Research Recherche

Bulletin of the World Health Organization, 59 (1): 61-66 (1981)

Cholera in Bahrain: epidemiological characteristics of an outbreak*

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In the period 10 August 1978–23 January 1979, 913 culture-confirmed cases of cholera caused by Vibrio cholerae, *biotype El Tor, serotype Ogawa, occurred in Bahrain. After discovery of the initial cases, others occurred sporadically, and the incidence reached a peak of 25–35 cases per day during the seventh week of the outbreak (16–22 September). The overall attack rate (27 per 10 000) was low and the outbreak subsided without mass immunization campaigns or rigorous border control of persons and imports. Investigation of 746 culture-confirmed cases that occurred in the period 10 August–13 October 1978, showed that cases occurred throughout most areas of the country and mainly affected infants, young children, and adult working-age males. Symptoms were very mild; fewer than 20% of patients required specific rehydration therapy. The highest attack rate (84 per 10 000) occurred in infants less than 1 year of age. No common vehicle or mode of transmission was identified. A matched-pair study of 35 cases and controls showed that adult cases were more likely than controls to have consumed food or beverage outside of the home before becoming ill.* V. cholerae *was isolated from stored drinking water in the houses of 8 cases but not from numerous samples of food and tap-water. It was presumed that cholera transmission occurred through a complex interaction of mild and asymptomatically infected persons with food, water, and the environment.*

The El Tor biotype of *Vibrio cholerae* O–Group 1 (O1) was first recognized in South-West Asia in 1965, when Bahrain reported one case. Since that time El Tor cholera has continued to spread throughout South-West Asia and westward into Africa and southern Europe (*1*). In 1972 and 1973, Bahrain experienced limited outbreaks caused by *V. cholerae* El Tor serotype Inaba, with 74 cases in 1972 and 37 cases in 1973. Routine cultures of stool, food, and water since 1973 have been negative for *V. cholerae* O1.

In this report we describe an investigation of an outbreak of 913 culture-confirmed cases of *V. cholerae*, biotype El Tor, serotype Ogawa, that occurred in Bahrain in the period 10 August 1978–23 January 1979. The epidemiological characteristics of the outbreak and the results of a case-control study designed to determine the vehicle and mode of transmission are reported.

OUTBREAK

Bahrain is a small island state situated in the Arabian Gulf with an area of 670 km² and a population of 341 000 (1978 estimate). Municipal drinking water is provided by approximately 60 deep wells and 10 desalinization plants. Desalinated water is preferred by most people for drinking and tea making.

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Well water is not chlorinated, whereas desalinated water obtained from the larger plants is reportedly chlorinated to not less than 0.2 mg/litre. Samples of water from deep wells and desalinated water are routinely monitored for coliforms and since 1973 also for *V. cholerae* O1. Chlorine residuals are monitored at the distribution point of desalination plants. Most households have piped-in well water, electricity, and refrigeration. Drinking water is often stored in 20-litre screw-top plastic containers.

On 10 August 1978, *V. cholerae*, biotype El Tor, serotype Ogawa, was isolated from a rice-water-stool specimen from a man residing in Manama City, the capital of Bahrain. The following day, 2 additional cases were reported among his immediate contacts. All 3 patients had recently had close contact with visitors from a country that in the subsequent week reported cholera cases. Despite quarantine of the patients and their contacts, other cases occurred sporadically in most regions of the country, sometimes in people who had had no known contact with cholera patients. The number of cases increased markedly in the week following the Eid religious holiday (3–5 September) and then gradually increased to a peak of 25–35 per day in the seventh week of the outbreak (16–22 September). There was no evidence that the patients had been exposed to a common vehicle. Almost all cases occurred in the poorer people, with a disproportionately large number (10.9%) in infants less than 1 year of age.

