A COMMON-SOURCE EPIDEMIC OF MIXED BACTERIAL DIARRHEA WITH SECONDARY TRANSMISSION

C. R. KAPADIA, P. BHAT, S. J. BAKER AND V. I. MATHAN


An epidemic of acute diarrhea in a village in southern India in 1972 was associated with a high rate of isolation of enteric pathogenic bacteria from the patients and lasted for three months. There was no significant association between the prevalence of enteroviruses or parasites and cases of diarrhea. The epidemic started as a common-source outbreak due to the contamination of well water, and there were many secondary cases probably due to a person-to-person spread. The illness did not produce chronic diarrhea or malabsorption.

diarrhea; disease outbreaks; Escherichia coli; Salmonella; Shigella

The control, prevention, and therapy of acute diarrhea in developing countries are dependent on a full understanding of epidemiologic characteristics. During the last 20 years, several epidemics have been studied in the rural areas of North Arcot District of Tamil Nadu (formerly Madras State) in southern India during a surveillance program. Several of these epidemics were of tropical sprue, while others were due to bacterial infection (1-3). The present report describes an epidemic due to bacterial infection with a possible common-source outbreak followed by extensive secondary transmission.

THE VILLAGE

Ala is an agricultural village 25 km south of Vellore with 1,098 people (535 males and 563 females) in 247 families (average family size 4.4 persons). The majority of the adults worked as farm laborers and ate a vegetarian diet similar to that described by Rao and Rao (4). Drinking water was obtained exclusively from wells and was never boiled. There were four semi-protected open public street wells (I-IV, figure 1) and several unprotected house wells. People used their own utensils for drawing water. There were no sanitary facilities, and the neighboring fields were used for defecation. Children and the ill often defecated within the village. During September and October, heavy rain made the entire area water-logged.

METHODS

A house-to-house survey of the village of Ala was completed by October 12, 1972, within five days of obtaining the first report of the outbreak. All persons who gave a history of increased number of stools or more liquid stools than previously were recorded as cases of diarrhea. Information regarding the dates of onset of diarrhea prior to the completion of the initial survey on October 12 was retro-

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Figure 1. Spread of the epidemic throughout the village in the three periods. Street wells I-IV and the private well (P) in the northeastern section are shown. Each square shows the position of one household. V, vacant houses. New cases occurring in each period are shown by symbols as indicated. Note the clustering of cases in the northeastern section of the village.
spective. It is our experience during epidemics that, in this part of rural southern India, by carefully relating to important village events, fairly precise information regarding the actual date of onset of an illness can be obtained retrospectively up to about a month. The epidemic curve is therefore plotted on a monthly basis up to August 31 and weekly thereafter. Since there were no medical facilities in the village, the field team gave treatment to patients who sought their help. The village was kept under surveillance until the subsidence of the epidemic and was re-surveyed at eight months and at 20 months.

Stool specimens for laboratory studies were obtained and transported on ice to the laboratory within one hour of collection. Specimens were obtained from 48 people with diarrhea, and from 38 people within two weeks and from 57 people more than two weeks after subsidence of diarrhea.

Standard methods were used for isolation and identification of fecal enteric bacterial pathogens, as described in detail elsewhere (5). Viral isolation was done in primary bonnet monkey kidney cells and in newborn mice by the Department of Enterovirology (6, 7). Stools were examined for parasites directly by preparing smears in normal saline and in Lugol’s iodine and after zinc sulfate concentration (8). Water collected from wells was tested by the presumptive coliform count method for fecal contamination (9). In an attempt to identify enteric pathogens from well water, several liters were filtered through Seitz filters, and the filter pads were subjected to bacteriologic analysis (5).

Six patients were admitted to a metabolic ward for detailed evaluation. In addition to clinical and sigmoidoscopic examination, each patient had detailed hematologic investigation and tests of intestinal absorption (10). Some of these tests were repeated 20 months later.

![Figure 2. Epidemic curve. The number of new cases of diarrhea detected are shown. Period A, July–August, data obtained by retrospective survey and shown as cases per month; period B, September 1–14; period C, September 15–November 30, data obtained by prospective survey and shown as new cases per week.](image)

**RESULTS**

**The epidemic**

During July and August, there were 22 cases of acute diarrhea detected by the retrospective survey (period A, figures 1 and 2). In the first two weeks of September (1st to 14th; period B), 45 new cases of diarrhea occurred. In the week immediately following (September 15–21), there were only three new cases; however, subsequently the weekly incidence of new cases increased and reached a peak in the second week of October (19 cases), decreasing gradually thereafter (September 15 to November 30; period C, 82 cases). Clustering of cases in the northeastern section of the village was apparent during periods A and B (figure 1). During period C, more new cases occurred in other areas of the village, although the attack rate was still higher in the northeastern section (figure 1).

