

Cholera

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Etiology

Cholera results from infection by *Vibrio cholerae*, a Gram negative, facultatively anaerobic rod in the family Vibrionaceae. Two serogroups, O1 and O139 (“Bengal”), can cause disease. Serogroup O1 contains two serologically indistinguishable biotypes, classical and El Tor. Mild or asymptomatic infections are seen more often with the El Tor biotype. Cholera caused by serogroup O139 emerged in 1992, in epidemics in India and Bangladesh.

Geographic Distribution

Cholera is endemic in the Middle East, Africa, Central and South America, parts of Asia and the Gulf Coast of the United States but outbreaks can occur in any country. Major epidemics occur periodically in less developed countries; outbreaks in developed countries are usually localized due to better sanitation.

Transmission

Transmission is by the fecal–oral route. Infections are particularly common after ingesting contaminated water or food. Cases are occasionally seen in people who have eaten raw or undercooked shellfish, particularly oysters, from contaminated waters.

V. cholerae is excreted in the feces and vomitus. Viable organisms can be found in feces for up to 50 days, on glass for up to a month, on coins for a week, in soil or dust for up to 16 days and on fingertips for 1 to 2 hours. Bacteria survive well in water and may remain viable in shellfish, algae or plankton in coastal regions.

Disinfection

V. cholerae is susceptible to many disinfectants, including 0.05% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde, 8% formaldehyde, 10% hydrogen peroxide and iodine–based disinfectants. Organisms are tolerant of alkaline conditions but are sensitive to acids and cold temperatures.

Infections in Humans

Incubation Period

The incubation period is a few hours to 5 days. Most infections become apparent after 2 to 3 days.

Clinical Signs

Cholera appears abruptly with painless, watery diarrhea, sometimes accompanied by vomiting. Infections may be subclinical, mild and self–limiting, or fulminant and severe. Severe fluid loss can be seen in more serious cases; thirst, oliguria, severe dehydration, acidosis, muscle cramps and shock may result. Most cases last approximately 2 to 7 days but death may occur within a few hours if the fluid loss is high. A self–limiting gastroenteritis is seen after infection with pathogenic non O1/O139 strains.

Communicability

Yes. Most people excrete *V. cholerae* while diarrhea is present and for a few days after recovery. A few individuals carry the organism in the gall bladder for several months. Rare long–term carriers have been reported; one woman was an asymptomatic carrier for 12 years before her infection spontaneously resolved.

Diagnostic Tests

Cholera can be diagnosed by observing the organism’s characteristic motility during direct, bright–field or dark–field microscopic examination of the feces; the addition of specific antibodies to *V. cholerae* stops the movement. Bacteria can also be identified in the feces by immunofluorescence. A polymerase chain reaction (PCR) assay or other genetic tests may be available in some laboratories.



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V. cholerae can be isolated from feces or rectal swabs. Distinctive yellow colonies are seen on selective thiosulfate–citrate–bile salts–sucrose (TCBS) agar. Other selective and nonselective media, including nutrient agar and bile salts agar, can also be used. Enrichment procedures and selective media may be necessary to identify carriers. Identification is by rapid slide agglutination test or immunofluorescence. Biochemical tests may also be helpful, particularly for non-O group 1 (nonagglutinable) vibrios. The “string test” – the development of a mucoid string when a colony is emulsified in 0.5% aqueous sodium deoxycholate – may also be useful. The classic and El Tor biotypes can be differentiated by chicken cell hemagglutination, hemolysis, polymyxin sensitivity or susceptibility to bacteriophages.

A rising titer is also diagnostic. Serologic tests include agglutination tests, complement fixation and tests to detect antitoxic antibodies. Enzyme –linked immunosorbent assays (ELISAs) and passive hemagglutination may also be available.

Treatment and Vaccination

Treatment relies on fluid replacement and the restoration of electrolyte balance. Antibiotics reduce the stool volume, decrease shedding of organism and shorten the course of the disease but may not be effective alone.

Vaccines have been developed but their efficacy is limited; some vaccines are effective for only short periods of time, particularly in children.

Morbidity and Mortality

Endemic cholera and epidemics are seen periodically in susceptible populations where sanitation and environmental conditions favor the spread of *V. cholerae*. In developed countries with good sanitation, outbreaks are usually limited. Approximately 0 to 5 cases occur annually in the United States.

Cholera is rarely fatal if the lost fluids and electrolytes are adequately replaced. The mortality rate with proper treatment is less than 1% and most patients recover within 3 to 7 days. In untreated cases, the case fatality rate is greater than 50%. Death may occur within a few hours if the diarrhea is severe.

Infections in Animals

Humans appear to be the only natural host for *V. cholerae*. Diarrhea can be induced in experimentally infected rabbits, mice and chinchillas by bacteria or their toxins. Dogs are susceptible if massive doses of bacteria are given.

Internet Resources

Centers for Disease Control and Prevention (CDC)
<http://www.cdc.gov/cholera/index.html>

Material Safety Data Sheets –Canadian Laboratory Center for Disease Control
<http://www.hc-sc.gc.ca/pphb-dgsp/psdms-ftss/index.html#menu>

Medical Microbiology
<http://www.ncbi.nlm.nih.gov/books/NBK7627/>

The Merck Manual
<http://www.merck.com/pubs/mmanual/>

U.S. FDA Foodborne Pathogenic Microorganisms and Natural Toxins Handbook (Bad Bug Book)
<http://www.fda.gov/food/foodsafety/foodborneillness/foodborneillnessfoodbornepathogensnaturaltoxins/badbugbook/default.htm>

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