The signs and symptoms were, in general, very mild and severe dehydration was rare; only 2 deaths occurred (a 6-week-old infant and an 83-year-old man). Control measures focused on improving community and restaurant sanitation and on active surveillance of new cases. Case households were visited by a health inspector within 24 hours of bacteriological confirmation of the first case; a prophylactic antibiotic (doxycycline—approximately 6 mg/kg body weight daily for 3 days) was administered to all household contacts. Throughout the country, efforts to monitor water supplies and health education activities were intensified. A single cholera immunization was provided only to those who requested it and no significant changes in immigration and import regulations were instituted.

Because cases were continuing despite initial control measures and the vehicle(s) and mode of transmission were unknown, the investigation was expanded and a case-control study was carried out.

MATERIALS AND METHODS

Case data were obtained from a log book on all symptomatic, culture-positive persons treated at a special, centrally located diarrhoea/cholera clinic during the period 10 August–13 October 1978.

A case-control study was conducted in a recently constructed (inhabited in 1968) urban community of 11 000 people situated 8 km south of the capital city. Houses were of concrete block construction, had piped water, and were connected to a municipal sewerage system. They were inhabited by people of all socioeconomic levels, except for the very poor and very rich. The cases (n = 35) included in the study were all identified culture-confirmed symptomatic patients living in the community and the surrounding villages whose illness began between the beginning of the outbreak and 25 September; however, most of the cases occurred in the 2 weeks before 26 September, when the study began. For each case, a control subject matched by age and sex was systematically selected from the neighbourhood. The control subjects were selected from households in which no cases of cholera or afebrile diarrhoea had occurred in the preceding 2 months.

Laboratory investigations included culturing faecal swabs, food, and water specimens (450 ml). Each specimen was enriched in alkaline peptone water for 6–8 hours, after which the enrichment culture was streaked on thiosulfate-citrate-bile salts-sucrose (TCBS) agar. After overnight incubation, suspicious colonies were agglutinated with polyvalent and type-specific antisera. *V. cholera* O1 isolates from water and randomly selected isolates from stools were biochemically identified. The 27 isolates of *V. cholerae* O1 obtained both during the early and later stages of the outbreak, including rough strains isolated in the last week of September, were phage typed (*2*).

RESULTS

All cases

In the period 10 August–13 October 1978, 746 culture-confirmed cases of *V. cholerae*, biotype El Tor, serotype Ogawa, were identified (Fig. 1). The distribution of infant cases was similar to that of other cases. Incidence was highest (84 per 10 000) among infants less than 1 year of age (Table 1), and Fig. 2 shows that males 20–39 years old also had a relatively high attack rate.

In the case-control study there were 17 cases in males and 18 in females; 15 (43%) were more than 12 years old, 17 (49%) were between 1 and 11 years of age, and 3 (8%) were less than 1 year old. Cases were distributed throughout the community without evidence of clustering. Of the 35 cases, 23 patients (66%) were admitted to hospital primarily for isolation from the community rather than for treatment; only 6 (17%) required rehydration therapy.

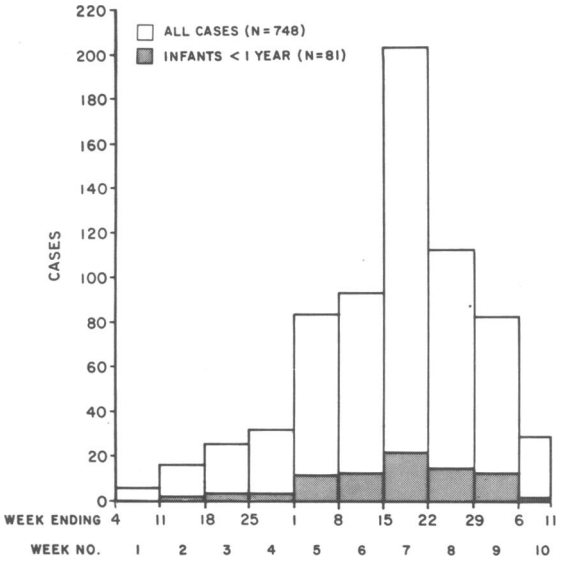

Fig. 1. Number of bacteriologically confirmed, clinical cases of cholera, by week of onset, Bahrain, 5 August–10 October 1978 (week no. 10 = 4 days only).

