

Waterborne Gastroenteritis due to the Norwalk Agent: Clinical and Epidemiologic Investigation

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Abstract: An outbreak of gastroenteritis occurred at a Pennsylvania summer camp in July 1978. Symptoms included abdominal pain (81 per cent), nausea (72 per cent), and vomiting (53 per cent); upper respiratory infection symptoms occurred in 35 per cent of the campers. Illness was associated with consumption of five or more glasses of water or water-containing beverages. Stool cultures from affected persons were negative for bacterial pathogens; however, a fourfold or greater rise to the Norwalk agent was demonstrated in serum samples of three of three ill persons tested and in none of eight controls ($p < .02$). Campers ill during the first session who were also present during the second session did not become ill during the second session ($p < .001$). (*Am J Public Health* 1982; 72:72-74.)

Introduction

Gastrointestinal illness due to the Norwalk agent is being increasingly recognized, but absence of routine surveillance data makes outbreak investigations the only readily available source of information concerning the clinical and epidemiologic features of infection. We investigated an outbreak of gastroenteritis associated with evidence of infection with Norwalk agent and found that: respiratory symptoms were a prominent feature of the illness; and short-term natural immunity probably exists after infection with this agent.

Background

Monroe County, located in northeastern Pennsylvania, contains numerous summer recreational camps. The camp investigated accepted children of both sexes 6-16 years of

age. They were housed in four camp areas according to age and sex. All campers swam twice daily without exception. All meals were prepared in the camp kitchen and eaten in the mess hall by campers and staff members. The camp had two summer sessions; in 1978, session 1 began June 26 and lasted until July 22, and session 2 began on July 23.

Methods

A camper or staff member who complained of vomiting, nausea, or diarrhea with date of onset June 26-July 22 was considered to have a first-session outbreak-associated case of gastroenteritis. Individuals meeting symptom criteria with date of onset from July 23 or later were considered to have a second-session outbreak-associated case of gastroenteritis.

Public health nurses conducted a telephone survey of neighboring summer camps and health care facilities to determine the number of cases of gastroenteritis reported compared to similar reports from the previous two years. Camp infirmary records from both first and second sessions for the years 1976-1978 were reviewed. On July 31 we administered a questionnaire to all campers and staff members regarding foods eaten in the week prior to the outbreak, amount of water drunk, and symptoms of illness.

Cooking facilities, waste disposal, and the camp water system were evaluated. Records on the quality of the lake water were also reviewed.

Stool cultures for bacterial pathogens including *Salmonella*, *Shigella*, *Escherichia coli*, and *Campylobacter* were obtained from eight ill individuals and 10 controls. Stool specimens for viral studies were obtained from three individuals who had become ill within the preceding 48 hours. Acute- and convalescent-phase blood specimens were obtained from three ill patients and eight controls.

Results

A telephone survey of other summer camps, physicians' offices, and hospital emergency rooms showed no increase in the number of gastroenteritis cases reported for the study period when compared with the number of cases reported for the previous two years. Six to 12 cases of gastroenteritis per session were seen in the camp infirmary during the two years before the outbreak. A review of the records from session 1 (June 26-July 22) revealed 73 cases of gastroenteritis out of 255 persons at risk (attack rate 28.6 per cent). When cases were stratified by residential area, no differences in attack rates were noted (Table 1). Review of infirmary records for

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TABLE 1—Infirmary Visits of Campers Due to Gastroenteritis, Session 1

Residential Area	Sex	Age Group (years)	At Risk	Ill	Attack Rate (%)
The Hill	Girls	10–16	66	19	28.8
The Line	Boys	6–9	59	22	37.3
The Row	Girls	6–9	41	13	31.7
The Village	Boys	10–16	54	12	22.2
Other			35	7	20.0
TOTAL			255	73	28.6

the second camp session revealed a sharp increase in the number of cases on July 25, a peak in the number of cases on July 26, and no new cases reported after July 28 (Figure 1).

A total of 215 (96 per cent response rate) completed questionnaires (distributed July 31 during the second session) were returned. Ten staff members and 10 campers had first-session illness, and these cases were analyzed separately; 195 questionnaires remained for analysis. One hundred twenty (61.5 per cent) of these 195 persons reported illness. The attack rate for the staff and older boys (The Village) was significantly higher ($p < .05$) than that for the other campers (Table 2). Predominant symptoms included abdominal pain (81 per cent), nausea (72 per cent), and vomiting (53 per cent). Headache (47 per cent), diarrhea (38 per cent), and upper respiratory tract symptoms (35 per cent) were also common. When symptoms of campers and staff were compared, abdominal pain and upper respiratory symptoms were reported significantly more frequently by campers (Table 3). The median duration of illness was two days (range one to four days).

There was no statistically significant association between any food eaten and illness. However, a significant association was found between drinking five or more glasses per day of water or a water-containing beverage called "bug juice" and illness (Table 4).

Thirty-six campers were present during both summer sessions. Of these, 10 were ill the first session but not ill the second session; 14 were ill the second session only; and 12 were not ill during either session. Attack rates were 27.8 per cent and 53.9 per cent for the first and second sessions, respectively. These rates are comparable to the rates for first

and second session illness for other campers (Tables 1 and 2). The absence of illness during the second session among campers ill during the first session is statistically significant ($p < .001$, Fisher's exact test two-tailed). Analysis of staff member illness repeated these findings.

