Swimming-Associated Cryptosporidiosis

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ABSTRACT

In July and August 1988, an outbreak of gastroenteritis affected 44 of 60 (73%) persons from 5 separate swimming groups who had used the same swimming pool in Los Angeles. *Cryptosporidium* was identified in 5 of 8 (63%) stool specimens, and the clinical picture was consistent with *Cryptosporidium* infection. Resistance of *Cryptosporidium* to chlorine, an inadequately maintained pool filtration system, repeated exposure to pool water, and possible continuing pool contamination may have contributed to ongoing transmission. *Cryptosporidium* should be considered a potential etiologic agent of gastroenteritis associated with recreational water use. (*Am J Public Health.* 1992;82:742–744)

Introduction

Since 1985, *Cryptosporidium* has been increasingly recognized as an important cause of gastroenteritis.[1] Outbreaks of cryptosporidiosis have been reported among day-care center attendees,[2] among travelers,[3,4] and in communities with contaminated water supplies.[5,6] Although outbreaks of enteric infection associated with recreational water contact have been documented,[7–10] however, swimming pool–associated cryptosporidiosis has not been previously reported.

In July and August 1988, an outbreak of diarrheal illness affected members of five separate groups of swimmers using the same private pool (Pool A) in Los Angeles County. The outbreak began within 1 week after a fecal accident in the pool in early July. The five groups, who had repeated pool contact during July, were a high school water polo team, a beginning scuba class, a masters swim group, elementary school students, and the lifeguard staff. The outbreak was not reported to health authorities until early August, when an investigation was initiated.

Methods

A standardized questionnaire was used to obtain information on the basic demographics of the persons involved in the outbreak and on clinical manifestations of and exposure to potential risk factors, including contact with day-care centers, travel outside the United States, consumption of untreated surface water, animal contact, common sources of food or water, and length and type of pool contact. To assess potential risk factors, the questionnaire was administered to a cohort of 32 elementary school students who had used two local pools; 16 who had used Pool A and 16 who had used another pool, Pool B. (Three students with exposure to both pools were not included in the analysis.) In addition, a case-control study was conducted among the remaining four groups. Thirty-six ill swimmers for whom family controls were available were compared with 72 unmatched, well family members. A suspect case was defined as any swimming group member, household contact of a group member, or school classmate with watery diarrhea or diarrhea plus cramping and/or fever from July 1 through August 31. Patients whose stool tested positive for *Cryptosporidium* were considered confirmed cases.

Data from an active surveillance system were reviewed to determine whether there had been a communitywide increase of gastrointestinal illness during July and August.

Laboratory results were obtained from physicians and/or laboratories where stool specimens were examined. Additional specimens were obtained from persons who continued to be ill. Samples were assessed for bacterial (*Salmonella, Shigella, Campylobacter*) and protozoan pathogens by standard methods. Stool specimens, which had been preserved, were obtained from private laboratories and evaluated for *Cryptosporidium* by the Los Angeles County Public Health Laboratories. Auramine O stain was used as an initial screen for *Cryptosporidium,* and a modified acid-fast stain was used as a confirmatory test. Viral studies were not performed.

An assessment of pool maintenance was made; however, attempts to recover *Cryptosporidium* from water were not made because the delay in investigation diminished the likelihood of recovering oocysts.

Chi-square, the Fisher Exact Test, a chi-square test for trend, and the *t* test were used to evaluate association of possible risk factors with illness. Relative risk (RR), odds ratios (ORs), and 95% confidence intervals (CIs) were calculated where appropriate.

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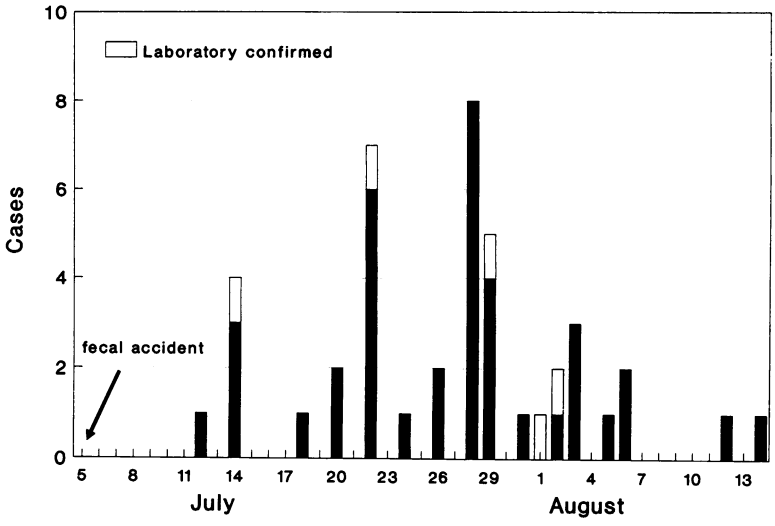

FIGURE 1—Cases of diarrheal illness among persons using Pool A, Los Angeles County, July–August, 1988.

