# EPIDEMIOLOGY OF CHOLERA

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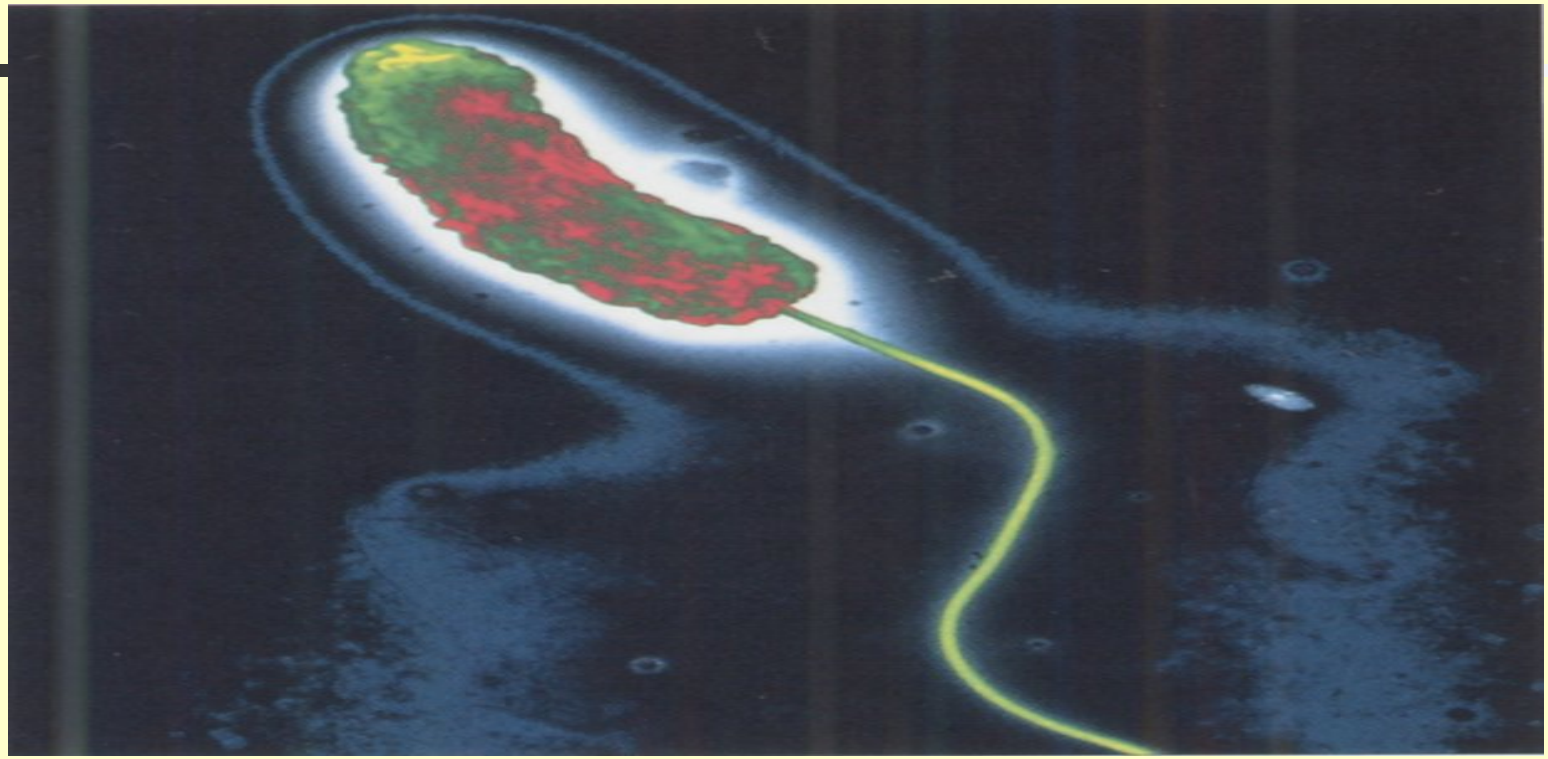


# V. CHOLERAE

- The organism is a comma-shaped, gram-negative, aerobic bacillus whose size varies from 1-3 mm in length by 0.5-0.8 mm in diameter.

