Epidemiologic Investigation of a *Yersinia* Camp Outbreak Linked to a Food Handler

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Abstract: In July 1981, an outbreak of gastroenteritis occurred at a summer diet camp. Of the 455 campers and staff, 35 per cent developed an illness characterized by abdominal pain, fever, diarrhea, and/or nausea and vomiting. A total of 53 per cent experienced abdominal pain. Seven persons were hospitalized, five of whom had appendectomies. *Yersinia enterocolitica* serogroup 0:8 was isolated from 37 (54 per cent) of 69 persons examined, including the camp cook and three assistants. An epidemiologic investigation demonstrated that illness was associated with consumption of reconstituted powdered milk and/or chow mein. *Y. enterocolitica* serogroup 0:8 was subsequently isolated from milk, the milk dispenser, and leftover chow mein. Information obtained during the investigation suggested that the *Yersinia* had been introduced by a food handler during food-processing procedures. (*Am J Public Health* 1984; 74:589–592.)

Introduction

Human illnesses caused by *Yersinia enterocolitica* have been reported with increasing frequency[1] since the organism was first isolated and described in New York State in the 1930s.[2,3] Most cases are sporadic or occur in small clusters, but large outbreaks have been reported worldwide in families,[4] schools,[5–7] a hospital,[8] and in association with community gatherings.[9]

Although *Yersinia enterocolitica* has been isolated from a number of environmental, food, and water sources,[1,10,11] there have been relatively few documented outbreaks of human illness where food was proved by culture to be the source of infection.[1,7,9,11,12] In the three well-documented outbreaks, contaminated chocolate milk, raw milk, and tofu were the vehicles of transmission.[7,9,12] Pasteurized milk was implicated epidemiologically in another outbreak.[13]

The role of a foodhandler as a source of *Yersinia* contamination is less conclusive. The only previous report implicating a foodhandler in an outbreak did so on the basis of an employee's high *Yersinia* antibody titer.[14] No other evidence supported this conclusion, as the employee was culture-negative and the outbreak had neither an epidemiologically associated nor a culture-proven food source. Other outbreak reports have suggested a food handler as a vehicle on the basis of even less documentation.[1,7,11]

The present epidemiologic investigation of an outbreak at a summer camp further documents foodborne *Yersinia* transmission and provides additional evidence for a potential role of food handlers in dissemination of this disease.

Background

The outbreak took place at a rural summer diet camp located in the Catskill Mountains of New York State. The camp population consisted of 327 campers, aged 9–18 years, and 128 counselors and staff members. All were housed in groups of 8–15 in separate buildings, each with its own bathroom and shower facilities. The camp had its own sewage system and was on a public water supply. A pet dog was the only on-site animal. Except for horseback riding, no off-site activities had been held prior to the outbreak.

All food was bought commercially or from local markets and prepared on-site. Consumption of off-camp food was prohibited. All camp personnel ate the same food. A full-time physician and three nurses provided medical coverage, which included a 14-bed infirmary and backup care at a local hospital. The camp session ran from June 27 through August 17, and most campers stayed for the entire season.

Methods

The investigation began on July 12, 1981, after reports were received of a large outbreak of gastrointestinal illness. On the basis of responses to a questionnaire, a case of yersinia-like illness was defined as any person who had developed symptoms of either abdominal pain and fever or abdominal pain and two other gastrointestinal symptoms (e.g., nausea, vomiting, diarrhea). One-third of the cases were then randomly selected for further questioning, along with an equal number of non-ill control persons matched for sex, cabin, age, and camp position. Chi square and McNemar tests were used to analyze the screening and matched pair questionnaires, respectively.

Environmental specimens collected on July 14 included multiple samples of water from various drinking sources, a local stream, and the swimming pool, multiple food specimens, and stool specimens from the pet dog. Fecal specimens were collected from all kitchen personnel, ill persons as they entered the infirmary, and from a small number of well persons. Acute- and convalescent-phase sera were obtained from a limited number of ill persons. Laboratory testing procedures are described elsewhere.[15,16]

Results

Epidemiologic Findings

A review of the camp infirmary log from June 27 showed three peaks from July 5 to 9 in the number of infirmary visits, the number of visits with abdominal pain as the chief complaint, and reports of abdominal pain in association with fever ≥ 101°F. The first recorded infirmary case of fever in association with abdominal pain occurred on July 2 when a counselor was admitted to a nearby hospital and had his appendix removed. The last person with symptoms was seen on July 19.

