Cholera epidemic in Baghdad during 1999: clinical and bacteriological profile of hospitalized cases

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ABSTRACT An epidemic of cholera in Iraq was anticipated for the year 1999 and a plan of notification and treatment of cases of diarrhoea was made. This paper documents the clinical and bacteriological profile of cholera cases admitted to the 6 hospitals in Baghdad during the epidemic. The number of stool culture-positive cases was 874. The peak incidence of cases (June 1999) was earlier than previous epidemics with no cases registered in November/December 1999. All age groups were affected and the case fatality rate was 1.3%. Strains isolated were *Vibrio cholerae* El-Tor O1, serotypes Ogawa (79.6%) and Inaba (12.1%), *V. parahaemolyticus* (2.0%) and non-agglutinable vibrios (6.1%). *V. cholerae* O139 was isolated from 2 cases (0.2%) for the first time in Iraq. Antibiotic resistance was noted, especially to tetracycline.

Épidémie de choléra à Bagdad en 1999 : profil clinique et bactériologique des cas hospitalisés

RÉSUMÉ Une épidémie de choléra en Iraq a été anticipée pour l’année 1999 et un plan a été établi pour la notification et le traitement des cas de diarrhée. Cet article documente le profil clinique et bactériologique des cas de choléra admis dans les six hôpitaux de Bagdad pendant l’épidémie. Le nombre de cas dont les cultures de selles étaient positives s’élève à 874. Le pic d’incidence des cas (juin 1999) était plus tôt que lors des épidémies précédentes, aucun cas n’ayant été enregistré en novembre/décembre 1999. Tous les groupes d’âge étaient touchés et le taux de létalité était de 1,3 %. Les souches isolées étaient *Vibrio cholerae* El-Tor O1, sérotypes Ogawa (79,6 %) et Inaba (12,1 %), *V. parahaemolyticus* (2,0 %) et non-agglutinables (6,1 %). *V. cholerae* O139 a été isolé chez 2 cas (0,2 %) pour la première fois en Iraq. Une résistance aux antibiotiques a été constatée, en particulier à la tétacycline.
Introduction

Cholera is a specific infectious disease of humans caused by *Vibrio cholerae* serogroup O1 and O139. It is an enzymatic process causing a brief, acute onset diarrhoeal illness, with recurrent vomiting and stools that resemble rice water. This leads to acute rapid dehydration with fluid and electrolyte loss, ending with acidosis and hypovolaemic shock, which is usually fatal if untreated [1–7]. Cholera spreads mainly via drinking water supplies contaminated by human excreta, especially from subclinical carriers and mild cases. Humans are the reservoir of the disease, despite isolation of *V. cholerae* O1 from blue crabs [8] and from the faeces of some aquatic birds [9].

Cholera differs from other enteric diseases in its clinical course (a very short incubation period) and epidemic pattern (rapidly spreading to different countries and disappearing rapidly when outbreaks subside) [10]. Cholera is endemic in Asia; from 1817 to 1923 there were 6 pandemic waves which moved through Asia then through the Americas and Africa [11–13]. When the 3rd pandemic begun in 1852, it extended to Persia, Mesopotamia and Europe as a whole. England was severely affected, allowing John Snow to complete his studies of the relationship of the disease to water supplies at the Broad Street pump [11]. Throughout history, cholera has engendered fearful reactions; nowadays, outbreaks often lead to inappropriate responses including trade embargoes, tourism restrictions, quarantine and excessive isolation and mass chemoprophylaxis.

Iraq is at risk of epidemics spreading from neighbouring countries because it lies on the routes of pilgrimage to Mecca and contains a number of holy shrines. During the epidemic of 1820, when cholera first spread to Basra, there were a great number of deaths and many sectors of the city were completely depopulated [14]. The disease spread to Baghdad, with similar consequences. After that, cholera continued to appear in several epidemic forms during the years 1871, 1889, 1894, 1899 and 1917 [15], after which the disease completely disappeared from Iraq to reappear again in August 1966 as a part of the 7th pandemic spread [11,16]. After subsidence of the 7th pandemic in Iraq, occasional outbreaks of cholera continued in Iraq: Figure 1 shows the registered cholera cases in Iraq from 1991–99. However, there was no official announcement of another epidemic until the year 1999. During 1991, a third of the total number of reported cases were in Baghdad city (602) whereas in the inter-epidemic period 1990–98, Baghdad has a smaller proportion of the total (197).

After the escalation in the incidence of cholera cases in Iraq during 1998, and due to serious shortages of safe water supplies for human use, the epidemic of cholera in the year 1999 was anticipated. A plan of notification for cases of diarrhoea and their treatment in general hospitals or isolation wards was arranged for a prospective study. This paper documents the clinical and bacteriological data of all cholera cases admitted to the 6 hospitals in Baghdad during the epidemic of 1999 in Iraq.

