Epidemic Shigellosis in a Rural Area

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IN the 19 months from May 1957 through November 1958, three epidemics of gastroenteritis in a small city-county area in northern Virginia, with a total population of 27,000, were shown to have been caused by *Shigella*. In two of these outbreaks the organism was transmitted from person to person, while in the third it was foodborne. Shigellosis in epidemic form is unusual, and only 94 outbreaks were reported to the Public Health Service from 1951 through 1958 (*1*).

Foodborne Outbreak

One outbreak of shigellosis occurred in a women's college in the city in May 1957, at the end of the school year. Investigation was hampered by the rapid and wide dispersal of the students, but the 79 known patients were given questionnaires, and 75 of the students returned them, although several were incomplete. Questionnaires were also completed by 94 of 100 faculty members.

The time of onset of symptoms in 46 cases is shown in figure 1. The first symptoms were noted at 9 a.m. on May 23, and the last patient became ill at 11 a.m. on May 26, a range of 74 hours with a median at 11:30 p.m. on May 23. The four cases on May 25 and 26 may well have been secondary.

In most of the cases reported on in the questionnaires, the disease began with a headache, generally accompanied by a chill. These symptoms were followed in a few hours by diarrhea, most frequently with abdominal pain, and generalized aching (table 1). Blood was noted in the stools of two patients. Maximum oral temperatures ranged from 98.4° F. to 104.2° F., with a median of 101.8° F., and most patients were afebrile within 48 hours. Prostration was marked, but only a few patients were in the college infirmary for more than 2 days. Three patients were readmitted, probably because they had been released prematurely to take their final examinations.

It is questionable whether the attack rate of 5.3 percent reflects the true incidence of disease among the 1,502 students, as random questioning of other students and college officials revealed that many students who were ill did not report to the college infirmary. Fecal specimens obtained from 13 patients yielded 9 cultures containing *Shigella flexneri*.

The distribution of cases pointed to a single common source. Contamination of water was ruled out by negative results of laboratory tests and absence of infection in faculty members, most of whom drank water daily on the campus. Sewage disposal facilities were adequate and in good repair. Milk was excluded because no cases of shigellosis occurred in other customers of the dairy supplying the college. Fly control was good. These findings eliminated every known possibility except a foodborne infection.

Inasmuch as 71 patients shared no common eating place outside the campus, attention was directed to the two eating facilities on the campus. These were the restaurant in the student activities building and the college dining hall. The restaurant was absolved when it was learned that none of the faculty members who ate there regularly was stricken and that seven patients had not eaten there since May 19. These seven patients stated that they had eaten only in the dining hall since that date. The

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investigation was therefore centered on the dining hall.

Generally, sanitation in the kitchen was good. A specimen of only one food, chicken à la king served on May 22, was available, and it yielded negative results. Accurate histories of gastrointestinal symptoms were difficult to obtain from kitchen personnel, although one cook's helper stated that he had had diarrhea on May 5, and three kitchen workers said that they had had diarrhea during the outbreak. All fecal samples and followup rectal swabs from the food handlers were negative. The source case was not discovered, the only pertinent history coming from the cook's helper.

One other finding may be relevant. The head student waitress had acute diarrhea on May 20 and spent half of that day in the college infirmary, where she was treated with kaopectate and aspirin. However, she served in the dining hall that day and the next. Her symptoms became more severe on May 23, and a fecal specimen on June 1 was positive for *S. flexneri.* As head waitress this girl could have contaminated a small portion of the food served, thus accounting for the relatively low attack rates.

Adequate followup was impossible because the outbreak coincided with the departure of the students for their homes. However, a letter sent by the college physician to each known patient advised the student to be examined by her family physician. Fecal specimens were collected from all known patients who returned to college in the fall and examined by the State health department laboratory. One of these specimens was positive for *S. flexneri.*

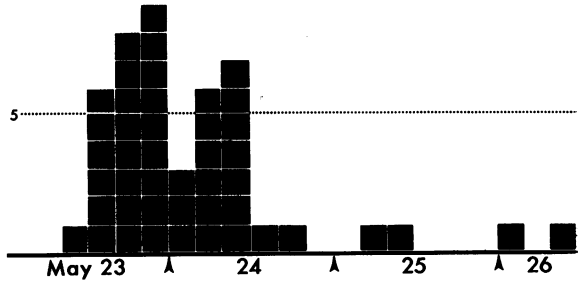
It was suggested that in the future fecal specimens be submitted routinely from all patients with diarrhea treated at the college infirmary. Reporting of illness of all food handlers, including student waitresses, was also recommended.

Table 1. Symptoms manifested by 72 patients in an outbreak of shigellosis at a women's college in Virginia, May 1957

Symptom	Number	Percent
Fever	71	98. 6
Diarrhea	70	97. 2
Headache	68	94. 4
Abdominal pain	61	84. 7
Chill	58	80. 6
Vomiting	40	55. 6
Generalized aching	36	50. 0

Figure 1.

College outbreak of shigellosis, May 1957



Onset by 4-hour intervals

Person-to-Person Outbreaks

The first two cases in a rural outbreak in June 1957 were reported by a private physician after the patients were admitted to the hospital. An interview with the patients' family revealed that other members of that family (X) and two members of a neighboring family (Y) had similar symptoms at that time. A case probably related occurred in a third family (Z) living nearby, it was learned at a later date. The first onset of symptoms was noted by a member of family X on June 10, and the last onset by a member of family Z on June 27 (fig. 2). Symptoms were comparable to those experienced by patients in the college outbreak. Three fecal specimens positive for *Shigella sonnei* were obtained, two from family X and one from family Y.