Of the 455 camp persons who responded to the initial questionnaire, 239 (53 per cent) had experienced an illness

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TABLE 1—Yersinia-like Illnesses by Age, Sex, and Camp Position

Subject Group	No.	Cases		(No. with Abdominal Pain)
		No.	Attack Rate (%)	
Age (yrs)				
8–14	242	106	44*	(153)
15–21	177	50	28	(78)
22+	36	3	8	(8)
Sex				
Male	148	55	37	(91)
Female	307	104	34	(148)
Camp Position				
Camper	327	129	39**	(201)
Counselor	101	28	28	(33)
Staff	16	1	6	(1)
Kitchen	11	1	9	(4)
Totals	455	159	35	(239) (53%)

*$X^2 = 23.06$ $P < .001$
**$X^2 = 14.27$ $P < .01$

which included abdominal pain since arriving at camp, and 159 (35 per cent) met the strict case definition (Table 1). The epidemic curve, showing cases of yersinia-like illness by date of onset, revealed a dramatic peak of illness beginning on July 4 and extending through July 10 (Figure 1). This paralleled the increase in infirmary visits.

Case attack rates (Table 1) did not differ significantly by sex, but were higher for campers and counselors than for other personnel. Younger age groups had significantly higher attack rates, but cases occurred in all groups. Wide fluctuations in cases and case rates by cabin were noted, but 90 per cent of cabins had at least one case.

Of the 53 matched pairs originally selected for the case-control study, 40 complete pairs (75 per cent) participated (52 females and 28 males; 74 campers and 6 counselors). For cases, the most common symptoms were abdominal pain (100 per cent), headache (78 per cent), fever (73 per cent), and nausea (63 per cent). A comparison of exposure to potential risk factors showed no differences between cases and controls in contacts with pets, horseback riding, pool use, hiking or off-camp creek exposure, or consumption of water, soda, chocolate milk, or water from various on- and off-site areas.

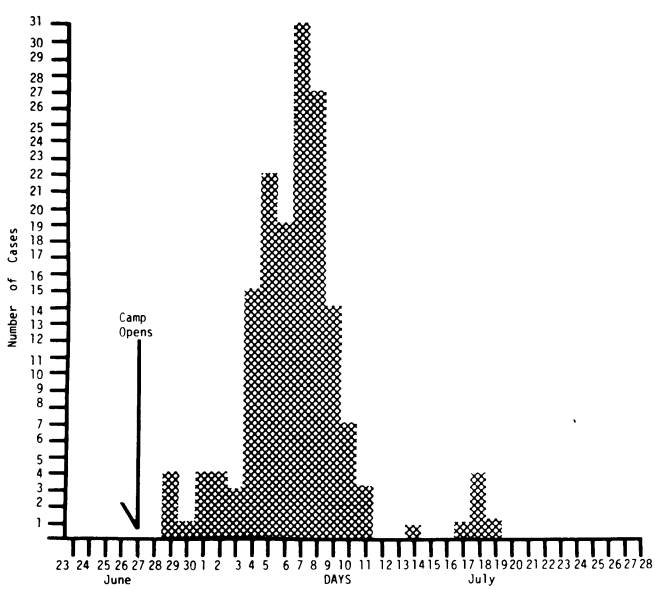

FIGURE 1—Epidemic Curve, Cases of Yersinia-like Illness by Date of Onset

Analyses of 42 food items served on the three days prior to the outbreak, showed three food items served on July 1 and 2 to be associated with illness: powdered milk (reconstituted on-site), commercially prepared cottage cheese, and turkey chow mein prepared by the kitchen staff (Table 2).

Extensive inspections of the water and sewage systems revealed no significant problems. A review of health histories of kitchen personnel showed none had been ill before arriving at camp, but three had developed transient gastrointestinal symptoms during the outbreak. One employee had experienced the earliest symptoms of abdominal pain on June 29 and 30 prior to the outbreak. This initial suspect index case and the head cook prepared the chow mein and were the only ones who prepared reconstituted powdered milk.

