Letter to Editor

Cholera: Small Outbreak in Winter Season of Eastern Nepal

Dear Editor,

The global resurgence of cholera is becoming an increasingly important public-health challenge as the number of countries affected by this infection continues to increase.[1] The current cause of the global cholera pandemic, *Vibrio cholerae* O1 El Tor (including hybrid strains), has replaced classical biotype organisms around the globe, is associated with prolonged cholera outbreaks, is able to persist in aquatic reservoirs and cause endemic disease, is increasingly resistant to antimicrobials, and is associated with high case-fatality rates.[2] The first report of cholera was officially published in the years 1958-1960 in Kathmandu by a medical doctor visiting Nepal.[3] Other reports of cholera outbreaks in Nepal have been reported in literatures.[3-5] In year 2007, more than 50,000 people of the 54 Village Development Committee (VDC) s in the Saptari district of Eastern Nepal had been in effect of the diarrhea with death toll to 25.[6] After 4 years (November, 2011) in the same district, two people from *Tilathi* village lost their lives due to diarrheal illness. Upon the request of Ministry of Health, Nepal, a rapid response team comprising of microbiologists and epidemiologists from B.P Koirala Institute of Health Sciences was sent to elucidate the cause of the recent small outbreak of diarrheal illness.

After a focus group discussion with the locals of affected village in presence of officials of District Public Health Office, Saptari, clinical and water samples were collected systematically from three wards with active cases of diarrheal illness. Five clinical samples from symptomatic individuals without any antibiotic administration and four water samples (pond and underground) were collected, transported, and investigated microbiologically following Centers for Disease Control Guidelines.[7,8] The causative agent of diarrheal illness was found to be *V. cholerae*, [O1: El Tor] Ogawa serotype, in three clinical and water samples each. Phenotypically, the isolates from the water samples (pond) were identical to the samples isolated from the patients. This finding suggests that the clinical isolates probably disseminated from pond water. The absence of organism in underground water keeps it safe from pathogenic organisms. In contrast to cotrimoxazole, nalidixic acid, and furazolidone; chloramphenicol and ceftriaxone are sensitive in all the isolates as depicted in Table 1. Ofloxacin, ampicillin, tetracycline, and ciprofloxacin are in decreasing order of susceptibilities. Similarity in the antibiogram among isolates A from clinical sample and E from water sample suggests the presence of same strain in both the samples, reflecting that the same strain of organism can persist in both water and humans. Dissimilar antibiogram among isolates from clinical sample suggests that the different circulating strains were responsible in causing this small outbreak. Dissimilar antibiotic sensitivity pattern among clinical isolates and environmental isolates may have been influenced by host factor, microorganism factor, and environmental factor. Extensive characterization of these strains would definitely bring some conclusion on factors responsible for genotypic variation of isolates within the same epidemic and its probable role in bacterial virulence.

The villagers had recently celebrated one of their important festivals, *chhath*; the rituals of which are performed in ponds. Open defecation habit and use of pond water as a major source of water for drinking, cooking, and bathing had a foremost role in the cholera outbreak. The people seemed to be aware of the spread of diarrheal illnesses but were not motivated to use their toilets indeed. Numerous studies have shown that *V. cholerae* belong to a group of organisms whose major habitats are aquatic ecosystems, and the role of water sources in the spread of cholera has been well documented with a suggestion that environmental concentration of *Vibrio* species might increase in response to zooplankton or phytoplankton blooms driven by global or local aquatic conditions.[9] Three-dimensional biofilm on surfaces of *V. cholerae* O1 El Tor and O139 facilitates its persistence within natural aquatic habitats during interepidemic periods,[10] which might be the reason in current case too.

Table 1: Antimicrobial susceptibility pattern of *Vibrio cholera* isolates

Antibiotics	Clinical samples			Water samples		
	A	B	C	D	E	F
Ampicillin (10 μg)	R	S	S	S	R	S
Chloramphenicol (30 μg)	S	S	S	S	S	S
Tetracycline (30 μg)	R	R	S	S	R	S
Ciprofloxacin (5 μg)	R	R	S	R	R	R
Ofloxacin (5 μg)	S	S	S	S	S	R
Cotrimoxazole (25 μg)	R	R	R	R	R	R
Ceftriaxone (30 μg)	S	S	S	S	S	S
Nalidixic acid (30 μg)	R	R	R	R	R	R
Furazolidone (100 μg)	R	R	R	R	R	R

S: Sensitive, R: Resistant

This report of a small cholera outbreak occurred in non-summer season. From an applied perspective, clarifying the mechanisms that link seasonal environmental changes to diseases' dynamics will aid in developing strategies for controlling diseases and improving disease forecast across a range of human and natural systems. We would like to recommend that exploration of presence of these organisms along the year and their molecular epidemiological typing would definitely benefit in tracing the method of spread of cholera in the area. This would also review the role of environmental factors responsible for cholera spread. We would like to acknowledge the support of Prof. SK Bhattacharya, Dr. Rajendra Gurung, Dr. Nimesh Poudyal, Dr. Ratna Baral, Dr. Narayan Raj Bhattarai, and Mr. Tejndra Pandit for their immense support and encouragement. We are greatful to officers of District Public Health Office, Rajbiraj, Nepal.

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