

CURRENT PERSPECTIVES AND CLINICAL CHALLENGES IN CHOLERA MANAGEMENT: AN EVIDENCE-BASED REVIEW FOR NURSING PRACTICE

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ABSTRACT

Background: Cholera remains a major global public health concern despite being a preventable and treatable infectious disease. Caused by *Vibrio cholerae*, it continues to produce substantial morbidity and mortality in regions with poor sanitation, inadequate access to safe water, humanitarian crises, and fragile healthcare systems. Nurses play a pivotal role in early recognition, prompt rehydration, infection prevention, patient education, and outbreak response. **Aim:** This review aimed to provide an evidence-based overview of the current perspectives on cholera, emphasizing its epidemiology, bacteriology, clinical manifestations, diagnosis, management, prevention, and the critical role of nursing practice in improving patient outcomes and supporting public health interventions. **Methods:** A comprehensive narrative review of contemporary literature and international evidence was conducted. Current recommendations from global health authorities and peer-reviewed publications were synthesized to examine advances in cholera epidemiology, pathogenesis, diagnostic approaches, clinical management, preventive strategies, and emerging challenges relevant to nursing care. **Results:** Cholera continues to affect approximately 2.9 million individuals annually and causes nearly 95,000 deaths worldwide, with the greatest burden occurring in Africa and other resource-limited settings. Disease transmission remains closely associated with contaminated water, inadequate sanitation, poverty, and humanitarian emergencies. Rapid diagnosis, aggressive fluid and electrolyte replacement, appropriate antimicrobial therapy, and continuous clinical monitoring remain the cornerstones of successful management. Oral rehydration therapy has dramatically reduced mortality, while vaccination, improved water, sanitation, and hygiene (WASH) programs, surveillance systems, and early outbreak detection represent essential preventive strategies. Nurses are central to patient assessment, dehydration monitoring, infection control, health education, vaccination support, and multidisciplinary coordination during outbreaks. **Conclusion:** Effective cholera control requires integration of evidence-based clinical management with comprehensive public health interventions. Strengthening nursing practice, expanding access to clean water and sanitation, improving surveillance, enhancing vaccination programs, and promoting global collaboration remain fundamental to reducing disease burden and achieving long-term cholera elimination.

KEYWORDS: Cholera, *Vibrio cholerae*, nursing practice, dehydration, oral rehydration therapy, epidemiology, infection prevention, public health, WASH, oral cholera vaccine.

INTRODUCTION

Cholera is a severe acute enteric infection caused by the Gram-negative bacterium *Vibrio cholerae* and remains one of the most important waterborne diseases affecting global public health. The disease is characterized by the rapid onset of profuse watery diarrhea, vomiting, and severe dehydration, which can progress to hypovolemic

shock and death if prompt treatment is not initiated. Despite major advances in sanitation, healthcare infrastructure, and disease prevention, cholera continues to cause recurrent outbreaks in many low- and middle-income countries where access to safe drinking water, adequate sanitation, and effective healthcare services remains limited. The disease disproportionately affects

vulnerable populations living in conditions of poverty, overcrowding, humanitarian crises, armed conflicts, and natural disasters, where disruption of water and sanitation systems facilitates rapid transmission. Consequently, cholera continues to represent a significant challenge for healthcare systems and international public health organizations because of its potential for rapid spread, high morbidity, and preventable mortality.^[1,2] The historical significance of cholera extends over two centuries and has profoundly influenced the development of modern epidemiology and infectious disease control. Numerous epidemics caused by *Vibrio cholerae* have been documented throughout history, with one of the earliest recognized outbreaks originating in the Ganges Delta during the early nineteenth century. From this region, the first documented cholera pandemic began in 1817 and rapidly expanded beyond Asia to affect large areas of Africa, Europe, and Australia through international trade and population movement. Since that time, seven major pandemics have been recorded, each contributing substantially to global disease burden and advancing scientific understanding of disease transmission and prevention.^[1,2] During the third pandemic in 1853, the pioneering epidemiologist John Snow demonstrated that cholera was transmitted through contaminated water rather than airborne exposure, laying the foundation for modern epidemiological investigation and public health intervention.^[1,3] Shortly thereafter, Filippo Pacini successfully identified *Vibrio cholerae* as the causative organism, although his groundbreaking discovery was not formally recognized until more than a century later in 1966.^[3]

