at Risk

■ People with low gastric acid levels

- Children: 10x more susceptible than adults

- Elderly

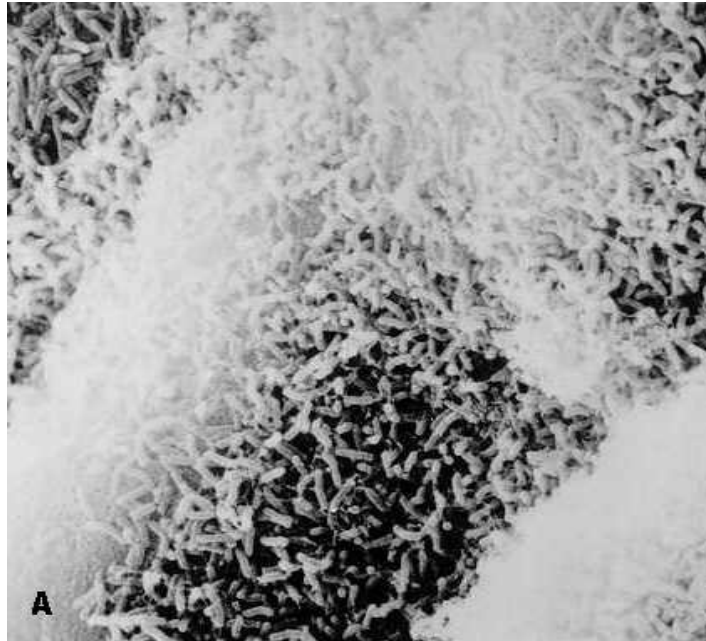
■ Blood types

- O more risk to infection >> B > A > AB less








Incubation

- Ranges from a few hours to 5 days
- Average is 1-3 days
- Shorter incubation period:
 - High gastric pH (from use of antacids)
 - Consumption of high dosage of cholera



How Does Cholera Toxin Work?

-  **G proteins stuck in “On” position**
-  **100 fold increase in cAMP**
-  **Activation of ion channels**
-  **Ions flow out and water follows**
-  **[animation](#)**

Symptoms




- Occur 2-3 days after consumption of contaminated food/water
- Usually mild, or no symptoms at all
 - 75% asymptomatic
 - 20% mild disease
 - 2-5% severe
- Vomiting
- Cramps
- Watery diarrhea (1L/hour)
- Without treatment, death in 18 hours-several days

Consequences of Severe Dehydration

- Intravascular volume depletion
- Severe metabolic acidosis
- Hypokalemia
- Cardiac and renal failure
- Sunken eyes
- Almost no urine production



Mortality Rate

-  Causes 120,000 deaths/year worldwide
-  With prompt rehydration: <1%
-  Without treatment: 50%-60%

Treatment

Even before identifying cause of disease, rehydration therapy must begin Immediately because death can occur within hours

 **Oral rehydration**

 **Intravenous rehydration**

 **Antimicrobial therapy**

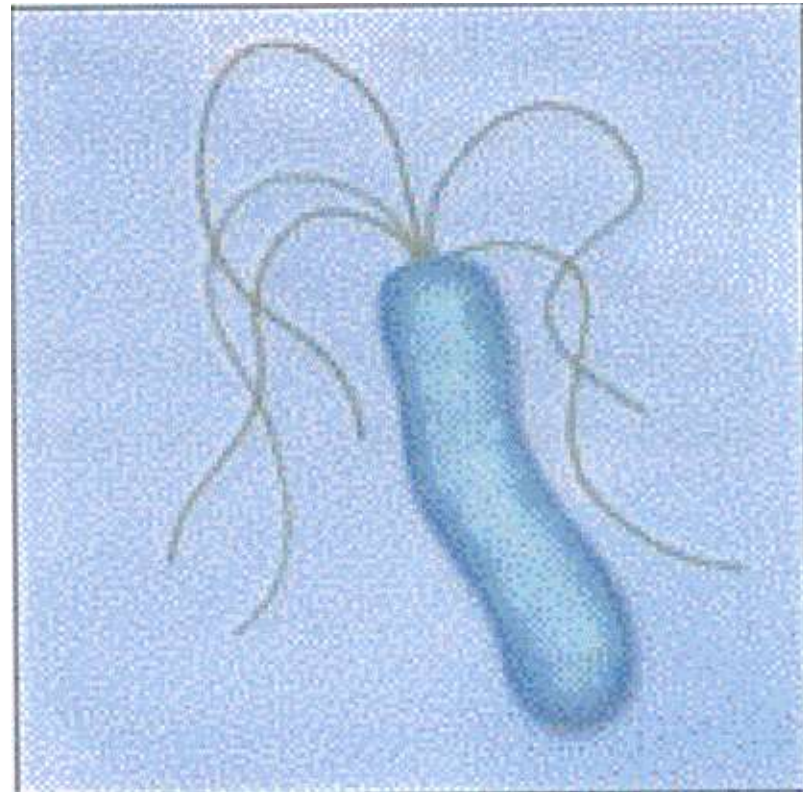
Helicobacter pylori

Abdulelah A Almayah

Helicobacter pylori

History

- In 1982 ,spiral-shaped bacterium from gastric biopsies of patients with gastritis was isolated
- The discovery was by Dr.Robin Warren and Dr.Barry Marshall