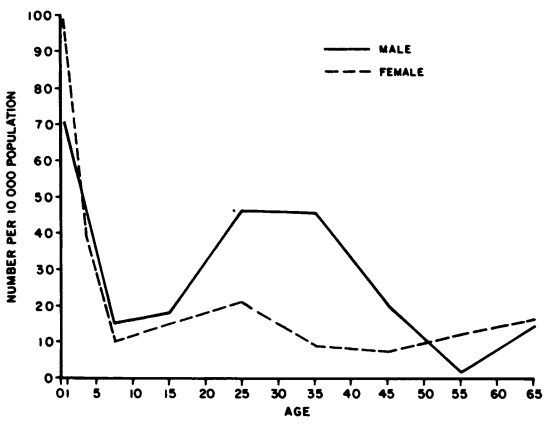

Fig. 2. Number of bacteriologically confirmed, clinical cases of cholera per 10 000 population, by age (years) and sex, 5 August–6 October 1978 (based on 1976 population estimate).

Table 1. Cholera cases,[a] age-specific attack rates, Bahrain, 10 August–6 October 1978

Age group	Population[b]	Attack rate[c]	
0–2 months	2 421	45.4	84.4
3–5 months	2 383	50.4	
6–8 months	2 357	132.1	
9–11 months	2 321	116.8	
1 year	8 893	78.7	42.8
2 years	8 359	50.2	
3 years	7 858	25.5	
4 years	7 386	20.3	
5–9 years	41 530	12.8	
10–19 years	72 970	16.7	
20–29 years	50 750	36.7	
30–39 years	28 190	25.9	
40–49 years	21 240	14.6	
50–59 years	13 550	6.6	
⩾60 years	10 070	14.9	
	280 278	25.3	

[a] Bacteriologically confirmed clinical illness.
[b] 1976 estimated population.
[c] Per 10 000 population.

The case-control study did not identify significant differences between cases and controls as regards most of the variables that were examined: persons per household (cases 9.8/controls 8.6), number of persons per household less than 15 years of age (cases 4.8/controls 4.0), number of persons per bedroom (cases 3.5/controls 3.3), standard of education of case or head of household, occupation of head of household, source of drinking water, intrahousehold handling and storage of drinking water, individual consumption of water per day, use of ice, ingestion of raw foods, subjective sanitary rating, or close contact with visitors from other cholera infected countries. Although we did not identify a common restaurant or vehicle of transmission, the study did show that adult cases (12 years or older, $n = 15$) were significantly more likely than controls to have eaten or drunk in "restaurants" (8 cases:0 controls) during the 5 days before they became ill ($P < 0.02$, McNemar's matched-pair test).

Infant cases

Eighty-one cases of cholera in infants less than 1 year of age were identified during the investigation period. Of these, 34 (42%) were male and 47 (58%) were female; 11 (13.6%) of the infants were less than 3 months old, 12 (14.8%) were 3–5 months old and 58 (71.6%) were 6–11 months old. The attack rates by age group, calculated from crude population estimates derived from a geometric progression of annual survival rates, show a markedly elevated rate in the 6–11-month age group (Table 1). Infant cases were not clustered by area or place of residence.

From medical records it was determined that of 43 hospitalized infants, 23 (53%) received intravenous rehydration, although only 19 were reported to be dehydrated. Dehydration was reported as moderate to severe in 11 cases (26%). Serum bicarbonate levels were studied in 15 infants and 6 (40%) were less than 20 mmol/litre; however, the lowest being 17 mmol/litre. Body temperature was recorded as >38.3 °C in 14 infants (33%) but this was not related to the presence of dehydration. The significance of the observed temperature elevation was often not commented upon in the medical notes. One 6-week-old infant died after approximately 11 hours of hospitalization. The mean hospital stay was 4.4 days (range 2–10 days).