The majority of historical cholera pandemics were caused by *Vibrio cholerae* serogroup O1 of the classical biotype. However, the epidemiological landscape evolved considerably with the emergence of new pathogenic variants. In 1992, a major epidemic across Asia was associated with the emergence of the O139 serogroup, demonstrating the organism's capacity for genetic evolution and adaptation. Earlier, at the onset of the ongoing seventh pandemic in 1961, the El Tor biotype replaced the classical strain and rapidly became the predominant global variant because of its enhanced environmental persistence and greater capacity for transmission. The El Tor strain initially spread throughout Asia before expanding into Africa, the Middle East, and the former Soviet Union during the 1970s, eventually reaching Latin America in the early 1990s and later contributing to major outbreaks in Hispaniola in 2010.^[2] Current epidemiological evidence indicates that cholera continues to impose a substantial global health burden. Recent estimates suggest that approximately 2.9 million cholera cases and nearly 95,000 deaths occur annually worldwide, highlighting the persistent impact of this preventable disease. Africa experiences the greatest burden, accounting for nearly 60% of reported cases and approximately 68% of cholera-related deaths, reflecting persistent disparities in

water quality, sanitation, healthcare accessibility, and socioeconomic development.^[4] Nevertheless, the true global burden is likely considerably higher than reported figures because surveillance systems remain inadequate in many endemic regions. Furthermore, concerns regarding potential political, social, and economic consequences often discourage accurate reporting of outbreaks, resulting in significant underestimation of disease incidence and mortality. These surveillance limitations hinder effective public health planning, delay outbreak response, and complicate efforts to implement comprehensive prevention and control strategies. Consequently, strengthening disease surveillance, improving access to clean water and sanitation, expanding vaccination programs, and enhancing healthcare preparedness remain essential priorities for reducing the global burden of cholera and preventing future epidemics.^[4]

Epidemiology

Cholera remains one of the most significant yet preventable infectious diseases worldwide and serves as a powerful indicator of persistent socioeconomic disparities, inadequate sanitation, and inequitable access to safe water and healthcare services. Although effective preventive measures and evidence-based treatment strategies have dramatically reduced mortality, cholera continues to disproportionately affect populations living in low- and middle-income countries, where poverty, overcrowding, weak healthcare infrastructure, and limited access to clean drinking water facilitate sustained disease transmission. The disease is closely associated with poor environmental hygiene and inadequate sewage disposal, making cholera not only a medical condition but also a reflection of broader public health, economic, and social inequalities. Consequently, the epidemiology of cholera is strongly influenced by environmental, demographic, political, and humanitarian factors that determine the risk of exposure and disease spread. Humans are the only known natural reservoir for *Vibrio cholerae*, and transmission occurs primarily through ingestion of food or water contaminated with human feces containing the organism. The incubation period is relatively short, ranging from approximately two hours to five days, allowing outbreaks to spread rapidly within susceptible populations. In healthy individuals, infection generally requires ingestion of a relatively large bacterial inoculum, estimated at approximately 10^8 organisms, because gastric acidity destroys many ingested bacteria before they reach the small intestine. Nevertheless, conditions that reduce gastric acidity, including malnutrition, chronic illness, or the use of acid-suppressive medications, increase susceptibility to infection. Although the majority of infected individuals remain asymptomatic, these persons continue to play an important epidemiological role because they may excrete *Vibrio cholerae* in their feces for several days. Individuals with symptomatic disease, particularly those experiencing the characteristic profuse "rice-water" diarrhea, may continue shedding the organism for two

weeks or longer, thereby contributing substantially to community transmission and sustained outbreaks.^[5]

The global epidemiology of cholera changed considerably with the emergence of the El Tor biotype in 1961, marking the beginning of the seventh cholera pandemic. Unlike previous pandemic strains, which were predominantly caused by the classical biotype of *Vibrio cholerae* O1, the El Tor strain first emerged in Sulawesi, Indonesia, and rapidly spread across Southeast Asia before becoming established throughout South Asia. Over time, the El Tor biotype replaced the classical strain as the dominant cause of annual cholera epidemics in countries such as India and Bangladesh because of its enhanced environmental persistence, increased survival outside the human host, and greater capacity for asymptomatic carriage. During subsequent decades, the seventh pandemic expanded into Africa, the Middle East, Europe, North America, and Japan. However, outbreaks in high-income countries remained relatively limited because of well-developed sanitation systems, effective wastewater management, reliable access to clean water, and efficient public health surveillance.^[6] A major epidemiological milestone occurred in 1991 when the seventh pandemic reached Peru, resulting in widespread transmission throughout much of Latin America and representing the first major cholera epidemic in the region in over a century. Shortly thereafter, a new serogroup, *Vibrio cholerae* O139, emerged in India and Bangladesh in 1992. Initially, this serogroup appeared capable of replacing the traditional O1 strain and was responsible for substantial outbreaks across parts of Asia. However, the O139 serogroup gradually declined in prevalence, allowing the O1 El Tor biotype to once again become the predominant circulating strain. To date, O139 has remained largely confined to Asia and has not established widespread transmission in other regions of the world.^[6,7]