Each family obtained its water supply from a shallow well which had inadequate physical protection. Sewage disposal facilities consisted of pit privies in reasonable repair. Flies were abundant in the privies and in the houses. Raw milk was obtained from the farm on which members of the three families worked, but no cases of shigellosis were reported in other families who used this raw milk supply. Other milk products were obtained from approved sources and no food which might have been the cause was disclosed.

There was no common source to explain this outbreak. The probable route of transmission was from family X to family Y by direct con-

tact aided by flies, and thence to family Z by flies.

The members of the families who worked in the dairy barn were kept from work until they each had three negative fecal specimens. One member of family X who worked in a cooky factory was not permitted to handle cookies until she had three negative fecal specimens. Other control measures were aimed at prevention of spread through the raw milk supply and in the schools, and general improvement in sanitation was urged. Boiling of raw milk used by the dairy workers was suggested. The children of the affected families were kept out of school until fecal specimens were examined. Privies were repaired as required by local ordinances, and recommendations were made for protection of the water supply.

Another rural outbreak occurred in 1958 and affected four families. Geographically and epidemiologically no connection was evident between this and the previous epidemics. The first case reported was in a 6-year-old boy who was kept out of school on November 11 with diarrhea. Subsequently, diarrhea developed in six other members of this child's family, and investigation revealed that members of three other families in the neighborhood also had diarrhea (fig. 3). *S. sonnei* was recovered from two members of one of these families and from six members of the first family. Symptoms were those of classic shigellosis; in six of the eight confirmed cases the patients had blood in their stools.

As in the previous rural outbreak, water was obtained from unprotected shallow wells. The pit privies used by the families were in poor repair, and flies were noted in large numbers in the houses and in the surrounding areas. Milk and milk products were obtained from ap-

Figure 2.

Rural outbreak of shigellosis, June 1957

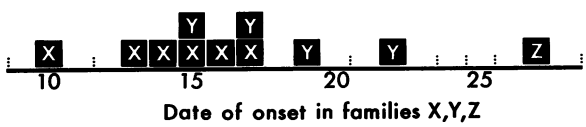
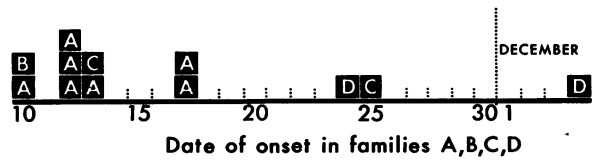


Figure 3.

Rural outbreak of shigellosis, November 1958



proved sources. No food history relevant to the outbreak was elicited.

The source of this outbreak was not revealed, but one possibility may be noted. A child who had been absent from school for 5 days with diarrhea and fever returned to school 2 days before the first child in family A developed dysentery. These children were in the same grade. No fecal specimens were obtained from the first of these children, and no other cases occurred in the school.

As in the rural outbreak which occurred in June 1957, control measures were aimed at preventing spread in the schools and in the local population. No members of any of these families worked with food or milk.

Discussion

These outbreaks indicate how easily an endemic reservoir of shigellosis may assume epidemic proportions. Studies made in Henrico County, Va., in 1931 by McGinnes and associates showed that a high percentage of rural families had shigellae in their stools (*2*). A similar survey by Watt and Hardy demonstrated that the problem was more marked in rural areas than in cities (*3*). Thus it is evident that endemic shigellosis is a problem of rural areas.

The endemic focus which produces localized person-to-person outbreaks may readily explode into a foodborne, milkborne, or waterborne epidemic. Despite modern therapy it is very likely that many patients with diarrhea retain *Shigella* in their intestines for a considerable time. As very few patients or contacts have fecal specimens examined, the true incidence of shigellosis, whether apparent or not, is difficult to ascertain. It is believed that further studies in an endemic-epidemic rural area would be beneficial.

Table 2. Reported number of cases and deaths for shigellosis and typhoid fever, United States, 1955–58

Year	Shigellosis			Typhoid fever		
	Cases	Deaths	Case fatality rate (percent)	Cases	Deaths	Case fatality rate (percent)
1955	13, 912	187	1. 3	1, 704	34	2. 0
1956	10, 306	156	1. 5	1, 700	54	3. 2
1957	9, 822	156	1. 6	1, 231	34	2. 8
1958	11, 861	157	1. 3	1, 043	23	2. 2

SOURCE: National Office of Vital Statistics, Public Health Service.

Shigellosis cannot be viewed with equanimity. It is serious and sometimes fatal in infancy, causing more deaths annually than typhoid fever (table 2). The downward trend evident in typhoid fever is not occurring in shigellosis. Detailed instructions are still issued on the followup of typhoid cases by health departments which almost completely ignore shigellosis. This emphasis, of course, reflects the traditional attitude toward the investigation of enteric disease.

Use of the public health laboratory by practicing physicians should be encouraged and local health departments should be alerted to follow up cases of shigellosis, particularly when infants may be exposed or a member of an infected family works with food, milk, or water. The third outbreak described in this paper was brought to light because an observant public health nurse had been indoctrinated in this way of thinking. Investigation of cases of shigellosis need not occupy much of a health department's time, and it is suggested that this should be included in the overall program of disease prevention.

The basic approach to the problem in rural areas is to determine the endemic foci as shown in a study in Arizona (*4*). Until these endemic foci are determined, intelligent control can not be established (*5*). Knowledge of endemic conditions will prevent spread not only in local person-to-person outbreaks, but also in explosive common-source epidemics. The ecology of shigellosis is still largely a mystery, and an understanding of the geographic incidence of the disease would be an excellent starting point from which to move toward complete control.

Summary

Three epidemics of shigellosis, two person to person and one foodborne, occurred in a small city-county area in Virginia. These outbreaks indicate how easily the endemic reservoir of shigellosis may assume epidemic proportions. It is believed that further studies in an endemic-epidemic rural area would be beneficial.

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