Laboratory

During a one-month period (9 September–8 October) 1629 stool specimens from food handlers were submitted to the Public Health Department of Bahrain for routine examination and during the outbreak approximately 4000 additional specimens were submitted. Of the food handlers tested, 25 were infected with *V. cholerae* O1 (infection rate approximately 0.4%). Of these, 7 had symptomatic cholera.

During August and September, 236 food and beverage samples were cultured and all were negative for *V. cholerae* O1. In the same period, more than 700 routine water samples obtained from bore-hole wells or their nearest distribution tap and from various desalination plants were also negative for *V. cholerae* O1. No samples showed grossly elevated coliform counts; in a small percentage of samples, low total coliform counts (less than 20 per 100 ml) were noted, but in these samples the counts soon returned to normal. In contrast, *V. cholerae* O1 was isolated from stored drinking water (either tap or distilled water) from 20-litre storage containers, thermos jugs, or refrigerated drinking pitchers in the houses of 8 cases. In 4 out of the 5 specimens examined for total coliforms, the counts were greater than 180 per 100 ml (most probable number). Samples obtained from tap-water in the same house and in some instances from neighbouring houses were all negative for coliforms and *V. cholerae* O1.

Of 27 confirmed isolates of *V. cholerae* O1 that were phage typed, all but 2 were El Tor, phage type IV, defined as sensitive to El Tor phages 1, 2, and 5.

DISCUSSION

The 1978 El Tor cholera outbreak in Bahrain, although considerably larger than outbreaks in 1972 and 1973, still affected a very small percentage (0.3%) of the inhabitants. Cases occurred sporadically throughout the country and clusters around common sources were not identified. The outbreak gradually reached a peak during the seventh week, quickly declined to only a few cases a day, and finally terminated on 23 January 1979. Although cholera had spread throughout all districts of Bahrain, the relatively small number of cases identified shows that a country such as Bahrain that has protected municipal water supplies, a moderate level of sanitation, and an active public health department can contain cholera and can do so without employing stringent border restrictions, as regards people and goods, and/or mass immunization campaigns.

One of the striking characteristics of the outbreak was the mild symptoms manifested by the majority of patients. Only 2 deaths occurred (case: fatality ratio 0.2%) and less than 20% of the cases required rehydration fluids. An outbreak of *V. cholerae*, El Tor, Ogawa, that occurred in the eastern provinces of Saudi Arabia in 1978, also demonstrated a similar pattern of very mild disease with low morbidity and mortality (Y. Watanbe, personal communication, 1978). Although the ratio of asymptomatic and mild cases to severe cases has been shown to be much greater with El Tor cholera than with classical cholera (*3*, *4*), the former can also cause dehydration and death; for example, recently in Africa and the Maldive Islands severe cases and deaths occurred (*5*, and CDC, unpublished data, 1978). The reason for the mild disease pattern observed in Bahrain and Saudi Arabia is unknown.

In this investigation, incidence was calculated on the basis of cases identified among persons who had attended a diarrhoea/cholera clinic rather than a random population survey which might have been more accurate. Even though the clinic was centrally located and transportation facilities were adequate, certain population groups may have been more likely to seek diagnosis and treatment than others. This may have accounted for the high incidence recorded in children 0-4 years of age. Other data from this study of infant cholera cases showed that bottle feeding was a significant risk factor for infants acquiring symptomatic cholera (*6*), and this is borne out by the very high attack rates in the 6–11-month age group, the age at which infants are weaned in this population. Numerous other studies have shown that diarrhoeal disease incidence in children is highest during and shortly after the weaning period (*7*).

The high attack rate in males in the working-age group in Bahrain suggests that their mobility outside the home may have increased their exposure to contaminated food or water.