Recent molecular and genomic investigations have demonstrated that circulating El Tor strains continue to evolve. Characterization of isolates recovered during the Haiti outbreak and from Bangladesh has identified hybrid variants that have acquired important genetic characteristics of the classical biotype while retaining the environmental resilience of the El Tor strain. These variant strains produce significantly greater quantities of cholera toxin than conventional El Tor isolates, potentially contributing to increased disease severity, enhanced transmissibility, and larger outbreaks. These observations highlight the dynamic evolution of *Vibrio cholerae* and emphasize the importance of continuous microbiological surveillance, genomic monitoring, and international collaboration to detect emerging strains that may alter the epidemiological profile of cholera.^[8] Despite remarkable improvements in water treatment and sanitation that have effectively eliminated endemic cholera from Europe and North America, the disease remains endemic in approximately 69 countries across Asia, Africa, and the Americas, placing an estimated 1.3

billion individuals at risk of infection.^[4] Epidemiologically, cholera has traditionally been categorized as either endemic or epidemic, although these classifications frequently overlap. Endemic cholera is defined as the occurrence of laboratory-confirmed cholera within a population during at least three of the previous five years, indicating sustained local transmission.^[9] In endemic settings, repeated exposure results in partial population immunity, causing children younger than five years of age to experience the highest disease burden because they have not yet developed sufficient natural immunity. In contrast, epidemic cholera occurs following the introduction of *Vibrio cholerae* into populations with little or no previous exposure, resulting in widespread susceptibility across all age groups and often producing larger, more severe outbreaks with higher attack rates.^[10]

Over the past decade, humanitarian emergencies have emerged as major drivers of cholera transmission. Armed conflicts, political instability, population displacement, earthquakes, flooding, drought, and famine frequently destroy essential water and sanitation infrastructure while simultaneously overwhelming healthcare systems. Since 2010, cholera has repeatedly affected vulnerable populations living under these conditions, demonstrating the close relationship between humanitarian crises and infectious disease outbreaks.^[11] The resurgence of cholera in the Middle East following regional conflicts illustrates this association, as large-scale displacement of populations and cross-border migration have increased the risk of disease spread, particularly among refugee communities with inadequate access to clean water and sanitation.^[11] Yemen has experienced the largest documented cholera outbreak in modern history, with more than 600,000 suspected cases and over 2,000 reported deaths during the initial phase of the epidemic beginning in 2017. Similar large-scale outbreaks have occurred in Somalia and the Democratic Republic of the Congo, where prolonged conflict, food insecurity, and fragile healthcare systems have facilitated sustained transmission. Haiti represents another important epidemiological example. Prior to the devastating earthquake in 2010, the country had remained free of cholera for many decades. However, following the disaster, widespread disruption of sanitation systems resulted in an epidemic that ultimately affected approximately one million individuals and caused more than 10,000 deaths, demonstrating how rapidly cholera can become established when environmental conditions favor transmission.^[12]

Bacteriology and Pathogenesis

Vibrio cholerae is a curved, comma-shaped, motile, Gram-negative bacillus belonging to the family *Vibrionaceae*. The organism possesses a single polar flagellum that provides high motility, enabling efficient movement through aquatic environments and the mucus layer of the gastrointestinal tract. *V. cholerae* is naturally found in marine, estuarine, and brackish water

ecosystems, where it survives by associating with plankton, zooplankton, shellfish, and other aquatic organisms. These environmental reservoirs play a critical role in maintaining the bacterium between epidemics and facilitate its transmission to humans through contaminated water and seafood. The organism demonstrates remarkable adaptability to changing environmental conditions, allowing it to persist in aquatic habitats for prolonged periods and contributing to the recurrent nature of cholera outbreaks, particularly in regions with inadequate sanitation and unsafe drinking water supplies.^[13,14] More than 200 serogroups of *V. cholerae* have been identified based on differences in the structure of the O antigen component of the lipopolysaccharide located on the bacterial cell wall. Despite this extensive antigenic diversity, only a limited number of serogroups possess the virulence characteristics necessary to produce epidemic cholera.

Historically, epidemic disease has been predominantly associated with serogroup O1, which was traditionally classified into two major biotypes: the classical biotype and the El Tor biotype. Both biotypes include the principal serotypes Ogawa and Inaba, while the Hikojima serotype is encountered only rarely. Identification of these epidemic strains relies on agglutination with O1-specific antisera directed against the lipopolysaccharide antigen together with confirmation of their capacity to produce cholera toxin. The genes responsible for cholera toxin synthesis are carried by a filamentous bacteriophage known as CTXΦ, which integrates into the bacterial chromosome. Although both classical and El Tor biotypes possess toxin-producing capability, minor genetic differences within the bacteriophage contribute to variations in toxin production, transmissibility, environmental survival, and epidemic potential.^[12]