The overall attack rate was 13.5 per hundred population. Males and females were equally affected. The attack rate in people drinking water from public well I
<table>
<thead>
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<th>Age group (years)</th>
<th>Total village</th>
<th>Periods</th>
<th>Wells</th>
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<tr>
<td></td>
<td></td>
<td>A</td>
<td>B</td>
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<tr>
<td>First year</td>
<td>7.3</td>
<td>0</td>
<td>1.8</td>
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<tr>
<td>1–4.9</td>
<td>32.9</td>
<td>2.6</td>
<td>4.0</td>
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<td>5–11.9</td>
<td>19.2</td>
<td>0.5</td>
<td>3.3</td>
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<td>12–17.9</td>
<td>8.0</td>
<td>1.5</td>
<td>3.7</td>
</tr>
<tr>
<td>18+</td>
<td>13.8</td>
<td>2.5</td>
<td>4.8</td>
</tr>
<tr>
<td>Total</td>
<td>13.5</td>
<td>1.9</td>
<td>3.3</td>
</tr>
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*Attack rates calculated per period. Since period A (eight weeks) and period C (16 weeks) are longer than period B, attack rates per week in period C are similar to those in period B.

1 Period A, July–August; period B, September 1–14; period C, September 15–November 30.

and adjacent private well P located in the northeastern section of the village was significantly (p < 0.0005) higher than that in the rest of the village in all three periods of the epidemic (table 1). In the population obtaining their drinking water from wells other than I and P, the attack rate in period C was significantly higher than that in the rest of the village in all three periods of the epidemic (table 1). The attack rate in preschool children was significantly higher than that in all other age groups only during period C, both among those obtaining their drinking water from wells I and P and those obtaining their drinking water from other wells (table 1). However, none of 13 children less than six months of age who were exclusively breast-fed (not even given drinking water) were affected in the epidemic, while one infant who was not breast-fed developed diarrhea.

Microbial and parasite isolations

Recognized enteric bacterial pathogens were isolated from 24 of 48 (50 per cent) specimens obtained during diarrhea; eight of 38 (21 per cent) specimens obtained up to two weeks after subsidence of diarrhea; and only five of 57 (9 per cent) obtained more than two weeks after subsidence of diarrhea (figure 3). Different species of *Shigella* constituted 88 per cent of the isolates, and enteropathogenic *Escherichia coli* (different serotype) and *Salmonella* groups accounted for 24 per cent and 8 per cent, respectively. *Plesiomonas shigelloides*, *Aeromonas hydrophila*, and *Edwardsiella tarda* were identified in the stools of nine, seven, and two subjects, respectively; however, their prevalence bore no relationship to the time of sampling the stools in the presence of diarrhea. They were therefore not considered etiologically significant.

The rate of enterovirus isolations (ECHO 10 isolations; coxsackie, 11 isolations; polio 2, two isolations; untyped, two isolations) and the prevalence of parasites (Giardia lamblia 16.4 per cent, Strongyloides stercoralis 6.3 per cent; Entamoeba histolytica 0.7 per cent) were similar in patients with diarrhea and those who had recovered at the time of stool sampling (figure 3).

In the second week of October, water from all four public wells and two private wells, including P, was sampled and was "highly unsatisfactory" by presumptive coliform counts. *P. shigelloides* and *A. hydrophila* were isolated from all the wells, but *Shigella*, enteropathogenic *E. coli*, and *Salmonella* were not grown.

**Clinical features**

Clinical features were studied in a group of 63 patients. Diarrhea alone was present in 68.3 per cent, diarrhea with
mucus in 19 per cent, and mucus and blood in 12.7 per cent of the patients. The majority of patients required only symptomatic therapy for their diarrhea, but eight were given chemotherapeutic agents in addition. Four patients with diarrhea died, three who did not seek any medical attention (ages two, 72, and 79 years) and one with Shigella dysentery and a fulminant pneumonia who died in spite of therapy in hospital.

The duration of illness ranged from one to 40 days (mean 11 days, median nine days), 29 per cent of cases lasted longer than two weeks, and 9 per cent lasted one month or longer.