Hand contact with milk was minimal during preparation, although some contact could have occurred while scooping out the powdered milk. However, a review of food handling procedures after milk had been implicated showed that spigots on the six-gallon storage containers sometimes malfunctioned, so that milk poured out. On at least three occasions, the head cook put his hand and arm into a partly filled container to plug the hole while the spigot was replaced. The milk may not have been thrown out. Also, the milk was prepared a day in advance and empty containers were cleaned only with cold or warm water. The spigots were not cleaned separately and when disassembled were found to contain accumulated milk powder residue.

Laboratory Data

All 16 drinking water specimens were negative for bacterial contamination. Three *Y. enterocolitica* serogroup 0:8 isolations were made from 29 food specimens: from a powdered milk suspension, from leftover frozen turkey chow mein, and from a swab of the top rim of a milk storage container. Samples of the unmixed powdered milk and raw products were negative. Cottage cheese was unavailable for testing.

Thirty-four of 66 persons submitting stool specimens had positive cultures for *Y. enterocolitica* serogroup 0:8.* The three original hospital *Yersinia* isolates (one fecal, two intraabdominal) were also confirmed as *Y. enterocolitica* serogroup 0:8. The positive isolates were obtained from 24 cases, seven noncases with gastrointestinal symptoms, and six asymptomatic persons. Overall, 69 per cent of cases, 64 per cent of non-cases with abdominal pain, and 26 per cent of asymptomatic persons cultured were positive for *Y. enterocolitica* serogroup 0:8.**

Four kitchen workers had positive stool cultures for *Y. enterocolitica* serogroup 0:8. One of these was the head cook whose stool yielded positive cultures without cold enrichment on three occasions over a five-week period. Stool from the initial suspect index case was positive only for *Y. enterocolitica* serogroup 0:34.

Sera were obtained from 23 persons, but only 10 were

*Nonpathogenic *Yersinia* isolates were obtained from specimens as follows: *Y. intermedia* from celery, *Y. enterocolitica* serogroup 0:4,33 from a dog fecal specimen, and *Y. enterocolitica* serogroup 0:34 from two milk suspensions and six human stool samples.

**Only 44 per cent of isolates were obtained on direct plating; 39 per cent became positive after one week and 17 per cent after three weeks of cold enrichment.

TABLE 2—Food Consumption Associated with *Yersinia-like* Illness, Cases vs Controls

Food	Consumption		Cases (n=40)			Controls (n=40)			Significance*	
	Date	Time	No. Ate	No. Didn't Eat	% Ate	No. Ate	No. Didn't Eat	% Ate	X^2	P
Milk	7/1	AM	31	9	78	21	19	53	4.55	<.05
		Noon	29	11	73	16	24	40	7.35	<.01
		Both	27	13	68	15	25	38	6.00	<.02
Chow Mein	7/1	PM	36	4	90	29	11	73	4.45	<.05
Cottage Cheese	7/2	Noon	29	11	73	14	26	35	10.71	<.01

*by McNemar Test

represented by both acute- and convalescent-phase specimens. Initial serologic testing with banked antigenic material showed no evidence of *Yersinia* infection. However, subsequent testing for *Y. enterocolitica* serogroup 0:8 with antigens prepared from outbreak *Yersinia* isolates showed five confirmed (fourfold titer change or single titer ≥ 256), nine borderline (titer ≥ 128), and nine nondiagnostic results. Paired sera from the head cook gave results consistent with previous *Yersinia* exposure (titers of 1:64, 1:64 ± 128); those from the initial suspect index case were negative.

Hospital Case Review

Of seven hospitalized persons (five male, two female, ages 10–18), all presented with fever (highest 102–104°F) and abdominal pain; three had experienced nausea, two vomiting, and two diarrhea. Most had localizing symptoms of right-lower-quadrant abdominal pain, with various degrees of rebound tenderness and guarding. All had elevated WBC counts (11,800 to 17,900). One stool and two appendiceal swab cultures were positive for *Yersinia enterocolitica* serogroup 0:8, but these cultures were identified as *Yersinia* only after the five appendectomies. Other cultures were negative. For the patients who had appendectomies, pathologic findings showed that one had acute suppurative appendicitis, two had microscopic inflammatory changes, and two were normal. A review of hospital emergency room and appendectomy records showed the outbreak to be limited to the camp.