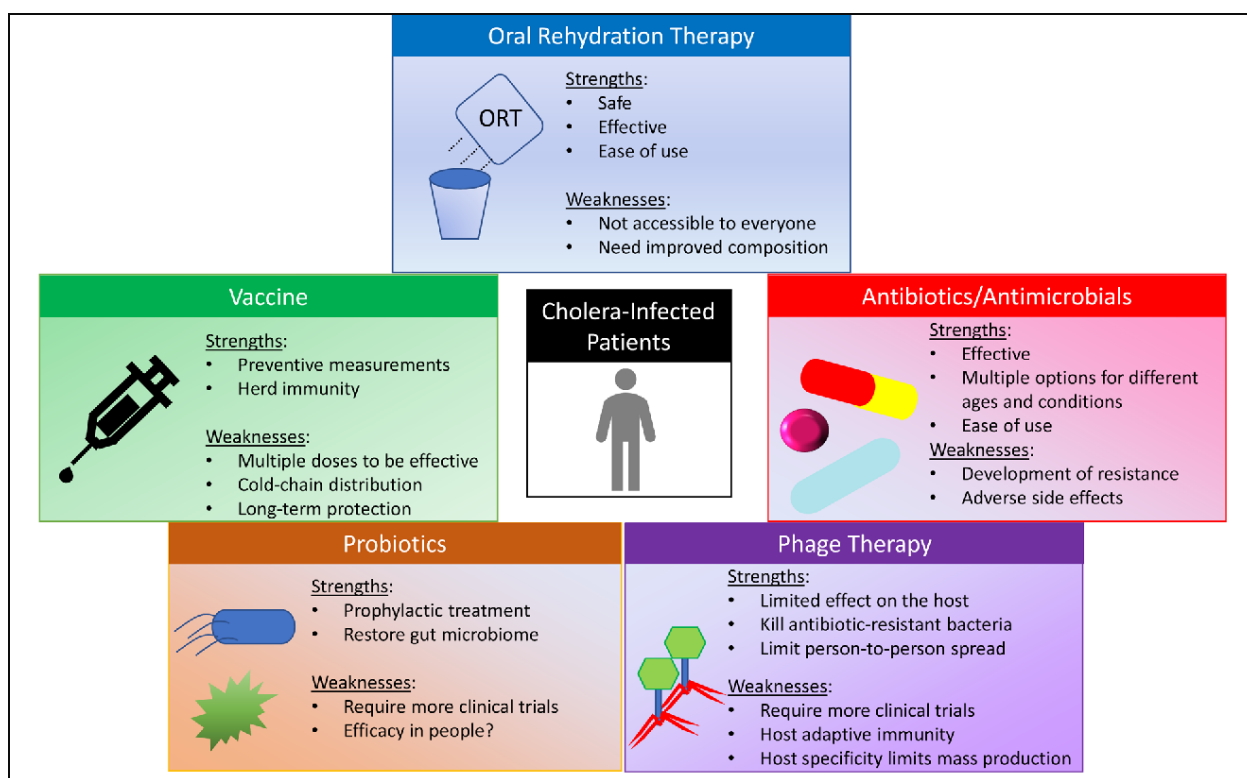


Fig. 1: Cholera Management and Prevention.

Unlike many enteric bacterial pathogens, *V. cholerae* is considered a noninvasive microorganism because it does not penetrate or destroy the intestinal epithelium. Instead, disease results primarily from the production of potent enterotoxins that disrupt normal intestinal secretory mechanisms. Following ingestion through contaminated food or water, the bacteria first encounter the acidic environment of the stomach, which serves as an important natural defense against infection. Gastric acid destroys the majority of ingested organisms, explaining why a relatively large infectious inoculum is generally required to establish disease in healthy individuals. However, factors that reduce gastric acidity, including malnutrition, achlorhydria, gastrectomy, or the use of

acid-suppressive medications, increase susceptibility to infection by allowing greater numbers of bacteria to survive gastric passage.^[15] Bacteria that survive the gastric barrier enter the small intestine, where they encounter bile salts and other antimicrobial factors. Although bile possesses bactericidal properties capable of destabilizing bacterial membranes and disrupting cellular homeostasis, virulent strains of *V. cholerae* have evolved adaptive mechanisms that enable survival within this hostile environment. Once established in the intestinal lumen, the organisms adhere to the epithelial surface of the small intestine and rapidly proliferate without invading deeper tissues. Successful colonization depends on the coordinated expression of several

virulence factors that facilitate attachment, persistence, and communication between bacterial cells.^[15] One of the most important colonization factors is the toxin-coregulated pilus (TCP), which enables bacterial adherence to intestinal epithelial cells and promotes bacterial aggregation through quorum sensing mechanisms. TCP also serves as the receptor for the CTXΦ bacteriophage, facilitating acquisition and transmission of cholera toxin genes among bacterial populations. Additional virulence determinants include hemagglutinin/protease (HapA), which assists bacterial penetration through the protective mucus layer, and the polar flagellum, which enhances motility and allows efficient migration toward the epithelial surface. These coordinated mechanisms enable the organism to establish stable colonization despite continuous intestinal peristalsis and mucus secretion.^[15,18]

The hallmark of cholera pathogenesis is the production of cholera toxin, an exceptionally potent enterotoxin responsible for the massive secretory diarrhea characteristic of the disease. After binding to GM1 ganglioside receptors located on the apical surface of intestinal epithelial cells, the toxin enters the cells and catalyzes adenosine diphosphate (ADP)-ribosylation of the stimulatory G protein associated with adenylate cyclase. This modification results in persistent activation of adenylate cyclase, producing markedly elevated intracellular cyclic adenosine monophosphate (cAMP) concentrations. Increased cAMP stimulates the cystic fibrosis transmembrane conductance regulator (CFTR) chloride channels, leading to excessive secretion of chloride ions into the intestinal lumen while simultaneously inhibiting sodium absorption. Water follows these electrolytes osmotically, producing the profuse watery diarrhea that characterizes cholera. This secretory process may result in the loss of several liters of isotonic fluid within a short period, causing severe dehydration, electrolyte disturbances, metabolic acidosis, hypovolemic shock, and, if untreated, death.^[16,17]