Investigation of this outbreak failed to identify a common vehicle or mode of transmission. Although

in past outbreaks, case-control studies were successful in epidemiologically implicating a vehicle of infection when likely vehicles were initially suspected (*8–10*), when this is not the case they are probably of limited value. In this outbreak, the case-control study suggested that adults may have acquired their infection from food or beverages consumed outside the home. Transmission among other family members may have occurred from contamination of food and water within the household. The occurrence of intra-household transmission is supported by the isolation of *V. cholerae* O1 from stored drinking water within 8 affected households, while concomitant examination of public piped-in water was negative for coliforms and *V. cholerae* O1.

Studies in Bangladesh showed that safe public water supplies (tube wells) did not prevent the spread of El Tor cholera because the population had contact with alternative unsafe water sources that were frequently used for food preparation, bathing, and dishwashing (*11*, *12*). Since infection with El Tor cholera, in contrast to classical cholera, results in a longer period of *Vibrio* shedding and a higher incidence of asymptomatic infection, and because the El Tor organism survives longer in the extra-intestinal environment, El Tor cholera may in some instances be transmitted through a complex interaction of contaminated food, water, and environment rather than through public drinking water supplies (*12*). Transmission of cholera in Bahrain probably occurred in this manner. Continued detailed microbiological and epidemiological investigations are needed to further delineate the precise mode of El Tor cholera transmission.

ACKNOWLEDGEMENTS

We gratefully acknowledge the support of Dr F. Partow and Dr V. Parisi of the WHO Regional Office for the Eastern Mediterranean , Alexandria, Egypt, under whose auspices this investigation was conducted. We also wish to thank Dr Ali Fakhro, Minister of Health; Dr Akbar Mohsin, Chairman, Department of Pediatrics; Dr Ahmad A. Ahmad, Chief of Staff, Salmaniya Hospital; and Dr Sameer Khalfan, Ebrahim Yousef, Iftikhar Nasimi, Khairia Asiri, Lila Philipose, and other members of the Public Health Directorate and Ministry of Health in Bahrain, who assisted in this investigation. Dr Charlotte Parker, Department of Microbiology, University of Texas, Austin, Texas, USA, performed the cholera phage typing.

RÉSUMÉ

LE CHOLÉRA À BAHREÏN: CARACTÈRES ÉPIDÉMIOLOGIQUES D'UNE ÉPIDÉMIE

Entre le 10 août 1978 et le 23 janvier 1979, on a observé à Bahreïn 913 cas de choléra, confirmés par culture, dus à *Vibrio cholerae*, biotype El Tor, sérotype Ogawa. Après la découverte des cas initiaux, d'autres se sont présentés de manière sporadique, atteignant un pic de 25–35 cas par jour durant la septième semaine de l'épidémie (16–22 septembre). Le taux d'atteinte global (27 pour 10 000) était faible et l'épidémie a régressé sans campagnes de vaccination de masse ni contrôles rigoureux aux frontières des personnes et des importations.

Les enquêtes portant sur 746 cas survenus au cours de la période 10 août–13 octobre 1978 et confirmés par culture ont montré que la plupart des régions du pays ont été atteintes et que la maladie a surtout affecté les nourrissons, les jeunes enfants et les hommes adultes en âge de travailler. Les symptômes étaient très discrets; moins de 20% des malades ont eu besoin d'une réhydratation spécifique. Le taux d'atteinte le plus élevé (84 pour 10 000) concernait les nourrissons de moins d'un an. Aucun véhicule ou mode de transmission courant n'a été identifié. Une étude de 35 cas et témoins par paires assorties a montré que les cas adultes avaient plus probablement que les témoins consommé de la nourriture ou des boissons hors de leur domicile avant de tomber malades.

V. cholerae a été isolé de l'eau de boisson conservée au foyer de 8 cas, mais non dans de nombreux échantillons d'aliments et d'eau du robinet. On a supposé que la transmission du choléra s'est faite grâce à l'interaction complexe de divers facteurs: personnes atteintes d'infections bénignes et asymptomatiques, aliments, eau et environnement.

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