- Its antigenic structure consists of a flagellar H antigen and a somatic O antigen. It is the differentiation of the latter that allows for separation into pathogenic and nonpathogenic strains.

# EPIDEMIOLOGY

- Since 1817, there have been 7 cholera pandemics. The first 6 occurred from 1817-1923 and were caused by V. cholerae, the classical biotype. The pandemics originated in Asia with subsequent spread to other continents.

- The seventh pandemic began in Indonesia in 1961 and affected more countries and continents than the previous 6 pandemics. It was caused by V. cholerae El Tor.

- In October 1992, an epidemic of cholera emerged from Madras, India as a result of a new serogroup (0139). Some experts regard this as an eighth pandemic.

- This Bengal strain has now spread throughout Bangladesh, India, and neighboring countries in Asia.

- Crowding & gathering of people during religious rituals (e.g. Muslims pilgrimage to Mecca or Hindu swimming festivals in holy rivers) enhance the spread of infection.
- Index cases when travelled back to their homes may pass the organism to at risk individuals leading to secondary epidemic or small scale infection.

# REPORTED CASES

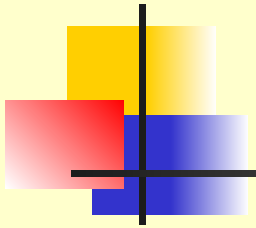
- The number of cholera patients worldwide is uncertain because many cases are unreported.
- The number of cases is increased during epidemics & is affected by environmental factors.
- In 1994, 94 countries reported 385,000 cases of cholera to WHO, but the number reported in 1998 was 121,000. 89% of these cases were reported from Africa.

# PATHOGENESIS

- V cholerae cause clinical disease by producing an enterotoxin that promotes the secretion of fluid and electrolytes into the lumen of the gut.

- The result is watery diarrhea with electrolyte concentrations isotonic to those of plasma.

- The enterotoxin acts locally & does not invade the intestinal wall. As a result few WBC & no RBC are found in the stool.



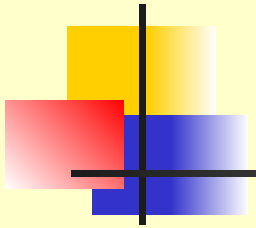
- **Fluid loss originates in the duodenum and upper jejunum; the ileum is less affected.**

- **The colon is usually in a state of absorption because it is relatively insensitive to the toxin.**

- **The large volume of fluid produced in the upper intestine, however, overwhelms the absorptive capacity of the lower bowel, which results in severe diarrhea.**

# TRANSMISSION

- Cholera is transmitted by the fecal-oral route through contaminated water & food.
- Person to person infection is rare.
- The infectious dose of bacteria required to cause clinical disease varies with the source. If ingested with water the dose is in the order of $10^3$-$10^6$ organisms. When ingested with food, fewer organisms are required to produce disease, namely $10^2$-$10^4$.



- V. cholerae is a saltwater organism & it is primary habitat is the marine ecosystem.
- Cholera has 2 main reservoirs, man & water. Animals do not play a role in transmission of disease.
- V. cholerae is unable to survive in an acid medium. Therefore, any condition that reduces gastric acid production increases the risk of acquisition.

# HOST SUSCEPTIBILITY

- The use of antacids, histamine-receptor blockers, and proton-pump inhibitors increases the risk of cholera infection and predisposes patients to more severe disease as a result of reduced gastric acidity.

- The same applies to patients with chronic gastritis secondary to Helicobacter pylori infection or those who have had a gastrectomy.

# AT RISK GROUPS

- All ages but children & elderly are more severely affected.
- Subjects with blood group “O” are more susceptible; the cause is unknown.
- Subjects with reduced gastric acid.