Clinical Manifestations

The clinical presentation of cholera is characterized primarily by acute secretory diarrhea, which represents the hallmark manifestation of infection with *Vibrio cholerae*. Disease severity varies considerably, ranging from asymptomatic infection or mild self-limiting diarrhea to fulminant illness associated with profound dehydration, electrolyte disturbances, circulatory collapse, and death. Following ingestion of contaminated food or water, the incubation period generally ranges from approximately 18 hours to 5 days, after which symptoms develop abruptly. The most characteristic clinical feature is the rapid onset of profuse watery diarrhea that progressively assumes the classic appearance of "rice-water stools," consisting of clear fluid containing flecks of mucus and possessing a distinctive fishy odor. The enormous volume of fluid lost through diarrhea may reach several liters per hour in severe cases, leading to rapid depletion of intravascular

volume and essential electrolytes if immediate fluid replacement is not provided.^[2,3,6] In addition to severe diarrhea, many patients experience recurrent vomiting, which further accelerates fluid and electrolyte losses and increases the risk of dehydration. Painful muscle cramps are also common and primarily result from substantial depletion of potassium and other electrolytes. Some individuals may develop intestinal ileus secondary to profound electrolyte imbalance and impaired gastrointestinal motility. Fever is generally absent in uncomplicated cholera because the disease produces minimal intestinal inflammation. Therefore, the presence of fever should prompt clinicians to investigate the possibility of concurrent bacterial, viral, or parasitic infections rather than attributing the symptom solely to cholera.^[6] The overall severity of cholera is determined largely by the degree of dehydration rather than the intensity of diarrhea alone. In untreated patients with severe disease, dehydration can progress rapidly, and death may occur within 6 to 12 hours following symptom onset if effective rehydration therapy is delayed or unavailable. Clinical manifestations of severe dehydration include extreme thirst, lethargy, weakness, diminished skin turgor, sunken eyes, dry mucous membranes, tachycardia with a rapid and weak radial pulse, oliguria or complete absence of urine output, hypotension, tachypnea, and progressive circulatory collapse culminating in hypovolemic shock.^[2,3] These findings require immediate recognition and aggressive fluid resuscitation to prevent irreversible organ damage.

The complications of severe cholera arise primarily from prolonged tissue hypoperfusion and profound metabolic disturbances. Reduced organ perfusion may result in acute kidney injury, particularly acute tubular necrosis, while severe hypotension can compromise cerebral circulation and increase the risk of ischemic neurological events, including stroke. Electrolyte abnormalities constitute another major source of morbidity. Hypokalemia, resulting from excessive gastrointestinal potassium losses, may cause generalized fatigue, muscle weakness, painful muscle cramps, paralytic ileus, and potentially life-threatening cardiac arrhythmias. Sodium disturbances, particularly hyponatremia secondary to disproportionate water loss, may produce neurological manifestations including confusion, irritability, weakness, and altered mental status. In addition, inadequate hepatic gluconeogenesis during severe illness may lead to hypoglycemia, particularly among children and malnourished individuals, further contributing to neurological dysfunction. When metabolic abnormalities become profound, patients may develop seizures, coma, and multiple organ dysfunction, emphasizing the importance of early diagnosis, continuous clinical assessment, prompt correction of fluid deficits, and careful monitoring of electrolyte and glucose abnormalities throughout treatment.^[2]

Diagnosis

The diagnosis of cholera is established through a combination of clinical assessment, epidemiological context, and laboratory confirmation. In regions experiencing cholera outbreaks, patients presenting with the sudden onset of profuse acute watery diarrhea and rapid dehydration are often managed presumptively while awaiting laboratory confirmation because delayed treatment may significantly increase morbidity and mortality. Nevertheless, microbiological confirmation remains essential for individual diagnosis, epidemiological surveillance, outbreak investigation, and implementation of appropriate public health interventions. Isolation of *Vibrio cholerae* from stool specimens or rectal swabs by bacterial culture remains the gold standard for confirming cholera infection. Fresh stool samples or rectal swabs are inoculated onto selective enrichment media, most commonly alkaline peptone water, which enhances the growth of *V. cholerae* while suppressing competing intestinal microorganisms. Following enrichment, specimens are cultured on selective media such as thiosulfate-citrate-bile salts-sucrose (TCBS) agar or taurocholate-tellurite-gelatin agar, which facilitate the isolation and identification of characteristic *V. cholerae* colonies. When non-selective culture media are used, definitive identification requires subsequent serological confirmation through slide agglutination testing using specific monoclonal or polyclonal antibodies directed against *Vibrio cholerae* serogroups O1 and O139, the principal serogroups responsible for epidemic cholera worldwide.^[19]

