Shigella dysenteriae Type I Infection

in Southern India During the 1970's

A change in the etiological pattern of acute diarrheal disease and dysentery in this region was seen during the 1970's with an increasing number of cases of Shigella dysenteriae type I being reported. In addition to the etiological change in patients attending hospital with dysentery, six epidemics due to this organism were studied between 1972 and 1978. Etiological data on some of these epidemics and a major vehicle for spread in several instances was the water supply.

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Preliminary information on the occurrence of unusual numbers of cases of diarrheal and dysenteric disease was obtained from several sources: patients attending the hospital, the government public health workers, the Wellcome Trust, London, U.K.

The work of the Wellcome Research Unit is supported by the Wellcome Trust, London, U.K.

RUNNING TITLE: Shiga dysentery in southern India.
The Shiga bacillus has been considered an unusual enteric pathogen in southern India (1). A change in the etiological pattern of acute diarrhoeal disease and dysentery in this region was seen during the 1970's with multi-drug resistant Shigella dysenteriae I becoming a major cause of such illness episodes (2). In addition to the etiological change in patients attending hospital with dysentery, six epidemics due to this organism were studied between 1972 and 1979. This paper records epidemiological data on some of these epidemics and documents that a major vehicle for spread in several instances was the water supply.

**Epidemiological Methodology**

Preliminary informations on the occurrence of unusual numbers of cases of diarrhoea and dysentery were obtained from several sources: patients attending the hospital, the government public health workers, newspaper reports or by the field team maintained by the Unit for diarrhoeal disease surveillance. Each initial report was followed up by a field visit. In
instances where the epidemic was farther than 100 km from Vellore, site visits for up to a week were done for spot surveys and obtaining bacteriological specimens. In nearer epidemics the entire village was enumerated and detailed clinical and epidemiological data were collected. Patients were treated in field clinics established in the villages and those requiring hospitalization were brought to the base hospital. The modifications of the methodology used will be clear as each epidemic is described in detail.

THE EPIDEMICS


Newspapers reported a serious diarrhoeal disease affecting a large number of people in a district of Tamil Nadu, 600 km south of Vellore in December 1973. This was confirmed by a telephone conversation with the Medical Superintendent of a hospital in that area. A field team visited three medium sized hospitals and the district headquarters hospital and clinical data was obtained on about 150 patients. The patients were hospitalised with an acute dysenteric illness with an
average duration of 3-5 days prior to hospitalisation. In 87% the stools were bloody and mucoid and passed frequently with lower abdominal cramps and tenesmus. Half the patients were febrile at the time of admission. They were usually treated with a variety of antibiotics without much relief but had a self limited illness lasting about 10-15 days. In preschool children and among those over 50 years the disease was more acute and systemic manifestation of toxæmia, decreased urinary output and complicating infections such as pneumonia were present.

Stool samples were obtained from 16 patients in one hospital and 10 of the 16 stools grew Shigella dysenteriae type I (table 1) resistant to all the usual antibiotics other than neomycin, kanamycin and gentamycin.

New cases continued to occur in this region from September 1973 to February 1974 and died down thereafter. A conservative estimate of the number of affected individuals based on available records was about 40,000 patients, preschool children being maximally affected.

1976 Vad

Vad is a small agricultural village about 40 km north of Vellore. 55 cases of an acute dysenteric illness were recorded in this village between 21.7.76 and 31.8.76
in a population of 395. Subsequent to 31.8.76, no further new cases were recorded (Fig. 1). All age groups were affected with the highest attack rate in preschool children (Table 2). Cases occurred in 38 percent of families.

The only pathogenic organism isolated in 7 of 23 patients and none of the controls who were sampled was Shigella dysenteriae type I which was sensitive only to kanamycin, neomycin, polymyxin and septran (Table 1).

The village was situated in a valley between two low hills and obtained its water supply from three unprotected shallow wells. The water from these wells were unsatisfactory by the presumptive coliform count method although Shigellae were not isolated. The village was first visited on 12.8.76 and all the water sources were chlorinated on 17.8.76. Subsequent to this the epidemic rapidly disappeared, no further cases occurring after 31.8.76. The chlorination of the wells was maintained till 30.9.76.

Clinically the patients resembled other cases of acute bacillary dysentery seen earlier. None of the patients required hospitalisation and all were managed in the field with Kaolin mixture and maintenance of
hydration and nutrition. Neomycin syrup was given to three children below five years of age. Prior to the arrival of the field team two children and an elderly man (about 70 years) had expired due to dysentery.

