



Cholera – management and prevention

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Summary Cholera is an acute secretory diarrhoeal infection caused by the bacterium *Vibrio cholerae*. It is likely to have originated in the Indian sub-continent; however, it spread to cause six worldwide pandemics between 1817–1923. The ongoing seventh worldwide pandemic of cholera began in 1961. The intensity, duration and severity of cholera epidemics have been increasing, signaling the need for more effective control and prevention measures. The response to the cholera pandemics of the 19th century led to the development of safe and effective sanitation and water systems which have effectively removed the risk of cholera in many settings. However, such systems are not in place to protect billions of people worldwide. Although some progress has been made in expanding access to water in recent years, achieving optimal infrastructure will, in the most optimistic scenario, take decades. Climate change, extreme weather events and rapid urbanisation suggests that alternatives to the current paradigm of providing large centralised water and sanitation systems should be considered, including smaller decentralised systems. The aim of this review paper is to provide an overview of current knowledge regarding management of cholera with a focus on prevention measures including vaccination and water and sanitation interventions.

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Introduction

Cholera is an acute enteric infection caused by the gram negative bacterium *Vibrio cholerae*, a member of the vibriaceae family.¹ It causes a profuse secretory diarrhoea that can lead to rapid dehydration, hypovolaemia and death if not treated. In 2014, the World Health Organization

reported a total of 190,549 cases of cholera. 55% of these reported cases occurred in Africa. The total case fatality rate was estimated by WHO to be 1.17%.² These reported figures are likely to be a gross underestimate of the true burden of cholera disease. Fear of negative impact on travel and trade, limitations in surveillance systems, inconsistencies in case definitions and lack of lab capacity

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all contribute to underreporting of cholera cases.³ The true burden of disease is estimated to be between 1.4 and 4.3 million cases/year.²

History

Sanskrit writings from the Sushruta Samhita in India as early as 500–400 BC describe an illness that resembles cholera. It is thought to have remained confined to the Indian sub-continent until the first global pandemic that began in 1817.⁴ It first reached Europe in 1829 and led to the formation of central and regional Boards for Health in Great Britain in preparation for the imminent arrival of the pandemic. It reached England in 1831 and large epidemics occurred between 1831–32 and 1848–49. In 1849 the English physician John Snow published his essay 'On The Mode of Communication of Cholera'. He discounted the widely held belief that cholera was spread by a 'miasma' coming from the river and proposed that cholera was a communicable disease and that infectious material was present in stool, that may be widely disseminated by the emptying of sewers into the drinking water of the community.⁵ Snow proposed a number of preventative measures including hand washing, treatment or filtration of drinking water and creation of a sewage free water supply,⁵ measures that remain key components of water and sanitation interventions to this day. Robert Koch later identified the bacterium and postulated that a toxin was responsible for the massive outpouring of fluid from the intestine.

The organism

The gram negative rods are found in coastal waters and estuaries.⁶ There are over 200 recognised serogroups. However, only two of these are associated with epidemic cholera, serogroups O1 and O139. Serogroup O1 can be further classified by its serotype (Ogawa/Inaba) and biotype (Classical or El Tor). Prior to the 7th global pandemic of cholera, the majority of cholera isolates were the Classical biotype of the O1 serogroup and El Tor isolates were responsible only for sporadic cases of diarrhoea. However, since 1961 the vast majority of isolates have been El Tor type.⁷ Since 1992 serogroup O139 Bengal has emerged as a human pathogen.⁸

Transmission

Since the early discoveries of Snow and Koch a huge amount has been discovered about the ecology and transmission of *V. Cholerae*. Cholera vibrios inhabit seas, estuaries, brackish waters, rivers and ponds in coastal areas. They thrive in saline water, however, they can also live in water of lower salinity if it is sufficiently warm and contains a high concentration of organic nutrients.⁹ *V. Cholerae* live in a commensal relationship with copepods.¹⁰ These tiny planktonic crustaceans serve as the normal host organism for vibrios. Copepods utilise phytoplankton as their food source. A study conducted in two coastal communities in cholera endemic areas of Bangladesh and India found that outbreaks were preceded by phytoplankton blooms.¹¹

The linkage between weather, phytoplankton blooms and the prevalence of cholera vibrios in the environment

suggests that climate change may affect cholera ecology and so its epidemiology. In 1992 the seventh pandemic of cholera reached Peru in South America and quickly spread along the entire coast from the Ecuadorian to the Chilean borders. This outbreak coincided with strong El Niño currents and plankton blooms.^{9,12} Thus it is likely that a complex interplay between macro and micro environmental factors including water temperature, salinity, nutrient concentrations and plankton all influence the amount of vibrios in environmental reservoirs.

Cholera can be transmitted to humans via ingestion of water contaminated with infected copepods. It can also be transmitted from person to person via the faecal-oral route and may be acquired from contaminated food or water. The stools of those afflicted by *V. cholerae* are extremely infectious as they contain up to 10⁹ vibrios per ml.¹³ *V. cholerae* genes coding for a hyperinfectious phenotype are expressed as the bacteria move towards the distal intestine, making *V. cholerae* excreted by human hosts more contagious than *V. cholerae* extant in a natural environment. After they are purged from the human intestine they remain in this hyperinfectious state in the environment for hours to days, and so can efficiently propagate an epidemic.^{14,15}

When *V. cholerae* is ingested, the organisms must withstand the acidic environment of the stomach in order to colonise the intestines. In a healthy individual, the infectious dose of bacteria is estimated to be 10⁵–10⁸ organisms. However, hypochlorhydria as a result of malnutrition, or medications to reduce gastric acid can lower the infectious dose. Host factors make some people more susceptible to infection, in particular persons with blood group O are significantly more likely to develop severe cholera disease.^{16,17}

Epidemiology

Endemic cholera

Cholera occurs in both endemic and epidemic patterns. It is endemic in 69 countries in Asia and Africa where an estimated 1.3 billion people are living at risk of cholera.¹⁸ In 2013, over 190,000 cholera cases were reported to the WHO from 47 different countries, this is likely to be a gross underestimate of the true burden of disease.² Countries with estimates of more than 100,000 cases annually include India, Ethiopia, Nigeria, Haiti, The Democratic Republic of the Congo, Tanzania, Kenya and Bangladesh.¹⁸ It can occur seasonally in some endemic settings and is temporally associated with monsoon rains and floods in the Asian subcontinent.¹⁹ In endemic settings children bear the greatest burden of disease.²⁰

Epidemic cholera

Cholera epidemics occur superimposed on endemic disease in long cycles. These cycles are determined by waning levels of population immunity and periods of climate variability.¹ When introduced into a cholera naive population, large epidemics can occur such as the ongoing Haitian epidemic that began in 2010. In epidemic settings where there is little or no natural immunity present, all age groups are equally affected by the disease. Epidemics occur unpredictably and are often associated with natural disasters and humanitarian

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