Dark-field microscopy provides a rapid but less sensitive diagnostic alternative, demonstrating approximately 50% sensitivity when compared with conventional culture methods. This technique allows direct visualization of the organism's distinctive rapid darting motility, often described as resembling "shooting stars." The characteristic movement is inhibited following the addition of *Vibrio cholerae*-specific antibodies, thereby supporting presumptive identification of the organism. Although dark-field microscopy offers the advantage of rapid bedside assessment, its relatively limited sensitivity and dependence on experienced laboratory personnel restrict its use as a definitive diagnostic method.^[2,20,21] Rapid, inexpensive, point-of-care diagnostic tests have become increasingly important, particularly in resource-limited settings and during large outbreaks where immediate clinical and public health decisions are required. Several commercially available rapid diagnostic assays, including the coagglutination test, the Sensitive Membrane Antigen Rapid Test, the Institut Pasteur rapid dipstick, the Crystal VC rapid dipstick, and Medicos rapid tests, provide timely presumptive identification of cholera and facilitate early case detection, surveillance, and outbreak control. Although these assays improve accessibility to diagnosis, positive results are generally recommended to be confirmed by bacterial culture whenever laboratory facilities are available.^[2] Molecular diagnostic techniques, particularly

polymerase chain reaction (PCR) assays, offer highly sensitive and specific detection of *Vibrio cholerae* and permit identification of virulence genes and epidemic strains. However, their routine application remains limited because they require specialized laboratory infrastructure, advanced technical expertise, and higher operational costs. Consequently, PCR testing is primarily reserved for reference laboratories, epidemiological investigations, and research settings rather than routine clinical diagnosis, especially in low-resource regions where cholera is most prevalent.^[2]

Management

The management of cholera is centered on the rapid restoration of intravascular volume, correction of electrolyte abnormalities, and prevention of complications associated with severe dehydration. Because cholera causes profuse secretory diarrhea that can result in substantial fluid and electrolyte losses within a short period, immediate assessment and initiation of appropriate therapy are essential to reduce morbidity and mortality. The primary objective of treatment is to replace ongoing fluid losses promptly while maintaining adequate tissue perfusion and organ function. Early recognition of dehydration severity enables healthcare professionals to select the most appropriate rehydration strategy and significantly improves patient outcomes.^[3,22,23] The initial clinical assessment focuses on determining the degree of dehydration through careful evaluation of the patient's general appearance, mental status, thirst, skin turgor, urine output, pulse quality, blood pressure, respiratory status, and capillary refill. Estimating the volume of fluid lost is critical for developing an individualized rehydration plan. In specialized cholera treatment centers, the use of a cholera cot, which incorporates a central opening that allows diarrheal stool to drain into a calibrated collection bucket, facilitates accurate measurement of stool volume and provides a practical method for estimating ongoing gastrointestinal losses. In healthcare settings where such equipment is unavailable, continuous fluid loss may be estimated by replacing approximately 10–20 mL/kg of fluid for every diarrheal stool or episode of vomiting, thereby ensuring that replacement therapy closely matches ongoing losses.^[3,22,23]

Rehydration therapy remains the cornerstone of cholera management and may be administered orally or intravenously depending on the severity of dehydration. Patients presenting without clinical evidence of dehydration can usually be managed successfully using oral rehydration solution (ORS) administered frequently in small volumes while continuing to replace ongoing diarrheal losses. Individuals with mild to moderate dehydration generally require more intensive oral rehydration, although intravenous fluids may be initiated when oral intake is insufficient because of persistent vomiting or clinical deterioration. Patients with severe dehydration require immediate intravenous fluid

resuscitation to rapidly restore circulating blood volume before transitioning to oral rehydration once clinical stability has been achieved.^[22] The World Health Organization recommends categorizing patients according to the degree of dehydration to guide fluid replacement. Individuals without dehydration, representing less than 5% body weight loss, should receive home-based oral rehydration therapy using ORS or appropriately prepared salted fluids together with replacement of continuing losses. Patients demonstrating signs of some dehydration, corresponding to approximately 5–10% body weight loss, require administration of 50–100 mL/kg of oral or intravenous fluids depending on clinical status, followed by replacement of ongoing fluid losses. Patients with severe dehydration, characterized by fluid deficits exceeding 10% of body weight, require urgent intravenous administration of approximately 100 mL/kg of isotonic crystalloid solution before transitioning to oral rehydration as tolerated, while continuously replacing additional gastrointestinal losses.^[22]

