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Practical field epidemiology to investigate a cholera outbreak in a Mozambican refugee camp in Malawi, 1988

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Summary

Of all populations affected by cholera, refugees are at particular risk of infection due to overcrowding and poor sanitation. Between 15 March and 17 May 1988, 951 cases of cholera were registered at the cholera treatment centre in a Mozambican refugee camp in Malawi. The epidemic duration was 65 days. *Vibrio cholerae* biotype El Tor serotype Inaba was isolated. To identify high-risk groups and potential risk of acquiring the disease, an epidemiologic investigation was conducted. The attack rate of recorded cases was 2.6% with a range from 0.9 to 5.1% for different sections of the camp. The case fatality rate was 3.3% and decreased from week 1 to week 6. The epidemic started in the section near the market place and radiated out. A matched-pair case-control study of food and water consumption was performed early in the outbreak. It showed that cases were more likely to use shallow wells (surface wells) instead of boreholes compared to controls (OR = 4.5, CI = 1.0–20.8, $P = 0.04$) and that cases were more likely to have had contact with the market than controls (OR = 3.5, CI = 0.7–16.8, $P = 0.09$). None of the food items available at the market was more likely to be preferred by cases than controls. Recommendations included early case finding and treatment, temporary closure of the market, tetracycline prophylaxis of contacts, and water chlorination.

Background

Between 1971 and 1988, large-scale population movement has occurred across international borders (Toole & Waldman 1988). Population displacement is associated with low nutritional status, inadequate water and food supply, lack of shelter, and overcrowding (Shears *et al.* 1987; Toole *et al.* 1988). These conditions encourage the rapid spread of epidemics. Cholera in refugee camps has been observed repeatedly within the past 5 years: Sudan 1985, Ethiopia 1984–85, Somalia 1985 and, more recently, Malawi 1986–88 (Médicine Sans Frontières 1985–86, unpublished data).

The seventh pandemic of cholera was first recognized in Africa in 1970 and was due to a *Vibrio cholerae* serotype Inaba. In 1973 the first epidemic occurred in Malawi. From 1973 major epidemics have caused up to 22 476 cases (1975) with a case fatality rate ranging from 1 to 50% in some places (Malawi Ministry of Health 1984). Worldwide transmission has been associated with consumption of infected food (Gunn *et al.* 1979; Holmberg *et al.* 1984) and water (Gangarosa & Mosley 1974) and with person to person transmission (Benenson *et al.* 1965; Deb *et al.* 1982). Despite the improving knowledge of cholera transmission, major epidemics continue to occur in the developing world (Ministry of Public Health and Affairs 1986; Rogers 1975).

Introduction

From 1986 to 1988, approximately 400 000 Mozambican refugees fled to Malawi and settled in camps or villages along the borders (Figure 1). On 15 March 1988, 15 suspected cases of cholera were reported from one of these camps located in the south of Malawi.

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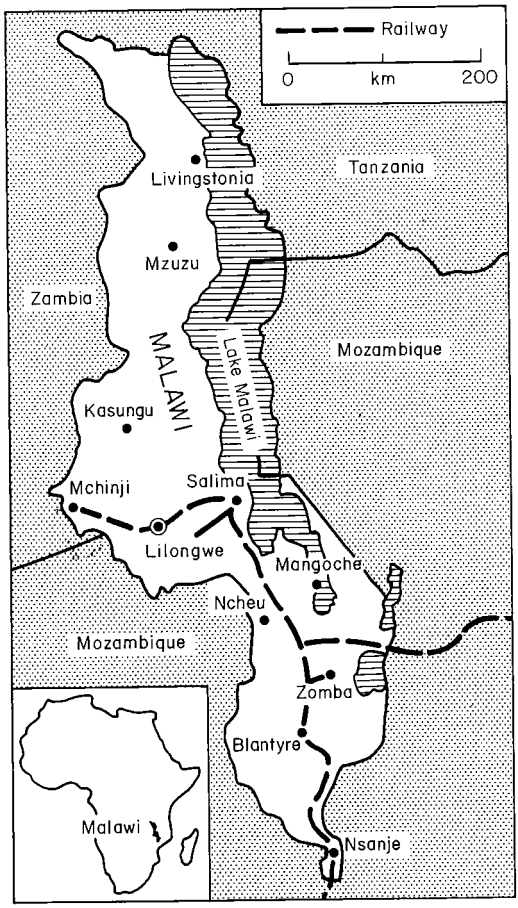

Figure 1. Malawi and its neighbouring states.

