



Original Article

An Ogawa Cholera Outbreak 6 Months After the Inaba Cholera Outbreaks in India, 2006

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BACKGROUND/PURPOSE: Cholera has been reported in the state of Orissa, India during the last decades. An explosive outbreak of diarrhea occurred in Central Cuttack Ward 22 of Orissa (population approximately 10,621), between March 12–23, 2006. This outbreak was investigated by a team from the Regional Medical Research Centre of Bhubaneswar to identify the causative agents and to determine the antimicrobial susceptibility pattern and associated virulent genes.

METHODS: Clinical and epidemiological data were collected from 100 hospitalized patients with diarrhea from the Sriram Chandra Bhanja Medical College, Cuttack, Orissa. Rectal swabs and water samples were collected and tested for diarrheagenic enteropathogens. Isolated *Vibrio cholerae* were subjected to antibiotic susceptibility tests and polymerase chain reaction analysis for the detection of virulent genes.

RESULTS: Of the 23 rectal swabs collected, 19 (82.6%) were positive for *V. cholerae* serogroup O1, serotype Ogawa. All strains were uniformly susceptible to ampicillin, gentamicin, chloramphenicol, ciprofloxacin, norfloxacin, neomycin, and tetracycline, but resistant to co-trimoxazole, furazolidone, nalidixic acid, and streptomycin. Polymerase chain reaction revealed that all strains had *ctxA*, *tcpA* (biotype El Tor), *zot*, and *ace* genes, suggesting their possible role in the outbreak.

CONCLUSION: This is the first localized outbreak of *V. cholerae* O1, serotype Ogawa, in the state of Orissa in 2006 after a gap of 6 months dominated by Inaba strains.

KEYWORDS: cholera, outbreak, Inaba, Ogawa, *Vibrio cholerae*

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Introduction

Epidemic cholera caused by toxigenic *Vibrio cholerae* serogroups O1 or O139 has been a major threat and constitutes a major public health problem in India. The emergence and reemergence of *V. cholerae* O139 has also been reported in various parts of India.^{1–5} The lack of clean water, overcrowding, insufficient understanding of personal and domestic hygiene, nutritional deficiency and overall poor

sanitation are the major contributing factors for the spread of cholera in India. Orissa, an Eastern state of India, has been recognized as an endemic zone for cholera due to *V. cholerae* O1 serogroup Ogawa, biotype El Tor, and serogroup O139.⁶⁻⁹ During a 6-month period in 2005, an outbreak of cholera, dominated by *V. cholerae* O1 Inaba, was reported in Orissa.¹⁰ Here, we report another outbreak of cholera in Cuttack, Orissa, between March 12 to March 23, 2006, and the antibiotic susceptibility and virulent genes of the isolated *V. cholerae* strains.

Methods

Cuttack is the former capital of Orissa, situated on the apex of delta formed by the rivers Mahanadi in the North and Kathajori in the South (Figure 1). It has a land area of approximately 60 km² and a population of 641,130 (2001 census). The Municipal Corporation area is demarcated into 39 administrative wards. It is an old city with old streets where rapid urbanization occurs with overcrowding due to the influx of migrating populations for commercial purposes.

A local outbreak of diarrhea was reported on March 12, 2006 at 12 pm, following a marriage ceremony held on

March 10, 2006 and continued up to March 23, 2006 in two lanes of Ward 22. Anticipating the extensive and explosive nature of the outbreak of diarrhea in the town, local health authorities, doctors, and paramedical workers visited patients with clinical symptoms. Diarrhea patients with severe dehydration were admitted to the diarrhea ward in Srirama Chandra Bhanja (SCB) Medical College, Cuttack. On the recommendation of the health authorities, all suspected water sources were disinfected.

An investigating team from the Microbiology Division of Regional Medical Research Centre (RMRC) collected clinical and epidemiological data from the hospitalized diarrhea cases. Patients were interviewed following World Health Organization (WHO) guidelines and appropriate management was advised. No fatal cases were reported. Twenty-three non-random rectal swabs were collected in Cary-Blair transport medium (Becton Dickinson, USA) from untreated hospitalized patients. Six water samples were collected in 1 L sterile bottles from different water sources (2 tube wells, 2 piped water, and 2 samples of stored drinking water collected from pipe water). Water was suspected as both the vehicle of transmission and mode of spread of this localized outbreak.