# CLINICAL PICTURE

- Incubation period is 24-48 hours.
- Symptoms begin with sudden onset of watery diarrhea, which may be followed by vomiting. Fever is typically absent.
- The diarrhea has fishy odor in the beginning, but became less smelly & more watery over time.

- The classical textbook “rice water” diarrhea, which describes fluid stool with very little fecal material, appears within 24h from the start of the illness.

- In severe cases stool volume exceeds 250 ml /kg leading to severe dehydration, shock & death if untreated.

# CHOLERA IN CHILDREN

- Breast-fed infants are protected.
- Symptoms are severe & fever is frequent.
- Shock, drowsiness & coma are common.
- Hypoglycemia is a recognized complication, which may lead to convulsions.
- Rotavirus infection may give similar picture & need to be excluded.

# LAB DIAGNOSIS

- Organism can be seen in stool by direct microscopy after gram stain and dark field illumination is used to demonstrates motility.

- Cholera can be cultured on special alkaline media like triple sugar agar or TCBS agar.

- Serologic tests are available to define strains, but this is needed only during epidemics to trace the source of infection.

# OTHER LAB FINDINGS

- Dehydration leads to high blood urea & serum creatinine. Hematocrit & WBC will also be high due to hemoconcentration.

- Dehydration & bicarbonate loss in stool leads to metabolic acidosis with wide-anion gap.

- Total body potassium is depleted, but serum level may be normal due to effect of acidosis.

# TREATMENT

- The primary goal of therapy is to replenish fluid losses caused by diarrhea & vomiting.

- Fluid therapy is accomplished in 2 phases: rehydration and maintenance.

- Rehydration should be completed in 4 hours & maintenance fluids should replace ongoing losses & provide daily requirement.

# FLUID THERAPY

- Ringer lactate solution is preferred over normal saline because it corrects the associated metabolic acidosis.

- IV fluids should be restricted to patients who purge >10 ml/kg/h & for those with severe dehydration.

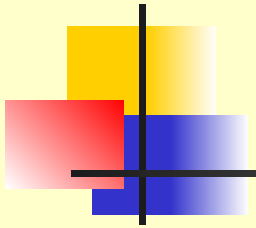
- The oral route is preferred for maintenance & the use of ORS at a rate of 500-1000 ml/h is recommended.

# DRUG THERAPY

- The goals of drug therapy are to eradicate infection, reduce morbidity and prevent complications.

- The drugs used for adults include tetracycline, doxycycline, cotrimoxazole & ciprofloxacin.

- For children erythromycin, cotrimoxazole and furazolidone are the drugs of choice.



- Drug therapy reduces volume of stool & shortens period of hospitalization. It is only needed for few days (3-5 days).

- Drug resistance has been described in some areas & the choice of antibiotic should be guided by the local resistance patterns .

- Antibiotic should be started when cholera is suspected without waiting for lab confirmation.

# COMPLICATIONS

- If dehydration is not corrected adequately & promptly it can lead to hypovolemic shock, acute renal failure & death.
- Electrolyte imbalance is common.
- Hypoglycemia occurs in children.
- Complications of therapy like over hydration & side effects of drug therapy are rare.

# PUBLIC HEALTH ASPECTS

- Isolation & barrier nursing is indicated
- Notification of the case to local authorities & WHO.
- Trace source of infection.
- Resume feeding with normal diet when vomiting has stopped & continue breastfeeding infants & young children.

# PREVENTION

- Education on hygiene practices.
- Provision of safe, uncontaminated, drinking water to the people.
- Antibiotic prophylaxis to house-hold contacts of index cases.
- Vaccination against cholera to travellers to endemic countries & during public gatherings.

# CHOLERA VACCINES

- The old killed injectable vaccine is obsolete now because it is not effective.
- Two new oral vaccines became available in 1997. A Killed & a live attenuated types.
- Both provoke a local immune response in the gut & a blood immune response.
- Cholera vaccination is no more required for international travellers because risk is small.