The 29 745 refugees were registered in the 20 sections of the camp and lived in a highly populated area with poor sanitary conditions. Health care was provided by Malawian medical personnel and Médecins Sans Frontières medical staff under the supervision and coordination of the Malawian government and the United Nation High Commission for Refugees (UNHCR). Food distribution occurred twice a month and water came from 135 water sources; 79 unprotected shallow wells, 27 protected shallow wells and 29 boreholes. An epidemiologic investigation was conducted to describe the characteristics of the epidemic by time, place and persons, to identify potential risk factors for the outbreak, and to develop recommendations to reduce the spread of the epidemic.

Materials and methods

EPIDEMIOLOGY

From the first day of the epidemic, a registry was developed at the cholera treatment centre established near the camp. Data collected among admitted cases included information on the demographic variables age, sex, ethnic group, location and date of arrival in the camp, clinical signs and symptoms, date of onset of illness, duration of illness and treatment, and outcome.

A case of cholera was defined as a person with an acute onset of profuse watery stools or profuse vomiting or collapse due to dehydration, who was treated at the cholera treatment centre between 15 March and 17 May 1988.

To identify risk factors for illness and a possible common source, a matched-pair case-control study was performed using the 51 earliest cases of the outbreak. Each case was matched for age and sex with one control. All persons who had had a recent history of diarrhoea or vomiting were excluded from the control group. To look for a control the investigator proceeded to the third-nearest household in the neighbourhood of each reported case household. If no control was found in the selected household, the next closest household was visited and so on. The information collected from cases and controls included family size, recent travel outside the camp, contact with the market (bought food at the market), use of left-over food, source of water supply, contact with the nearby river, contact with the under-five clinic and the supplementary feeding centre, contact with a case, and existing latrine for the family. Each case and control was asked to answer a food preference questionnaire based on food items available at the camp market. For each of these variables the 7 days prior to the onset of illness among cases was used to define the period of exposure. Interviews were conducted by two epidemiologists assisted by two translators.

Attack rates (AR) were calculated according to time, place and persons characteristics. Matched paired Odds Ratios (OR) were calculated as well as 95% JR, SG, NB Confidence Intervals (CI) (Robins *et al.* 1986). McNemar

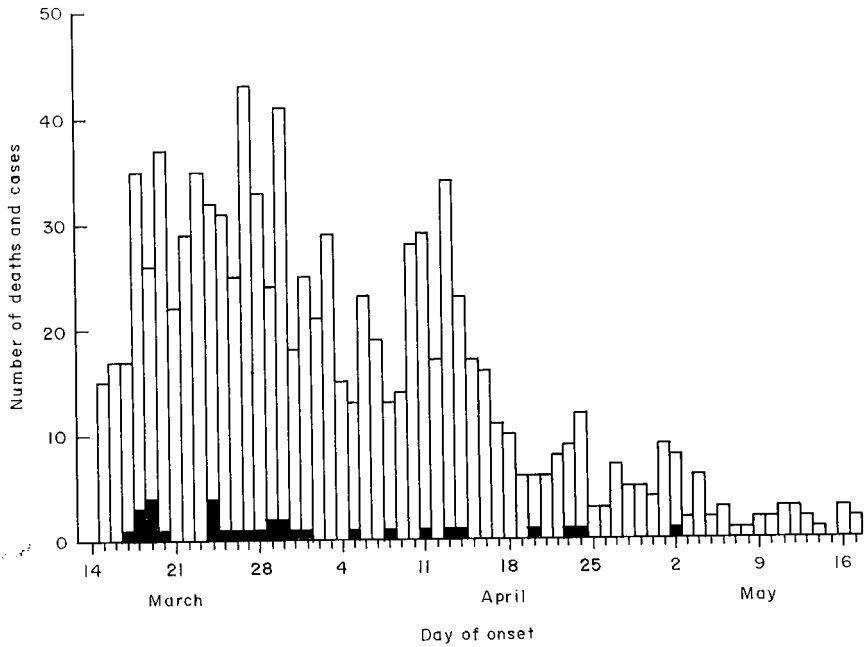

Figure 2. Cholera, Malawi, 15 March–17 May 1988. Cases and deaths by day of onset. ■, Deaths; □, cases. Sources: Médicins Sans Frontières and Malawian Ministry of Health.

uncorrected chi squared and derived *P*-value were calculated.