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A camp for transients about 450 km south east of Vellore was reported to be having unusual number of cases of diarrhoea. This camp was on the sea shore and transients would arrive there once or twice a week and stay for periods up to three weeks before they went away. A field visit in May 1977 showed that there were about 900 inmates in the camp on that day and sixty one cases of acute diarrhoea were identified by a rapid survey. The clinical picture was not homogenous with only about half the patients having bloody mucoid diarrhoea with abdominal cramps and fever. The stools were watery, pea soup like or mushy in the rest.

Piped water was available for the inmates of the camp but the cast iron pipes for the water supply and the earthenware pipes for the water carriage sewage system laid about 60 years earlier, were side by side in porous sandy soil and showed considerable effects of corrosion. Water sampled from an overhead tank and four distribution taps was found to be heavily faecally
contaminated by the presumptive coliform count technique.

_Shigella dysenteriae_ I was cultured from the stool of 6 and _Shigella flexneri_ I from 3 of 28 patients. **1977-78 Kil**

In Kil, an agricultural village about 25 km south of Vellore, 462 of 1028 people developed episodes of acute diarrhoea between April 1977 and February 1978 (Fig. 2). The epidemic was in 2 waves with large number of new cases in July and November. The overall attack rate (44.8 per hundred) was high, with the highest attack rate in pre-school children and decreasing attack rate with increasing age (Table 2). This pattern was the same in both waves of the epidemic. Among those who first developed diarrhoea during June to August, 86 again developed a further, usually short, episode during October to December (Fig. 2). Bloody mucoid diarrhoea typical of bacillary dysentery was complained of by only 42% of those affected in July but in November 85% gave such a history. Other patients had watery or mushy stools. A history of fever was given by nearly half of all affected patients. The mean duration of diarrhoea (12 days) and median duration (8 days) was similar in both waves of the epidemic. Patients who developed a second episode of diarrhoea in November also had similar duration.
Stools were obtained from 65 patients in July and 38 in November. In July several organisms were isolated, Shigella dysenteriae I (3), Shigella flexneri 2 (6) and Salmonella group B (2). In November, Shigella dysenteriae I alone was isolated from 8 of 38 stool samples from patients. Two of 37 control stools contained Shigella flexneri 3 and Shigella boydii 9.

21 patients affected in the epidemic died due to the illness between June 77 and February 78. Of the 331 affected during June to August 77, there were 6 deaths, a case fatality rate of 1.8 per hundred. The number of deaths and case fatality rates were much higher during October to February. Five of the 86 patients who developed symptoms again during this period died (case fatality rate 5.8 per hundred). Among those who developed their illness for the first time during this period there were 10 deaths (case fatality rate 8.5 per hundred). A field clinic in the village provided symptomatic therapy and the more sick were taken to the base hospital when they agreed to hospitalisation. The antibiotic resistance pattern of the organism was known only by the end of November and subsequently clinically severe patients were given neomycin in appropriate doses. However,
the major difficulty in this village was the failure

to make the people accept the necessity of main-

taining nutrition and fluid intake as considerable resis-

tance was offered by several indigenous practitioners in the village. The change in etiology of hospitalized cases with

The village obtained drinking water from a well from which water was pumped to an overhead tank and distributed through a system of street taps. Water supply was intermittent and generally available only in the mornings. Water sampled at four of the distribution taps was heavily faecally contaminated in July. The well from which the water was pumped was chlorinated and chlorination maintained till end of August/November during the second wave of the epidemic water samples again grew abundant faecal coliforms and the well was rechlorinated. No pathogenic bacteria could be detected in the water on either occasion.

**DISCUSSION**

The four epidemics described here and other reports clearly establish that there was widespread *Shigella dysenteriae* type I infection in southern India during the 1970's. The *Shiga bacillus* was considered a rare pathogen in southern India accounting for less than 1% of enteric pathogen isolations.
However, during the 1970's a number of cases of children with an acute dysenteric illness with major complications were seen in hospitals in this region. Noteworthy among the complications was the haemolytic uraemic syndrome (1). Coincident with the change in etiology of hospitalized cases with dysentery the epidemics described here and elsewhere were detected.