Fluorescein dye studied failed to reveal any evidence of cross-contamination in the water system. Analysis of water from the system revealed fecal coliforms in the unchlorinated well water, increased fecal streptococci in kitchen tap water, and increased total coliforms in water from the end of the line (Table 5). Chlorine levels were measured on July 27; there was no residual on the end of the line. After adequate chlorination, chlorine levels of 2.5 ppm were obtained on July 28 and 1.5 ppm on July 29.

No pathogens were found in bacterial stool cultures from persons in the control groups. Three of three paired blood specimens from acutely ill patients compared to zero of eight controls ($p < .02$, Fisher's exact test 2 tailed) showed a fourfold or greater rise to Norwalk agent by radioimmunoassay (Table 6). Stool specimens examined by electron microscopy were negative, as were enteroviral cultures on two stool filtrates.

Discussion

Gastrointestinal illness in campers who attended the second session at camp appears to have been caused by the Norwalk agent. Although stool specimens were negative, serology has been shown to be more sensitive and as specific

TABLE 2—Results of Questionnaire Survey on Illness, Session 2

Residential Area	No. at Risk	No. Ill	Attack Rate (%)
The Hill	37	18	48.7
The Line	48	25	52.1
The Row	29	16	55.1
The Village	44	34	77.3
Staff	27	20	74.1
TOTAL	195	120	61.5

Hill vs Village $X^2 = 5.97$
 $.02 > p > .01$
 Overall attack rate = 61.5%

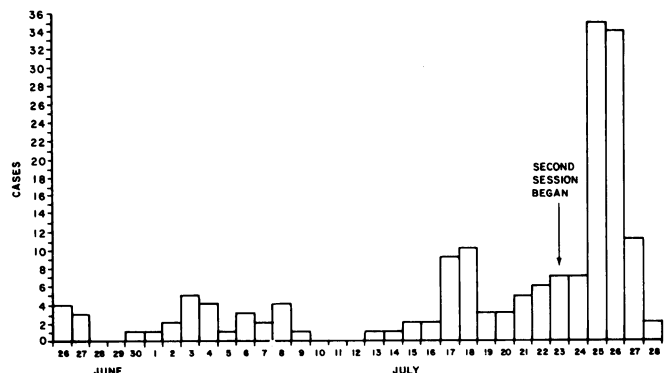
**FIGURE 1—Gastroenteritis Cases, by Date of Patients' Visit to Infirmary, Monroe County, Pennsylvania, June 26–July 28, 1978**

TABLE 3—Comparison of Symptoms in Campers and Staff Members

Symptom	Per Cent of Campers Affected (N=100)	Per Cent of Staff Affected (N=20)
Abdominal pain*	90	35
Nausea	74	65
Vomiting	55	45
Headache	50	30
Chills	37	40
URI symptoms*	42	15
Myalgia	17	25
Rash	2	0

* = $p < .05$ **TABLE 4—Consumption of Water and/or "Bug Juice" Per Day**

Item	No. of Glasses Consumed	No. Ill	No. Not Ill
Water	≥ 5	39	12
	< 5	81	63
"Bug Juice"	≥ 5	43	16
	< 5	77	59
Water and "Bug Juice"	≥ 10	20	2
	< 10	100	73

for identifying the Norwalk agent.¹ A waterborne mode of spread is supported bacteriologically and epidemiologically. An interesting feature of this outbreak is the absence of illness during the second session among persons ill the first session, suggesting that short-term immunity to Norwalk agent does occur naturally. This finding supports observations made in volunteer studies.² This finding underscores the need to obtain both acute and convalescent phase serologic specimens from persons involved in any outbreak suspected to be caused by the Norwalk agent.

The prominence of upper respiratory symptoms in younger children suggests that under certain circumstances respiratory transmission may be likely. Respiratory transmissions of rotavirus has been proposed³ but challenge with nasopharyngeal filtrates from patients with Norwalk agent gastroenteritis failed to produce disease in volunteers.⁴

Further outbreak investigations should center on defining disease spectrum, modes of transmission, and immunity to this increasingly important gastrointestinal pathogen.

TABLE 5—Colliform Counts in Water Samples

Source	Contaminant	Count
Well	Total coliforms	18
	Fecal coliforms	1
	Fecal streptococci	2
Kitchen	Total coliforms	2
	Fecal coliforms	0
	Fecal streptococci	55
Cabin 8 (end of line)	Total coliforms	3
	Fecal coliforms	0

TABLE 6—Antibody Titers to Norwalk Agent by Radioimmunoassay for Ill Patients

Case No.	Titers	
	Acute	Convalescent
1	< 100	400
2	< 25	100
3	100	1600

REFERENCES

- Greenberg HP, Wyatt RG, Voldesuso J, *et al*: Solid phase microtiter radioimmunoassay for detection of the Norwalk strain of acute nonbacterial epidemic gastroenteritis virus and its antibodies. *J Med Virol* 1978; 2:97-108.
- Parrino TA, Schreiber DS, Trier JS, Kapikian AZ, Blacklow NR: Clinical immunity in acute gastroenteritis caused by Norwalk agent. *N Engl J Med* 1977; 297:86-89.
- Foster SO, Palmer EL, Gary GW Jr, Martin ML, Hermann KL, Beasley P, Sampson J: Gastroenteritis due to rotavirus in an isolated Pacific Island group: an epidemic of 3,439 cases. *J Infect Dis* 1980; 141:32-39.
- Dolin R, Blacklow NR, DuPont H, *et al*: Biological properties of Norwalk agent of acute infectious nonbacterial gastroenteritis. *Proc Soc Exp Biol Med* 1972; 140:578-583.

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