LABORATORY

Stool specimens obtained from five patients were sent to the Blantyre Laboratory, Malawi, as well as to the Pasteur Institute in Paris. Transportation was done using filter paper sealed in a plastic bag. Positive specimens were serotyped and antibiotic sensitivity was tested. Water samples obtained from 24 wells in the camp were tested for faecal coliforms using OXFAM kits for sanitation.

Results

EPIDEMIOLOGY

Time

Between 15 March 1988 and 17 May 1988, 951 persons seen at the cholera treatment centre had symptoms consistent with the case definition. Among them 784 came from the camp (attack rate = 2.6%) and 167 from the neighbouring villages (denominator unknown).

The cases and deaths by day of onset are shown in Figure 2. The curve is marked by a rapid increase, a peak on 28 March with subsequently smaller peaks, and a gradual fall in cases over time. Several waves can be identified with decreasing size with time.

Person

Deaths were recorded during the outbreak with an overall case fatality rate (CFR) of 3.3%. The CFR was highest during the 1st week and decreased slowly to reach 0% towards the second part of the outbreak (Table 1). The mean duration of illness was 3 days. Patients received intravenous rehydration for 24 h, the median amount of i.v. fluid administered was 3.25 l per case (range 0–15 l). Four cases of collapse without diarrhoea or vomiting were observed. Among the 26 deaths observed at the cholera treatment centre, five were due to pulmonary oedema secondary to intravenous rehydration. The attack rate among males was higher (2.9%) than among females (2.4%). Among the 784

Table 1. Cholera: Mankhokwe, Malawi, 15 March–17 May 1988. Case fatality rate by week

Week	Deaths	Cases	CFR %
1	9	161	5.6
2	6	165	3.6
3	5	140	3.6
4	2	133	1.5
5	1	100	1.0
6	3	33	9.1
7	0	28	0.0
8	0	13	0.0
9	0	11	0.0
Total	26	784	3.3

Table 2. Cholera: attack rate by age group. Mankhokwe camp, Malawi, 15 March–17 May 1988

Age group (years)	Cases	Total	Attack rate (%)
<5	131	5303	2.5
5 to 14	261	12 351	2.1
⩾15	392	12 091	3.2
Total	784	29 745	2.6

cases in the camp, 382 were males (48.7%). Among the 402 women, 22 were pregnant and four had an abortion (third trimester); one of the abortions concerned a twin pregnancy. The CFR was 2.4% (9/382) among males compared to 3.7% (15/402) among females (RR = 0.6, 95% CI = 0.28–1.43). The AR was highest in the over 15 years age group (Table 2) and only four cases were observed in the under 1 year age group. The case fatality rate decreased with increasing age (5.3% among <5 years, 4.6% among the 5–14 age group and 1.3% among those ⩾ 15 years of age).

Place

The AR by section ranged from 0.9% (section 15) to 5.1% (section 5). The outbreak started in section 5 where the market-place is located and spread centrifugally (Figure 3). No precise index case could be identified; however, the wife of one of the first cases who lived in section 5 of the camp had been admitted to a nearby hospital 4 days prior to the beginning of the epidemic. This women was admitted with profuse diarrhoea and vomiting and died within 12 h of admission. The interview with the husband did not reveal any travel by his wife outside the camp within 7 days prior to her illness.

Sections 5 and 11 kept the highest AR, sections 19 and 20 reached a high AR towards the end of the epidemic. Section 5 had the highest AR for the age group 5–14 (6.7%).