Discussion

Since 1933, when human illness caused by *Y. enterocolitica* was first described, through 1976, when the largest documented *Y. enterocolitica* foodborne outbreak occurred, New York State has been a noted site for *Yersinia* activity.[2,7,10] In this 1981 *Yersinia* foodborne outbreak, consumption of reconstituted powdered milk and chow mein was epidemiologically associated with gastrointestinal illness. Cottage cheese was felt to be a less likely vehicle, because of its rapid turnover time, minimal exposure to hand contact, lower frequency of use, and lack of association with illness when used by other community groups. *Y. enterocolitica* serogroup 0:8 was isolated from stool specimens of several ill persons and from reconstituted milk, the milk dispenser, and leftover chow mein. All 0:8 isolates were identical by serogroup and biogroup testing.[15]

The epidemiologic investigation which documented the foodborne nature of this outbreak also suggested that the food was contaminated by a foodhandler. Although the four culture-positive foodhandlers may have only been victims of the outbreak, there is circumstantial evidence to suggest that one of them was its source. This is supported by the culture proven contamination of more than one food item made with separate ingredients and the observation of poor food handling practices. The head cook, while reporting no symptoms, had stool cultures positive for *Yersinia* without cold enrichment over five weeks of observation, supportive of a high rate of shedding. He was the only foodhandler intimately involved in preparing all the culture-positive food items made from culture-negative ingredients, and his milk-handling procedures would have allowed for direct inoculation of bacteria. Serologic testing while not diagnostic for an acute *Yersinia* infection was consistent with previous exposure. His involvement provides further evidence for a potential role of kitchen personnel in *Yersinia* transmission.

Foodhandlers should not prepare meals when ill, but symptoms in the kitchen personnel involved here were mild enough to go unnoticed and unreported. The outbreak emphasizes the need for foodhandlers in general to maintain strict personal hygiene, to discontinue work immediately when ill, and to avoid practices which may lead to direct contamination of food.

Like other recent outbreaks, milk was again implicated as a vehicle of *Yersinia* transmission.[7,9,13] It may have been contaminated during mixing or during repair of a broken spigot on a storage container. Once contaminated, it may have remained so for several days because of cleaning procedures insufficient to kill the organism, as evident by *Yersinia* isolation from the container rim weeks after the start of the outbreak. Unlike other organisms, the ability of *Yersinia* to grow in the cold,[11] in conjunction with the camp's practice of holding milk 24 hours before use, could have provided an excellent medium for growth.

As in other episodes of yersiniosis among school-age children,[7,17] abdominal pain and fever were the predominant symptoms. Illness rates were higher in younger age groups, a finding consistent with other reports.[1,4,18] Persons over age 22 were relatively unaffected, despite having the same food exposures and occasional positive stool cultures. This suggests a difference in susceptibility by age, but we cannot rule out differences in quantitative milk consumption. Several persons presented with signs, symptoms, and laboratory findings consistent with acute appendicitis and, although most postoperative pathologic findings were grossly normal, it would have been extremely difficult to predict this outcome.

Unlike Canada and Northern Europe, where yersiniosis is most commonly caused by *Y. enterocolitica* serogroups 0:3 and 0:9,[7,17,18] the United States has a variety of serogroups, with 0:8 predominating in outbreaks, as occurred

here.[1,7,10,16] We found that special media and cold enrichment techniques were useful in increasing the chances of isolating *Yersinia* from stool cultures. Unfortunately, as in this outbreak, such methods are too slow to be of help in the differential diagnosis of acute abdominal pain or in rapidly identifying the source or cause of an outbreak. Serogrouping and biogrouping of isolates helped us separate pathogenic from nonpathogenic isolates and to conclusively link human with environmental isolates. Serogroup 0:8 is a human pathogen, while 0:34 is not. However, the fact that 0:8 and 0:34 have identical biogroup patterns, together with the isolation of 0:34 from two environmental and six human specimens including the initial suspect index case, raises the question of possible biotransformation. This observation seems worthy of further study.

Despite the numerous positive cultures, *Yersinia* infections were not confirmed by the initial serologic testing with stock antigens, including those for the 0:8 serogroup. However, when the sera were tested against antigenic material prepared from cultures of specimens from outbreak cases, 61 per cent of persons tested had confirmatory or borderline titers, despite the limited availability of convalescent-phase specimens. This finding suggests that standard serologic testing with stock antigens for *Yersinia*, until refined, has only limited use for diagnostic purposes.

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