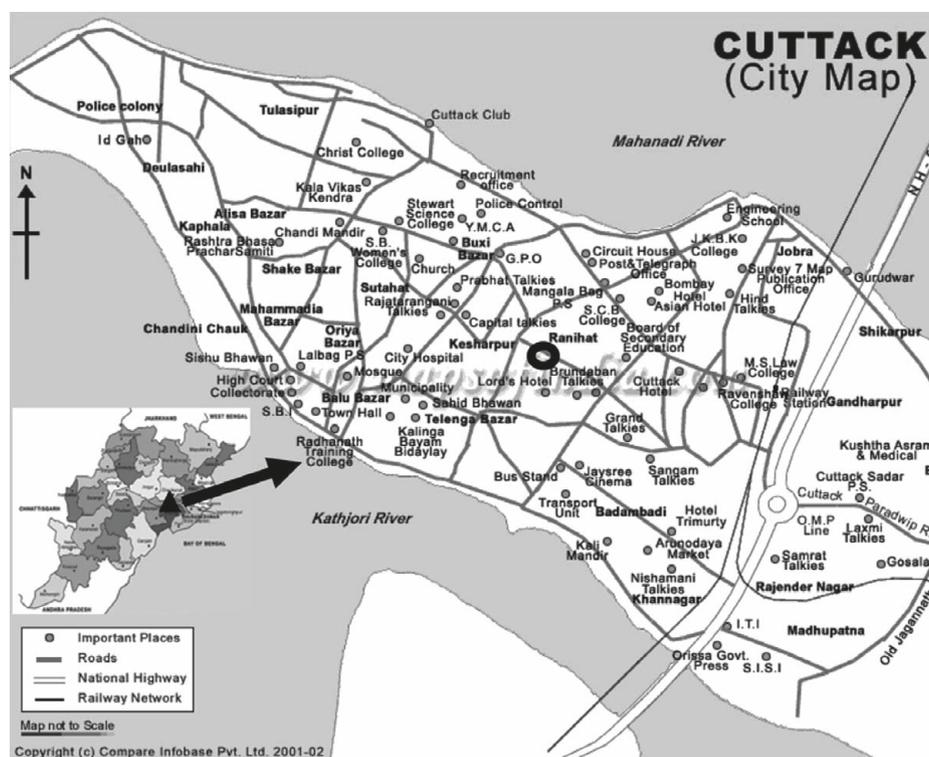


Figure 1. Location map of the city of Cuttack showing cholera outbreak area in Ward 22 (dark circle), situated centrally in the city.

All rectal swabs were processed at the microbiology laboratory, Regional Medical Research Centre for detection of diarrheagenic pathogens using standard techniques.¹¹ Water samples were analyzed to find out the source of contamination following previously described methods.¹² Antibiotic susceptibility was tested by the modified Kirby-Bauer disk diffusion technique¹³ using commercially available discs (Himedia, Mumbai, India) including ampicillin (10 µg), chloramphenicol (30 µg), co-trimoxazole (25 µg), ciprofloxacin (5 µg), furazolidone (100 µg), gentamicin (10 µg), neomycin (30 µg), nalidixic acid (30 µg), norfloxacin (10 µg), streptomycin (10 µg), and tetracycline (30 µg). Characterization of strains as being susceptible or resistant was based on the size of the inhibition zone around each disc, according to manufacturer's instructions that matched the interpretive criteria recommended by WHO.¹⁴ Strains with an intermediate inhibition zone were interpreted as being resistant on the basis of minimum inhibition concentration studies conducted with *V. cholerae*.¹⁵ A polymerase chain reaction-based assay was performed as described elsewhere for the detection of several virulent genes *ctxA*, *tcpA*, *ace* and *zot*.¹⁶

Results and Discussion

One diarrhea case was reported at 12 pm on March 12, 2006, 24–48 hours after the marriage ceremony, and within few hours at least 10 people were admitted to hospital with severe to moderate dehydration. The total number of affected people in the study area was about 10,621, from 1,327 families. A total of 146 diarrhea cases representing all age groups were reported from the affected Ward 22. Most cases presented with profuse watery diarrhea and vomiting, and 100 cases were admitted to the diarrhea ward of SCB Medical College. The index case was an 18-year-old man with severe dehydration admitted to the hospital on March 12, 2006. He had attended the marriage ceremony and did not visit any cholera outbreak area before the disease appeared. The outbreak peaked during the night of March 12, 2006 (Figure 2). By that time, awareness about early hospitalization had spread, and patients were reporting early. No new cases were reported after March 23, 2006. Of 146 cases, 110 (75.3%) were adults and 36 (24.7%) were children.