During the initial phase of rehydration, oral feeding is generally postponed for approximately four hours to minimize vomiting and improve fluid retention. Breastfeeding, however, should be continued throughout treatment because it provides essential nutrition, immunological protection, and hydration without increasing gastrointestinal complications. Following successful correction of dehydration, gradual reintroduction of an age-appropriate diet supports nutritional recovery, enhances intestinal repair, and prevents further deterioration of nutritional status.^[22] Although fluid replacement alone is capable of preventing most cholera-related deaths, antimicrobial therapy provides important additional clinical benefits in patients with moderate to severe disease. Appropriate antibiotic treatment shortens the duration of diarrhea, decreases stool volume, reduces the period of bacterial shedding, and limits disease transmission within the community. Selection of antimicrobial therapy should be guided by local antimicrobial susceptibility patterns because resistance profiles vary geographically and continue to evolve. Commonly recommended agents include macrolides, fluoroquinolones, and tetracycline derivatives. In many endemic regions where reduced susceptibility to fluoroquinolones has become increasingly prevalent, a single oral dose of azithromycin is considered the preferred therapeutic option because of its favorable efficacy, simplified dosing regimen, and excellent patient adherence.^[24] Despite its widespread use, increasing antimicrobial resistance presents an important challenge to cholera management. Resistance to azithromycin has become increasingly recognized since the 1990s through several mechanisms, including plasmid-mediated resistance genes and macrolide resistance proteins that reduce antibiotic effectiveness.^[24] Consequently, continuous microbiological surveillance, antimicrobial stewardship, and regular updating of regional treatment guidelines remain essential for

preserving the effectiveness of currently available antimicrobial agents and ensuring optimal clinical outcomes. Comprehensive management extends beyond acute rehydration and antimicrobial therapy to include continuous clinical monitoring throughout hospitalization. Frequent reassessment of hydration status, vital signs, urine output, stool losses, electrolyte concentrations, renal function, and acid-base balance allows healthcare professionals to adjust treatment according to the patient's evolving clinical condition. Particular attention should be directed toward identifying complications such as hypokalemia, hypoglycemia, acute kidney injury, metabolic acidosis, and circulatory shock, all of which require prompt intervention. Through timely assessment, aggressive fluid resuscitation, appropriate antimicrobial therapy, and meticulous supportive care, most patients with cholera can achieve rapid clinical recovery with minimal long-term complications.^[3,22–24]

Prevention

Prevention remains the cornerstone of global cholera control because the disease is largely avoidable through effective public health interventions that interrupt transmission and reduce population vulnerability. Historically, remarkable reductions in cholera incidence across high-income countries have been achieved through sustained investments in safe drinking water, sanitation infrastructure, wastewater management, and improved personal hygiene practices. These environmental and public health improvements have virtually eliminated endemic cholera from many industrialized nations. In contrast, many low- and middle-income countries continue to experience recurrent outbreaks because of inadequate water and sanitation systems, rapid urbanization, population displacement, poverty, and limited healthcare resources. Consequently, comprehensive prevention strategies extend beyond clinical management and require coordinated investments in public health infrastructure, community education, environmental protection, and disease surveillance.^[12] Recognizing the persistent global burden of cholera, the Global Task Force on Cholera Control (GTFCC) introduced an ambitious international strategy known as the Global Roadmap to 2030, which aims to eliminate cholera as a public health threat through coordinated international collaboration. This initiative emphasizes three interconnected strategic priorities. The first focuses on strengthening early detection and rapid response systems capable of identifying outbreaks promptly and implementing immediate containment measures before widespread transmission occurs. This objective includes the development of integrated surveillance systems, establishment of efficient laboratory networks, pre-positioning of essential medical supplies such as oral rehydration solutions (ORS) and intravenous fluids, deployment of rapid response teams, improvement of healthcare facility preparedness, implementation of oral cholera vaccination campaigns, and promotion of community participation in hygiene education and

disease prevention activities. The overarching objective is to significantly reduce mortality while minimizing the geographical spread of outbreaks through timely intervention.^[12]

The second pillar of the Global Roadmap emphasizes long-term prevention through targeted interventions in cholera hotspots, where transmission occurs repeatedly because of persistent environmental and socioeconomic risk factors. Rather than relying exclusively on emergency outbreak responses, this strategy advocates sustained investment in Water, Sanitation, and Hygiene (WASH) programs designed to improve access to safe drinking water, effective sewage disposal systems, hygienic food preparation, and proper waste management. Identification of high-risk communities enables healthcare authorities to allocate resources efficiently, implement continuous disease surveillance, strengthen community engagement, and coordinate cross-border prevention efforts in regions experiencing frequent population movement. By addressing the underlying determinants of cholera transmission, these interventions aim to prevent recurrent outbreaks and establish long-term disease control.^[12] The third strategic component focuses on strengthening technical collaboration, international partnerships, financial investment, and resource mobilization to ensure sustainable implementation of cholera prevention programs. Successful disease control requires coordinated efforts among governments, international organizations, humanitarian agencies, research institutions, healthcare professionals, and local communities. Sustained political commitment, adequate financial support, and effective governance are essential for expanding preventive programs and maintaining healthcare infrastructure. Without sufficient investment and long-term planning, many countries remain vulnerable to recurrent epidemics despite improvements in clinical treatment.^[12] Although these global initiatives provide a comprehensive framework for cholera elimination, multiple obstacles continue to impede their successful implementation. Financial limitations remain one of the most significant barriers, particularly in resource-constrained settings where investments in water infrastructure and sanitation systems compete with numerous other public health priorities. In addition, inconsistent political commitment, weak healthcare systems, armed conflicts, humanitarian crises, population displacement, natural disasters, and climate-related emergencies frequently disrupt preventive programs and facilitate disease transmission. Limited production capacity and periodic shortages of oral cholera vaccines further restrict the ability to conduct large-scale preventive vaccination campaigns in high-risk populations. Therefore, achieving the objectives outlined in the Global Roadmap requires sustained political leadership, international cooperation, and continuous financial investment to expand WASH coverage, strengthen healthcare delivery, and ensure adequate vaccine availability.^[12]