**Studies of intestinal function**

In five of the six patients hospitalized, d-xylene excretion was low at the time of initial illness. In three of these five patients, the xylose absorption returned to normal during follow-up study; in one, it remained abnormal, and in the other, the test could not be repeated. Only two patients had an elevated stool fat excretion. One of these had only very mild steatorrhea, which disappeared on treatment of a concomitant Strongyloides infestation. The other had elevated stool fat, which declined but did not subside on treatment of her shigellosis with an antibiotic. All six patients had normal vitamin B12 absorption when first tested.

**DISCUSSION**

The rate of isolation of pathogenic bacteria in relationship to diarrheal illness (figure 3) is strong evidence that this epidemic was due to bacterial infection. The rate of isolation of enteroviruses was not related to clinical illness, but the techniques used did not identify other viruses such as rotavirus, adenovirus, or Norwalk-like agents which are established as causing acute diarrhea. The bacterial isolation rate of 50 per cent obtained during diarrheal illness is higher than that obtained in some studies carried out under field con-
ditions (11). However, in one study using similar techniques, higher isolation rates were obtained in an epidemic in southern India due to *Shigella dysenteriae* type 1 (12).

The variety of bacterial pathogens seen in the present epidemic appears to be unique and raises the question whether these pathogens were responsible for the illness or whether the diarrhea was due to some undetected agent which resulted in the "flushing out" of pathogenic organisms. In another village affected by an epidemic of tropical sprue, a variety of enteropathogenic bacteria were isolated from the stools of only 6 per cent of 127 patients with diarrhea and from 6 per cent of 271 asymptomatic controls, an isolation rate similar to that in a normal southern Indian rural population studied on a longitudinal basis (3). It therefore appears that, although unusual, this epidemic of diarrhea was due to a mixed bacterial infection.

In all three periods of the epidemic, the number of cases and attack rates were higher in the northeastern section of the village near wells I and P. The reasons for the clustering of cases in this area during period A are not apparent, but the abrupt increase in the number of cases during period B (September 1–14) and the similar attack rates in all age groups suggest a common-source type of epidemic mainly confined to the northeastern section of the village. The significantly higher attack rates in individuals obtaining their drinking water from wells I and P suggest that these wells could be the source of infection. The total absence of illness among infants exclusively breast-fed (even without water supplementation) and the fact that there was only one child who was not breast-fed who developed diarrhea further support this theory.

Well P is unprotected and level with the surface. Since there was heavy precipitation toward the end of period A, it is conceivable that, with the concentration of ill people in this area, infected fecal material was washed into the well. Well I is surrounded by a concrete wall; however, this affords only partial protection. In addition, there is considerable sharing of utensils for drawing water from wells I and P. Contamination of well I, therefore, could easily have occurred. None of the protected wells in other areas of the village were shared by the same users to the extent that wells I and P were. The well water tested only during period C showed heavy fecal contamination, although no pathogenic organisms were isolated. This type of contamination of shallow wells is not unusual in this part of southern India.

A change in the pattern of the appearance of new cases is seen in period C. Cases occurred throughout the entire village during a 10-week period with a significantly higher attack rate in the preschool age group. Since the ill and children defecate inside the village, especially near dwelling places, fecal contamination of utensils and of hands can easily occur as well as secondary spread by person to person.

Acute bacterial diarrheas, although usually of short duration, may last for longer periods in the rural populations of developing countries (13, 14). The high proportion (29 per cent) of patients in whom symptomatic diarrhea lasted for longer than two weeks may be a reflection of lowered general resistance (15), but, in the present context, could equally well have been due to repeated reinfection with different organisms.

In ill-nourished populations, temporary malabsorption following acute bacterial diarrheas has been recorded (16). Epidermics of tropical sprue with many patients having persistent malabsorption have been reported from southern India (1–3). Although the population affected in the present epidemic is nutritionally similar to the population affected by epi-
demic tropical sprue, none of the patients developed features suggestive of tropical sprue. This is further evidence that not every type of gastrointestinal infection will result in the syndrome of tropical sprue.

Acute diarrhea continues to be a major cause of morbidity and mortality in the developing world, but the dynamics of spread of infection in the community is not fully understood. The data presented here suggest that 1) in the context of the crowded habitat of the southern Indian village, drinking water sources may be important in transmission of enteric pathogens; 2) more than one form of transmission can occur; and 3) several agents may be involved at the same time in causing an epidemic.

REFERENCES