Epidemiology

- Infection occurs worldwide
- Overall *prevalence* strongly correlates with socio-economic conditions
- In Middleaged adults in developing countries prevalence is 80%, in industrialised countries 20-50% (rate of acquisition decreasing)
- *Acquisition*: Oral Ingestion of the bacterium
- *Transmission*: Within families in early childhood, not isolated from water etc, e

Pathogenesis

- *H. pylori* is found only on gastric epithelium where the organisms tend to cluster around the junctions between cells and virtually never penetrate the cells themselves.
- *H. pylori* is able to survive in the gastric environment which is hostile to growth of most bacteria.

Standard triple therapy-Eradication therapy, which is probably the most widely used treatment for eradication of *H. pylori* for 7 days minimal

Proton pump inhibitor B.D. (e/g Lansoprazole 30 mg BD)

+

Clarithromycin 500mg B.D.

+

Amoxicillin 1g B.D.

Or If penicillin allergic

Proton pump inhibitor B.D. (e/g Lansoprazole 30 mg)

Clarithromycin 500mg B.D.

Metronidazole 400 mg B.D

If treatment failure refer to Gasterenterologist

BRUCELLOSIS

Etiology

- *Brucella*:
 - *Brucella abortus*(infect Cattle),
Brucella melitensis (infect
Sheep,Goat)
 - *Brucella suis*(Swin),Canins(Dog)
- Brucella* are
- G-ve Coccobacilli
 - Aerobic, Non-spore forming
 - Non motile
 - Grown on Blood or Chocolate
agar



Clinical Manifestations

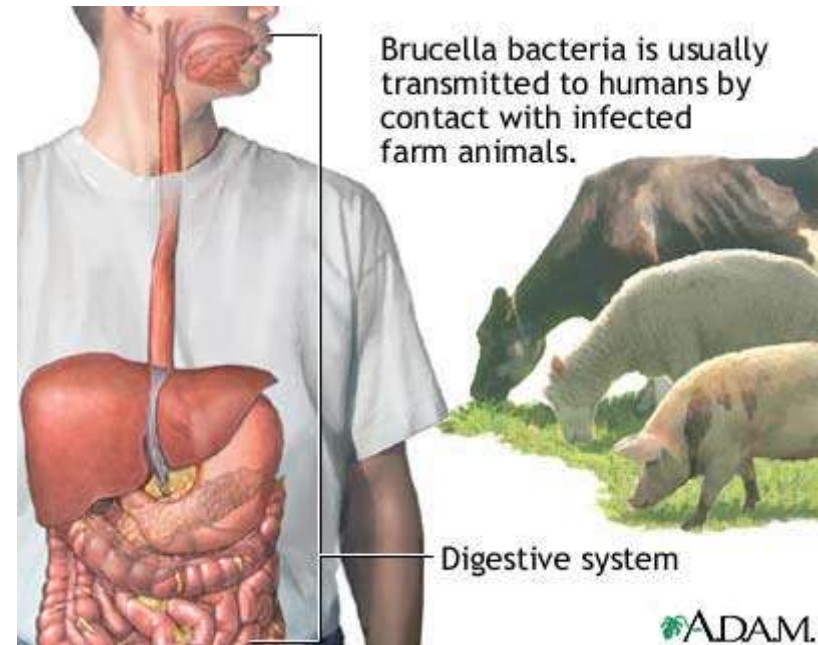
- **GIT** : anorexia, abd. pain, vomiting, diarrhea, constipation, hepatosplenomegaly.
- **LIVER** : Involved in most cases .
 - granulomas (*B. abortus*).
 - hepatitis (*B. melitensis*).
 - abscesses (*B. suis*).

Clinical Manifestations

- **Neurologic**
 - Meningitis, encephalitis, radiculopathy & peripheral neuropathy, intracerebral abscesses
 - Meningitis
 - acute or chronic
 - neck rigidity < 50%

Epidemiology

- Unpasteurized milk
- Occupational events



Pertussis

(Whooping Cough or Hundred Day
Cough)

Epidemiology of Pertussis

Mode of transmission

- Person to person via
 - Aerosolized droplets from cough or sneeze
 - Direct contact with secretions from respiratory tract of infectious person
- 80% - secondary attack rate
- Older children and adults are important sources of disease for infants and young children
- Infants <12 months of age greatest risk for complications and death
-