Methods

The 6 Baghdad hospitals chosen for the management of diarrhoea cases during the 1999 epidemic were the Medical City Teaching Hospital, Al-Mansour Paediatric Hospital, Ibn El-Khateeb Hospital for Infections Diseases, the Central Paediatric Hospital, Al-Kindy General Hospital and Al-Kadisia General Hospital.
Information about the presenting symptoms, including the state of dehydration, was recorded for all patients attending the hospitals. Stool samples were collected for analysis. Blood urea, serum creatinine and serum electrolytes were tested for severe cases of dehydration. Haematocrit values and other investigations were not routinely done. The clinical course and the outcome of cases were recorded.

The intravenous fluids available for re-suscitation of severe cases were Ringer’s lactate, normal saline (0.9%), sodium bicarbonate (1.4%) and molar (M/6) sodium lactate. The oral fluid used for rehydration of mild cases was the World Health Organization’s recommended dextrolyte solution containing 3.5 g NaCl, 2.5 g NaHCO₃, 1.5 g KCl and glucose 20 mg/L (or sucrose 40 g/L). Because of concerns about resistant stains, different antibiotics, singly or in combination, were used for 3–5 days.

The stool samples were transported in saline containers to the Reference Laboratory at the Central Public Health Laboratory, where further processing of the stool was done in peptone water and TCBS (thiosulfate–citrate–bile salts–sucrose agar) medium. Stool cultures were isolated and confirmed using specific sub-strains Ogawa and Inaba antisera. Patients with negative stool culture results were omitted from the study. Antimicrobial resistance testing was carried out routinely as part of the general stool examination according to Vandepitte et al. [17], although for time reasons the choice of antibiotics for treatment was not dependent on the results of sensitivity tests.

The number of cases of cholera admitted to the Baghdad hospitals during each month of 1999 was charted.

Results
A total of 874 patients diagnosed with cholera were admitted to the 6 hospitals in Baghdad governorate (Table 1). They were 424 males and 450 females with a male:female ratio of 0.94:1. Table 2 shows the age and sex distribution of the patients;
those below puberty were 61.4% of the total number of cholera cases admitted. Most of the cases were from the centre of Baghdad city and from Al-Thawra district in the eastern side of Baghdad, which are poor districts suffering from shortages in the water supply with unsafe drinking water most of the time.

The main presenting symptoms and signs were diarrhoea (100% of patients), dehydration (98.9%), vomiting (98.1%), and abdominal colic (13.0%). Dehydration was graded severe for 58.6%, moderate for 29.3% and mild for 11.0%; 10 cases were not dehydrated. Fever (raised temperature between 37.5–38.0 °C) was present in 24.0% of patients, mainly children. The stool was reported as white (rice water) for 83.8% of the patients; the rest were yellow (13.1%), bloody (1.3%), green (1.1%) or brown (0.7%).

The strains isolated from the stool samples of these hospitalized cases were mostly *V. cholerae* biotype El-Tor, serogroup O1, with serotypes Ogawa comprising 79.6% and Inaba 12.1%. *V. parahaemolyticus* was found in 2.0% of cases and non-agglutinable vibrios (poly-negative isolates) in 6.1%. The strain *V. cholerae* O139 was isolated from 2 cases (0.2%).

The period of stay in the hospital was ≤ 5 days for 72.9% of the admitted patients except those with complications who needed a further few days in hospital (6–8 days 20.9%, 9+ days 6.1%). The outcome was recovery without complications for 92.1% of cholera cases, recovery with complications for 6.6% and death for 1.3%. Of the 11 patients who died, 5 were males and 6 females from all age groups (Table 2). The cause of death was mainly due to severe dehydration, uraemia and acute heart failure among severely dehydrated patients presenting late. Of the 58 cases developing complications most of these were due to uraemia (43 cases), the rest due to convulsions, intrauterine death, haematemesis or other causes (15 cases).

A variety of antibiotics, singly or in combination, were used to treat cases; the choice of antibiotics was according to their availability in the hospitals. First-line treatment was with tetracycline, ampicillin or cotrimoxazole; second-line treatment was erythromycin, doxycycline, chlorampheni-
colon, gentamicin or metronidazole. Single drug use was for 34.3% of patients and combinations for 65.7%. Resistance of up to 30% was recorded for tetracycline and cotrimoxazole and lower rates for other antibiotics, but with a good sensitivity of more than 90% to ampicillin.

The time trend of reported cholera cases for the year 1999 is shown in Figure 2. This shows no case registration during the winter and spring times, while the peak admission was during June, which was nearly halved during July then rapidly decreased to zero during November and December 1999.