MATCHED-PAIR CASE-CONTROL STUDY

Cases were more likely to have had contact with the market within 5 days prior to onset of illness than controls (OR = 3.5, CI = 0.7–16.8, $P = 0.09$) (Table 3). Two water sources were available: shallow wells and boreholes. Cases were more likely to use shallow wells as a water source than controls (OR = 4.5, CI = 1.0–20.8, $P = 0.04$). None of the food items available at the camp market was more likely to have been preferred by cases than controls. Cases were not more likely to use left-over food than controls.

ENVIRONMENTAL

Ten boreholes, 12 protected shallow wells and two unprotected shallow wells were tested for faecal coliforms. Four protected shallow wells and one unprotected shallow well were positive. None of the 10 boreholes tested (including the borehole located at the market-place) showed evidence of faecal coliforms.

LABORATORY

The five stool specimens that were sent to Blantyre laboratory and to Pasteur Institute were positive for *Vibrio cholerae* serotype Inaba. The vibrio was sensitive to all antibiotics tested.

Discussion

In this epidemic of 784 cholera cases the early phase of the outbreak is consistent with a possible common source. The rest of the epidemic curve with several waves agrees with person to person transmission.

The epidemic period is consistent with the seasonal pattern seen on surveillance data where cholera incidence increases every year

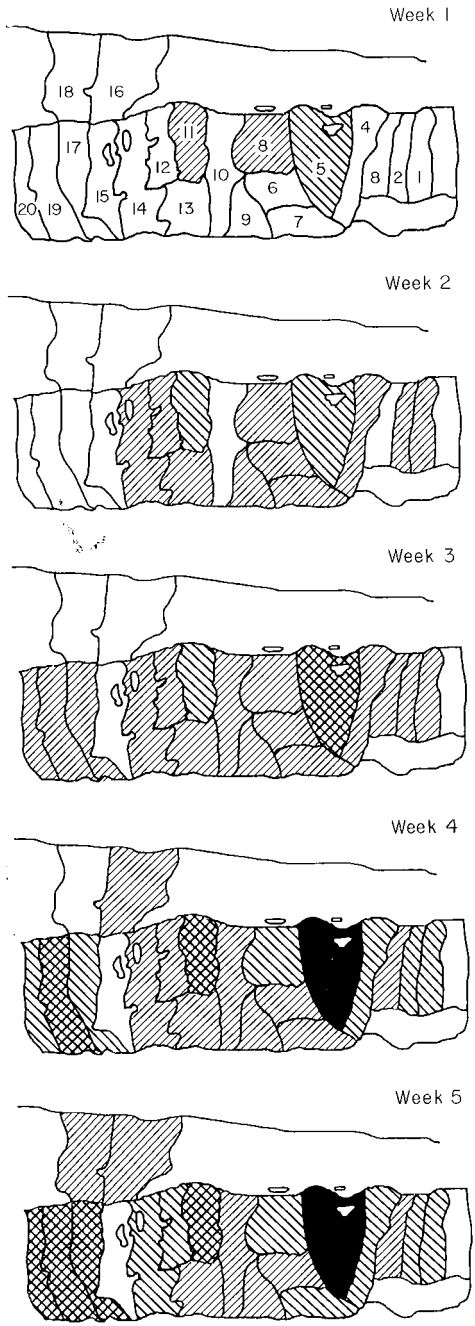

Figure 3. Cholera: attack rates/100 by sections and by weeks. Mankhokwe, Malawi, 15 March–18 April 1988. □, <1; ▨, 1–1.9; ▧, 2–2.9; ▩, 3–3.9; ■, ⩾4%.

from January to May (end of the rainy season in Malawi). The occurrence of a major epidemic may have been favoured by the recent gathering of almost 30 000 refugees in a camp with a high-density population. The prevalence of malnutrition in the camp (10% of the children less than 5 years of age were less than −2 s.d. of the median weight for height) may have also increased the risk to acquire disease. It should be noted that the overall attack rate of 2.6% reflects only severe cases treated at the cholera treatment centre and that milder cases may have gone undetected, therefore underestimating the AR.

