Introduction
Cholera is an acute bacterial infection of the intestine caused by toxigenic *Vibrio cholerae*, characterized by severe watery diarrhoea. *Vibrio cholerae* is serologically diverse, environmental, Gram negative bacterial species. Although *Vibrio cholerae* comprises more than 200 O-antigen-based serogroups (*Shimada et al., 1994*), only the O1 and O139 serogroup strains are known to cause epidemics of cholera. Infection by *Vibrio cholerae* remains a major cause of morbidity and mortality throughout the world (*Jia-yun Wu et al., 2001*). Globally an estimated death of 120,000 due to cholera occurs each year (*Kaper et al., 1995*).

Cholera is endemic in southern Asia and parts of Africa and Latin America, where seasonal outbreaks occur widely and are particularly associated with poverty and poor sanitation. In assessing the public health significance, two critical properties of *Vibrio cholerae* are taken into account. These include the production of Cholera toxin [CT], which is responsible for the severe diarrhoea, and the possession of the O1 or O139 antigen, which acts as a marker of epidemic potential, since the actual determinant of such potential is not clearly known (*Kaper et al., 1995*).

In most cases, infection causes only mild diarrhoea or no symptoms at all. In 5–10% of cases, however, patients develop very severe watery diarrhoea and vomiting from 6 hours to 5 days after exposure to the bacterium. In its severe form cholera gravis, the clinical disease is characterized by the passage of voluminous stools of ‘rice water’ character that rapidly leads to dehydration. Hypovolemic shock, acidosis and death can occur in adults as well as in children, if prompt and appropriate treatment is not initiated (*Kaper et al., 1995*).
*Vibrio cholerae*- the causative agent of cholera is "comma" shaped, short "curved" cylindrical rod which measures about 1.5 μm x 0.2-0.4 μm in size, with rounded or slightly pointed ends. It is actively motile with a single sheathed polar flagellum. It exhibits darting motility with a speed of about 200μm/second and is asporogenous, non-capsulated and facultatively anaerobic. It is oxidase positive and fermentative in nature (*Lewis, 1998*).

CT is the major virulence factor of *Vibrio cholerae* which is encoded by the *ctxAB* operon, and is part of a larger genetic element originally termed the CTX genetic element (*Pearson et al., 1993*). CTX genetic element corresponds to the genome of CTXΦ, a lysogenic filamentous bacteriophage (*Waldor and Mekalanos, 1996*). However, molecular analysis has revealed that in addition to genes encoding CT, all strains capable of causing cholera invariably carry genes for a colonization factor known as toxin – coregulated pilus [TCP] and a regulatory protein, ToxR, which coregulates the expression of CT and TCP (*Herrington et al., 1988*). Thus cholera pathogenesis relies on the synergistic effect of a number of pathogenic factors produced by toxigenic *Vibrio cholerae* (*Faruque et al., 1998*).

**Transmission and Epidemics**

When a person is affected by cholera, large numbers of *Vibrio cholerae* are discharged in the feces of the infected individual. Inadequate sewage treatment allows water systems to become contaminated and infection is transmitted via the consumption of contaminated drinking water or food. Common sources of infection include raw or poorly cooked seafood, raw fruit and vegetables, and other foods contaminated during preparation or storage. Studies of the organism in the environment have found that the bacterium is able to survive for extended periods in aquatic and estuarine areas (*Kaysner et al., 1994*).
Cholera is considered as an ancient affliction of humans. The distinct symptomatology and the explosive onslaught of cholera epidemics are believed to be depicted in Arabian, Greek and Roman writings. The word “cholera” has been used for over 2,500 years to describe any diarrhoea and vomiting, not just that caused by bacteria (Kaysner et al., 1994).

Two distinctive epidemiologic features of cholera are its tendency to appear in explosive outbreaks often starting in several distinct foci simultaneously and its potency to cause the pandemics that progressively affect many countries in multiple continents over the course of many years (Kaper et al., 1995). Cholera epidemics have been described as either protracted or explosive. Explosive epidemics occur when the pathogenic agent is transmitted by contaminated food or water. Protracted forms occur when Vibrio cholerae is transmitted by feces – contaminated objects [fomites] (Wistreich et al., 1984).

Cholera is highly contagious, can affect all age groups, with younger people (below 2 years) more susceptible. It can destroy the human resources and affect the economy of a country through reduced production, failing food exports and decreased tourism (Kaper et al., 1995; Kaysner et al., 1994).

Clinical Manifestations (Finkelstine, 1973)

After infection, and an incubation period of 6 to 48 hours, cholera begins with the abrupt onset of watery diarrhoea. The initial stool may exceed 1 liter, and several liters of fluid may be secreted within hours, leading to hypovolemic shock. Vomiting usually accompanies the diarrhoeal episodes. Muscle cramps may occur as water and electrolytes are lost from body tissues. Loss of skin turgor, scaphoid abdomen and weak pulse are characteristic of cholera. Various degrees of fluid and electrolyte loss are observed, including mild and sub-clinical cases. The disease runs
its course in 2 to 7 days; the outcome depends upon the extent of water and electrolyte loss and the adequacy of water and electrolyte repletion therapy. Death can occur from hypovolemic shock, metabolic acidosis and uremia resulting from acute tubular necrosis.

**Prevention and Treatment**

Cholera is a preventable disease. People living in high-risk areas can protect themselves by following a few simple rules of good hygiene and safe food preparation. These include scrupulous washing of hands, especially before food preparation and eating, thorough cooking of food and consumption while hot, boiling or treatment of drinking water and use of sanitary facilities. By taking a few basic precautions, travellers can likewise protect themselves against cholera. Above all, travellers should be very careful with food and water, including ice.

The most important treatment for cholera is rehydration, which consists of prompt replacement of water and salts lost by severe diarrhoea and vomiting. Early rehydration can save the lives of nearly all cholera patients. Most can be rehydrated quickly and easily by drinking large quantities of a solution of oral rehydration salts (Kaper et al., 1995). Tetracycline, chloramphenicol and co-trimoxazole reduce the period of excretion of *Vibrio cholerae* in the stools of cholera patient. Tetracycline is often given to reduce environmental contamination and to reduce the risk of cross-infection (Lewis, 1998). Apart from good hygienic practices, safe food and drinking water, one of the most effective preventive measures against cholera is vaccination. Oral cholera vaccines which provide good protection for up to 3 years are now available. Vaccination is also required to protect travellers visiting the endemic areas (Kaper et al., 1995).
Epidemiology

The molecular epidemiological surveillance of cholera in areas of endemic infection has also revealed changes in the properties of toxigenic Vibrio cholerae and emergence of new epidemic clones which often replace existing clones (Faruque et al., 1997). Moreover the propagation of phage CTXΦ may be associated with the origination of novel toxigenic Vibrio cholerae from non-toxigenic progenitors. Epidemiological studies by molecular techniques revealed the emergence of new clones of toxigenic Vibrio cholerae, possibly through natural selection involving unidentified environmental factors and immunity of the host population (Faruque et al., 1998a).