Effective cholera prevention also depends on comprehensive implementation of evidence-based public health measures. Early recognition of suspected cases through efficient surveillance systems allows rapid initiation of outbreak investigations and containment activities. Universal access to microbiologically safe drinking water remains one of the most effective preventive interventions, while adequate sanitation infrastructure prevents environmental contamination with human fecal waste. Personal hygiene practices, including regular handwashing with soap, safe food preparation, proper food storage, and protection of water supplies from contamination, significantly reduce transmission risk. Equally important is ensuring timely access to oral rehydration therapy and intravenous fluids during outbreaks to reduce mortality while limiting disease spread through prompt case management. Despite their proven effectiveness, many cholera control programs continue to prioritize emergency response activities rather than addressing the fundamental environmental and socioeconomic determinants responsible for recurrent outbreaks. The continuing lack of access to safe drinking water for approximately 844 million individuals, the consumption of fecally contaminated water by more than two billion people, and the absence of basic sanitation facilities for approximately 2.3 billion individuals illustrate the enormous global challenges that continue to sustain cholera transmission.^[7,25] Vaccination has emerged as an increasingly important component of integrated cholera prevention strategies. Protective immunity against cholera depends primarily on mucosal immune responses within the gastrointestinal tract, making oral vaccination particularly effective for stimulating local immune protection. Several oral cholera vaccines (OCVs) have been developed, including both inactivated whole-cell vaccines and live attenuated formulations. Currently available inactivated vaccines include Dukoral®, which contains inactivated *Vibrio cholerae* with cholera toxin B subunit, and Shanchol™, Euvichol®, and mORCVAX™, which contain inactivated whole-cell *V. cholerae* without the toxin B subunit. These vaccines provide protective immunity for at least three years and have received prequalification from the World Health Organization for use in endemic regions and outbreak settings.^[26] Depending on the formulation, vaccination schedules typically require two or three oral doses to achieve optimal protection.

The development of live attenuated oral vaccines has further expanded preventive options, although their effectiveness has varied across different epidemiological settings. Vaxchora™ (CVD 103-HgR), a live attenuated single-dose oral vaccine, is currently the only vaccine approved by the United States Food and Drug Administration for adults aged 18–64 years traveling to cholera-endemic regions.^[27] Since 2010, the World Health Organization has recommended the strategic use of oral cholera vaccines in endemic populations and during outbreak situations as part of comprehensive

disease control programs. To improve rapid emergency response capacity, a global oral cholera vaccine stockpile was established in 2013, enabling timely vaccine deployment during humanitarian emergencies and epidemic outbreaks. Between 2014 and 2015, the International Coordinating Group authorized distribution of hundreds of thousands of vaccine doses to countries experiencing major outbreaks, including South Sudan, Guinea, Haiti, and the Democratic Republic of the Congo.^[22] Furthermore, recent evidence suggests that when vaccine supplies are severely limited, large-scale single-dose vaccination campaigns may prevent a greater number of cholera cases and deaths than conventional two-dose schedules by allowing broader population coverage, thereby maximizing public health impact during emergency situations.^[28,29] Collectively, sustained improvements in WASH infrastructure, strengthened surveillance systems, comprehensive community engagement, international collaboration, and expanded access to effective oral cholera vaccines represent the most effective integrated strategy for achieving long-term cholera prevention and eventual disease elimination.

CONCLUSION

Cholera remains one of the most significant yet preventable infectious diseases affecting vulnerable populations worldwide. Although remarkable advances have been achieved in sanitation, surveillance, vaccination, and clinical management, recurrent outbreaks continue to occur in areas affected by poverty, conflict, displacement, and inadequate access to safe water and healthcare services. The disease continues to challenge healthcare systems because of its rapid transmission, potential for severe dehydration, and ability to cause large-scale epidemics when timely intervention is unavailable. Evidence consistently demonstrates that early recognition, prompt assessment of dehydration, rapid initiation of oral or intravenous rehydration, appropriate antimicrobial therapy, and continuous monitoring substantially reduce mortality and prevent serious complications. Equally important are comprehensive preventive measures, including strengthened surveillance systems, effective WASH programs, community health education, and expanded access to oral cholera vaccines. These interventions provide the foundation for sustainable cholera control and support the global objective of eliminating cholera as a public health threat. Nurses occupy a central position in cholera prevention and management through early clinical assessment, implementation of evidence-based treatment protocols, infection prevention and control, patient and family education, and participation in multidisciplinary outbreak response. Strengthening nursing competencies, healthcare preparedness, and international collaboration, together with sustained investment in public health infrastructure, will remain essential for reducing the global burden of cholera and improving patient outcomes in both endemic and epidemic settings.

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