**Discussion**

The 7th pandemic of El-Tor cholera that started in 1961 was still active in 1998, when a marked increase was recorded in the number cases in all countries affected, with a total of 293 121 cases and 10 586 deaths reported to the World Health Organization [18]. This amounts to almost double the number of reported cases in 1997 (147 425 cases and 6274 deaths) [18–20]. This was the same upsurge of the disease observed in Iraq in 1998 (Figure 1), which led to the expectation of more cases during 1999.

In the Iraqi epidemic of 1991, and the inter-epidemic period, the number of cases of clinical disease typically peaked during the autumn months, especially September and October. The earlier peak of the 1999 epidemic in Baghdad (June) was perhaps related to the great shortage of drinking water supply for human use in addition to the lowered standards of hygiene and continuous contamination of the water sources by human faeces at this time. The attack rate among previously uninfected populations living in crowded unsanitary conditions can be very high, even though most infections are mild or sub-clinical. The epidemic ended in November 1999 where there were no clinical cases registered and no culture-positive results for *V. cholerae* were found from patients presenting with diarrhoea.

All ages were affected, but the majority (61.4%) were 15 years or under. These findings are similar to those reported from other cholera-endemic areas, where the highest incidence of clinical cholera is usu-
ally observed among toddlers, pre-school children and women of childbearing age [2,3,7,10,21,22].

The clinical presentation of diarrhoea, vomiting, rice-water stool and severe dehydration observed in our patients is the classic symptomatology of cholera [1,2,10]. The case fatality rate among our hospital patients was 1.3%, less than the global case fatality rate of cholera of 4.3% during 1997 and 3.6% during 1998 [18]. A high mortality rate in cholera epidemics could be due to inadequate use of oral rehydration, use of inappropriate intravenous fluids and inadequate experience of health workers in management of severe cholera [23–28]. The higher global case fatality rate might be because nearly 72% of world cholera cases were in African countries where poverty and lack of resources are a more serious problem. Furthermore, our anticipation of the 1999 epidemic meant that hospitals and wards had made preparations to receive the clinical cases, ensuring the availability of proper fluids for rehydration and training in their correct use; this is also likely to greatly reduce the case fatality rate.

*V. cholerae* O139 was isolated from 2 cases who had severe dehydration diarrhoea indistinguishable from the clinical signs of cholera due to *V. cholerae* O1. A resurgence has been seen of this Bengal strain in many countries since 1992 [29] and now in Iraq. Non-O1 vibrios strains have been implicated in outbreaks of food-borne disease, isolated from as many as 13% of patients with cholera-like disease during cholera epidemics. Eko et al. reported the spectrum of *Vibrio* spp. diarrhoea in the lower cross-river of Nigeria [30]. In agreement with our study, they reported the non-O1 cholera infection was associated with watery diarrhoea, mild to moderate in severity, and the diarrhoea was sometimes bloody, occasionally accompanied by vomiting, abdominal cramps and low-grade fever.

Due to the emergence of multiple-resistant strains of *V. cholerae* [24–26], we used a variety of antibiotics singly or in combination. However, antibiotic resistance, especially to tetracycline, was noted. Mahon et al. found in a study of cholera O1 in the United States that the proportion of isolates resistant to at least 1 antibiotic rose from 3% in 1992 to 93% in 1994 [27]. A single dose of ciprofloxacin is currently more effective than a single dose of doxycycline and may be particularly valuable where resistant strains to tetracycline are common [24].

**Conclusions and recommendations**

The study has shown *V. cholerae* serostrain O139 isolated in Iraq for the first time. Early planning to contain the disease may have reduced the case fatality rate, especially through proper regimens of rehydration. Resistance to different antibiotics was noted. Careful and regular stool culturing from diarrhoea cases suspected of cholera throughout the year is recommended for detection of index cases before epidemics start.

**References**


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**International travel and health 2005. Situation as on 1 January 2005**

International travel is undertaken by large, and ever increasing, numbers of people for professional, social, recreational and humanitarian purposes. Travellers are exposed to a variety of health risks in unfamiliar environments. Most such risks, however, can be minimized by suitable precautions taken before, during and after travel, and it is the purpose of this publication to provide guidance on measures to prevent or reduce any adverse consequences for travellers’ health. The book is addressed primarily to medical and public health professionals who provide health advice to travellers, but it is also intended to provide guidance to travel agents and organizers, airlines and shipping companies. As far as possible, the information is presented in a form readily accessible to interested travellers and non-medical readers. The printed edition is now published only every second year but an Internet version (http://www.who.int/ith) allows on-going updating and provides easy links to other information, such as news of current disease outbreaks of international importance. Further information on this publication can be obtained from WHO Press: http://www.who.int/bookorders/anglais/home1.jsp?sesslan=1