Microbiological analysis of 23 rectal swabs collected during the investigation revealed that 19 (82.6%) was positive

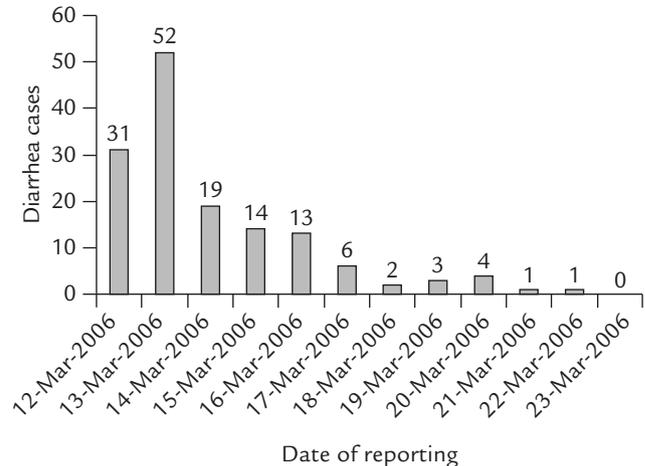


Figure 2. Date-wise distribution of hospitalized patients with acute secretory diarrhea at Srirama Chandra Bhanja Medical college Cuttack, India from March 12–23, 2006.

for *V. cholerae* O1 Ogawa, El Tor biotype. No other enteropathogen was isolated. Of six water samples collected during the investigation, *V. cholerae* O1 Ogawa was found in one sample of stored water. The strains were uniformly susceptible to ampicillin, gentamicin, chloramphenicol, ciprofloxacin, norfloxacin, neomycin, and tetracycline; and uniformly resistant to co-trimoxazole, furazolidone, nalidixic acid and streptomycin. Polymerase chain reaction revealed that both clinical and environmental *V. cholerae* strains carried *ctxA*, *tcpA* (El Tor biotype), *ace* and *zot* genes.

In Cuttack, the network of pipelines was built about 30 years ago, and had not yet been replaced at the time of the outbreak. The city drainage system is open, passes through every ward and connects to the main drain which ultimately joins to the river encircling the city. People of the slum without latrines defecated in the open drain. The water pipes either pass through or adjacent to the drain at many places. Before Day 1 of the outbreak, there were two leakages in the pipeline passing the side of the drain near to the outbreak house. Probably due to the leakage, the water inside the pipe could have been contaminated, and was then used at the marriage ceremony. Immediate steps were taken by the health authority to repair the leakage in the pipeline. Extensive chlorination of all water sources resulted in a sharp decline in diarrhea cases, and no new case has occurred since March 23, 2006. Early rehydration at the hospital and use of intravenous fluids avoided any fatalities.

Contamination of water by *V. cholerae* by any means is one of the major contributing factors to cholera outbreaks.

There was lack of proper maintenance of the pipelines, which might cause leakages. This may very likely be the major contributing factor to the outbreak.

In the state of Orissa, reported cholera outbreaks were due to the dominant serogroup O1 of *V. cholera*, Ogawa biotype El Tor coexisting with serogroup O139.^{5,6} However, in 2005, cholera outbreaks in the state were dominated by *V. cholerae* O1, Inaba.¹⁰ *V. cholerae* O1 strains are known to inter-convert between Ogawa and Inaba forms due to host immune pressure in the population.¹⁷⁻²⁰ This was evidenced in Latin America between 1991-1992 in Kolkata, where Ogawa isolates began to appear 7-12 months after an epidemic caused by the Inaba strain, and in South India in 2000, where both Ogawa and Inaba were found to have homology at the molecular level, predicting interconversion.²⁰⁻²² These observations advocate that the appearance of *V. cholerae* O1 Ogawa as a dominant serotype in the present outbreak may be due to the interconversion of Inaba to Ogawa within a couple of months, or to a newly introduced strain of *V. cholerae*, Ogawa biotype El Tor. The predominance of adults (75%) affected by Ogawa serotype shows a lack of exposure as evidenced earlier, and serotype conversion predicts alterations at the genetic level.²³ However, detailed genetic studies are required to confirm this.

There is a need for continued vigilance and effective strategies to provide safe drinking water and strengthen disease surveillance to counter the outbreaks of cholera, especially in vulnerable areas such as urban slums and overcrowded populations.

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