Section 5 (the market-place) was the starting point of this outbreak. The shape of the early phase of the epidemic curve led us to look for a common source located at the market. The AR among children 5–14 years of age was much higher (6.7%) than in other sections. Children aged 5–14 years used to spend most of their day time at the market and were therefore more likely to have been exposed. But overall, the association between the market and illness did not reach statistical significance and none of the food items included in our food questionnaire was associated with illness. Furthermore, the borehole located at the market was tested negative. Nevertheless, section 5 maintained the highest AR during the outbreak and no levelling of AR by section occurred with time. It remains unclear why that section kept a higher AR. Although not measured, the high population density in this section may have contributed to the high AR.

If week 6 when the case fatality rate was high due to small numbers is excluded, the case fatality rate decreased from week 1 to week 9. This trend remains when taking out the five iatrogenic cases (respectively 4.3, 3.6, 2.1, 1.5, 0, 9.1 and then 0% for weeks 7–9). This can be due to a better treatment facility, an earlier case finding, the decrease of the vibrio virulence and the development of mass immunity.

Overall the case fatality rate of 3.3% is low if we take into account the general health status of the population (refugee situation, malnutrition aggravated by a recent major measles epidemic). Other cholera epidemics described in refugee camps showed attack rates and case fatality rates of respectively 3.5 and 5.7% in Kobo, Ethiopia, 1985; 2.9 and 21% in Korem, Ethiopia, 1985; 3.8 and 1% in Guirba, Sudan,

Table 3. Cholera: Mankhokwe, Malawi, 15 March–17 May 1988. Distribution of cases and controls matched for age and sex according to exposure

Exposure	Discordant pairs		Odds Ratio and 95% Conf. Int.	*P*
	Case exposed	Control exposed		
Market contact	7	2	3.5 (0.7–16.8)	0.09
Food left-over	9	10	0.9 (0.4–2.2)	0.81
River contact	10	4	2.5 (0.8–7.0)	0.10
Travel outside camp	10	4	2.5 (0.8–7.0)	0.10
Shallow wells	9	2	4.5 (1.0–20.8)	0.04

1985; 0.96 and 2.5% in Wad Kowali, Sudan 1985 (Shears *et al.* 1987).

Results from the matched-pair case-control study remain difficult to interpret due to the small sample size and a high proportion of cases and controls exposed to the studied risk factors, leading to small numbers of discordant pairs for the matched-pair analysis. However, contact with the market (OR = 3.5) and consumption of water from shallow wells (OR = 4.5) may have played a role in the transmission of this epidemic. In this refugee camp the water-table is located 2–4 m below the surface; shallow wells take their water from 5 m below the surface whereas boreholes take their water from 40 m. Fifteen days prior to the outbreak, major rains occurred in the camp, destroying half its latrines. Most of these latrines were 3–4 m deep and were therefore in contact with the water-table. Only five of the wells tested during the investigation revealed the presence of faecal coliforms. It is possible that the different attack rates among sections of the camps may be explained by the different distribution of shallow wells and boreholes among them. Unfortunately this hypothesis was not tested.

Tetracycline prophylaxis (250 mg tablets × 6/day for 3 days) was recommended for family contacts of all cases. Early case finding was performed by home visits conducted by the health care workers in the camp. Early treatment by oral rehydration therapy (ORT) or intravenous therapy was implemented at a cholera treatment centre established near the camp. Despite the poor nutritional status of this refugee population the CFR was kept as low as 3.3% through intensive early case finding. Recommendations were made to build boreholes instead of shallow wells. Chlorination of the water supply was recommended and a daily distribution of chlorine for family reservoirs was developed. Limitation of population movements was advised to health authorities and the market was temporarily closed. Particular recommendations were given in order to divide the food distribution sessions in several places so as to avoid the gathering of almost 100 000 refugees from the neighbouring area in the camp. In addition, to reduce the risk of spread of epidemic during funerals, each corpse was disinfected with chlorine at the cholera treatment centre and put in a plastic bag that was not reopened before or during the funeral or burial. Burials were closely supervised by health care workers in each of the sections of the camp.

This outbreak illustrates the risk to displaced people in refugee camps where there is crowding and poor sanitation which increases the risk of epidemics such as cholera. We would like to emphasize the need for concurrent epidemiologic surveillance and outbreak investigation as well as therapeutic and preventive measures. Descriptive information of epidemics and their investigation in the early phase can greatly help to identify high-risk groups and to define intervention priorities.

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