Epidemiology of Pertussis

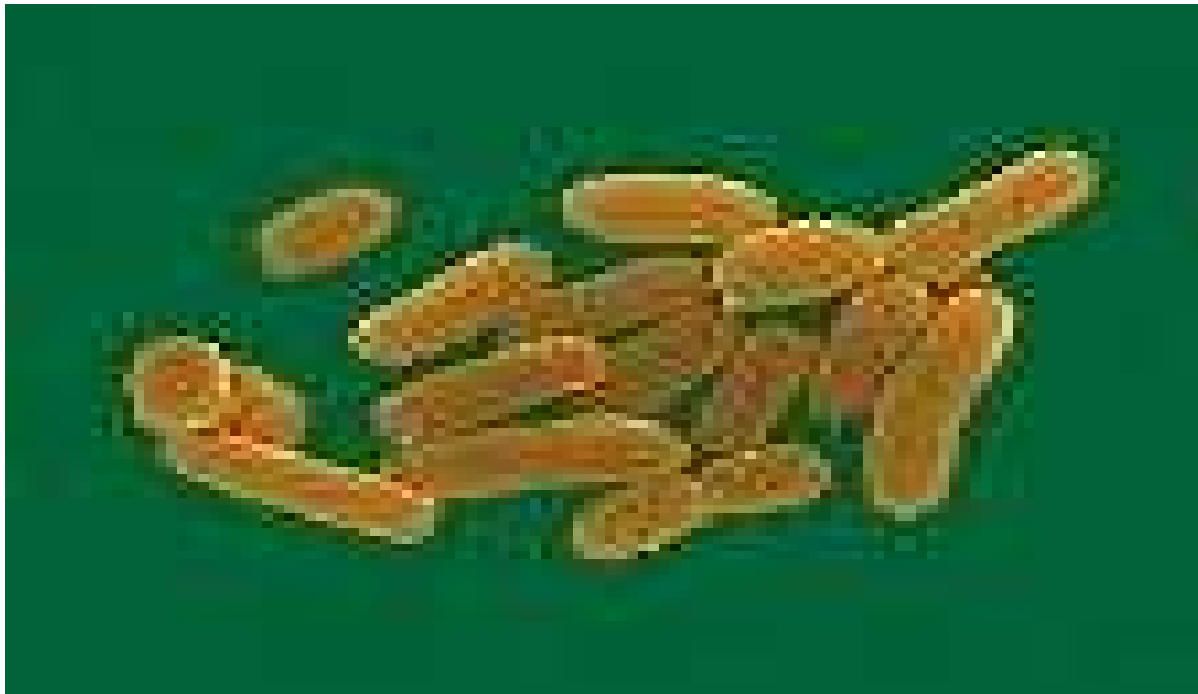
- **Reservoir** - Humans
- **Incubation period** – 7-10 days (5-21 days).
- **Infectious period** – Most contagious during the first 2 weeks after cough onset
- **Duration of illness:**
 - Children: 6-10 wks.
 - ~ 1/2 of Adolescents: 10 wks or longer

Yersinia pestis



- Member of the *Enterobacteriaceae* family

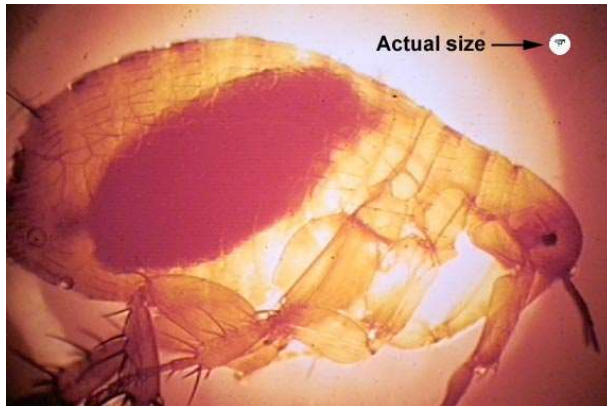
Yersinia is a Gram-negative coccobacilli



Ecology and Infection Process

Y. pestis multiply into intestinal Flea.

Some *Y. pestis* in the flea infect next blood meal thus transferring the infection to a new host.



A few bacilli are taken up by tissue macrophages after they lose their capsular layer. Macrophages can't kill *Y. pestis* and provide protected environment for bacilli so they can re-synthesize their capsular layer.

The re-encapsulated organisms then kill the macrophage and are released into the extracellular environment where they travel to draining lymph nodes.



Symptoms

Bubonic Plague

bacteria infect lymph nodes

- Bubos

- Fever
- Headache
- Vomiting Blood



SYPHILIS

- **Caused by *Treponema pallidum*.**
- **Transmission: sexual; maternal-fetal, and rarely by other means.**
- **A dramatic increase in cases in men from 2000 to 2002 reflected syphilis in MSM.**
- **Syphilis increases the risk of both transmitting and getting infected with HIV**

STAGES OF SYPHILIS

1. Primary

2. Secondary

3. Latent

- Early latent
- Late latent

4. Late or tertiary

- May involve any organ, but main parts are:
 - Neurosyphilis
 - Cardiovascular syphilis
 - Late benign (gumma)

Oral chancres in primary syphilis



Late syphilis - serpiginous gummata of forearm



Late syphilis - ulcerating gumma





Treponema pallidum Dark field examination of exudate from a penile ulcer (x1000) in a patient with syphilis. The spirochete Treponema pallidum, which is too small to be seen using ordinary microscopy, appears as a delicate spiral rod when dark field illumination is employed. Courtesy of Harriet Provine.