The high rate of isolation of multidrug resistant strains of Shigella dysenteriae type I in all the four epidemics described here, from patients but not controls, suggests that this organism was the major enteric pathogen causing these epidemics. In Vad and Kil during November, the epidemic was exclusively due to this organism as shown by the isolation from nearly 1/3 of the cases sampled with very few other pathogens isolated. The situation in Dpm and Kil in July is different with a variety of other enteric pathogens also isolated from the population. In these two epidemics the Shiga bacillus was one among several enteric pathogens presumably spread by faecal contamination of the water supply. Since only a small number of hospitalised patients with recent onset were sampled in Kan, it is difficult to decide the role of other pathogens in this large epidemic. The biotypes and antibiotic sensitivity patterns of all the isolates from these, as well as other
epidemics, suggest that it was likely that a single strain was responsible for these epidemics. No information is available regarding the characteristics of isolates from endemic cases studied in various hospitals during this period.

The relationship of three of these epidemics to sources of water is significant. In Vad and Kil, adequate chlorination of drinking water supply apparently terminated the epidemic with recrudescence in Kil when chlorination was discontinued. At DPM the water distribution system was old and heavily faecally contaminated and was relaid shortly after the studies described here, with a marked reduction in the number of cases of gastroenteritis. The water distribution system in Kil was newly constructed, but, as at DPM, there was no arrangement for regular chlorination to ensure adequate quality. It is important to emphasise that the mere provision of an overhead tank and a pipe system for distribution of water may only create a vehicle for the occurrence of water borne common source epidemics of diarrhoea. Education of the villagers on the proper maintenance of the water supply is an absolute requisite which, unfortunately in many rural areas, is not fulfilled.

The data reported here is reminiscent of the large pandemics due to multidrug resistant Shigella Shiga
reported from Central America and Bangladesh (7-9). These pandemics were associated with a high mortality which at least in Central America was in part ascribed to wrong therapy for Amoebiasis with Emetine, a potentially toxic drug (8). In the first epidemic due to multi drug resistant shiga bacillus detected in southern India (3), the mortality was very low (case fatality rate less than 2 per hundred) and it was felt that the maintenance of nutrition and hydration, without the use of antibiotics in all but a few cases, was a successful field approach to this problem. In Vad this technique was very successful with no deaths occurring after the field team started its work and chlorination successfully terminated the epidemic. The experience in Kil was different. The village was more than thrice the size of Vad and the mortality especially during that part of the epidemic which could be ascribed to Shiga bacillus was quite high. A possible explanation for this phenomenon is that the virulence of the organism had increased. However, there was considerable resistance in the village to accepting the necessity for maintenance of nutrient and fluid intake. The traditional regime for all diarrhoea in this area is starvation and fluid deprivation. The attempts to alter this tradition
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REFERENCES


Table 1. Rate of isolation of enteric bacteria

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<th>Patients</th>
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<th>Controls</th>
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<tr>
<td></td>
<td>No.</td>
<td>Shi. dys.</td>
<td>Other</td>
<td>No.</td>
</tr>
<tr>
<td>1973</td>
<td>Kan 16</td>
<td>8</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>1976</td>
<td>Vad 23</td>
<td>7</td>
<td>-</td>
<td>7</td>
</tr>
<tr>
<td>1977</td>
<td>Dpm 28</td>
<td>6</td>
<td>3</td>
<td>28</td>
</tr>
<tr>
<td>1977</td>
<td>Kil J* 76</td>
<td>3</td>
<td>9</td>
<td>37*</td>
</tr>
<tr>
<td>1977</td>
<td>Kil N* 27</td>
<td>8</td>
<td>2</td>
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The isolation of Shigella dysenteriae I and other enteropathogenic organisms in the four epidemics. * J July 1977, N November 1977 date on controls for N are pooled for both the periods.
Figure 1. Epidemic curve of VAD showing number of new cases for week from 21 July 1976.

Table 2. Attack rates in VAD and Kil.

<table>
<thead>
<tr>
<th>Age group</th>
<th>Rate per hundred population</th>
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<tr>
<td></td>
<td>1976 VAD</td>
</tr>
<tr>
<td>0 - 4.9 years</td>
<td>27.1</td>
</tr>
<tr>
<td>5 - 11.9 years</td>
<td>25.8</td>
</tr>
<tr>
<td>12 - 17.9 years</td>
<td>4.8</td>
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<td>18 +</td>
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new cases occurring for both from start of July and to November. The chart represents patients who first developed symptoms during June to July who subseqently developed symptoms in October or December.
LEGEND TO FIGURES

Figure 1. Epidemic curve of Vad showing number of new cases for week from 21 July 1976. Arrow indicates time of chlorination of all drinking water sources.

Figure 2. Epidemic curve for KIl showing number of new cases occurring per month from March 1977. The drinking water was chlorinated at end of July and in November. The shaded bars represent patients who first developed symptoms during June to July who subsequently developed dysentery in October to December.

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