TABLE 1—Age Range and Occurrence of Diarrhea by Group among Swimmers with Exposure to Pool A, Los Angeles County, July and August 1988

Swimming group	Median Age (y) (range)	Total Members	Interviewed (%)[a]	Ill (%)[b]
Scuba class	27 (16–34)	15	13 (87)	13 (100)
Masters group	27 (20–57)	17	13 (76)	11 (85)
Pool lifeguards	18 (14–20)	5	4 (80)	3 (75)
Water polo team	15 (13–16)	15	11 (73)	8 (73)
Elementary school group	6 (5–39)	26	19 (73)	9 (47)
Total	17 (5–57)	78	60 (77)	44 (73)

[a]Percent of total members.
[b]Percent of total interviewed.

TABLE 2—Attack Rate by Hours of Water Exposure to Pool A among Members of Five Swimming Groups, Los Angeles County, July and August 1988

Exposure in Hours[a]	Number[b]	Number Ill	%
1–3	13	5	39
4–6	8	5	63
>6	37	33	89

Note. Chi-square test for trend $P < .001$.
[a]Total hours contact during July.
[b]Two persons with undetermined length of exposure.

Results

Interviews were completed for 60 of the 78 swimming group members (77%). Between July 13 and August 14, 1988, 44 group members developed gastrointestinal illness (Figure 1). The overall attack rate was 73% (44/60) and ranged from 47% to 100% by group. The median age of patients was 17 years (range 5 to 57) (Table 1); the male:female ratio was 1.2:1. Illness was characterized by watery diarrhea (88%), abdominal cramping (86%), and fever (60%), and it was often protracted (median duration 5 days). Thirty-two percent (14/44) sought physician consultation; two patients, both from the scuba class, were hospitalized.

Among the cohort of 32 elementary school students, illness was significantly more common among the 16 using Pool A (Fisher's Exact Test, $P < .01$, RR = 8, 95% CI = 1.1, 56.8). Differences in age or gender between those with and without exposure to Pool A were not noted.

For the remaining four groups, cases (35/36) were more likely than family controls (1/72) to have a history of exposure at Pool A during the outbreak period ($P < .001$, OR = 2485, 95% CI 125,22026).

The attack rate was highest for those having heavy (more than 3 total hours) water exposure (chi-square test for trend, $P < .001$) (Table 2). Cases with fever reported twice the extent of water contact than those without fever (mean hours 23.1 and 12.2, respectively, t test $P = .09$). No other common exposures or risk factors could be identified.

Cryptosporidium was identified in stool specimens from five of eight (63%) patients tested; no other bacterial or parasitic enteric pathogens were found. Stool samples from two control patients were negative for *Cryptosporidium.*

No increase in reported diarrheal illness during July and August in the county or the community affected by the outbreak was observed. Inspection of the pool during the outbreak period revealed adequate chlorine levels (2 ppm). However, one of three diatomaceous earth filters was inoperative, and the total filtration flow rate was 30% below the 230 gallons per minute recommended.

Discussion

Several factors support the conclusion that this was a swimming pool–related outbreak of cryptosporidiosis. The epidemic curve is consistent with a continuing common source outbreak. The observed association between exposure to Pool A and illness, the increasing attack rate and severity of disease with increasing water exposure, and the temporal occurrence of the outbreak following the fecal accident implicate the pool as the most likely source of infection. Identification of *Cryptosporidium* in affected patients and the absence of other parasitic or bacterial enteric pathogens indicate that *Cryptosporidium* was the etiologic agent in this outbreak. The clinical manifestations, together with the protracted nature of the illness, are also consistent with *Cryptosporidium* infections. It is unlikely that *Cryptosporidium* was an incidental finding, given that asymptomatic infection is rare.[11] The organism is infrequently found in stool samples during nonepidemic situations.[12] *Cryptosporidium* was not a reportable disease at the time of this outbreak. Therefore, the background prevalence of *Cryptosporidium* among immunocompetent persons in Los Angeles County was unknown. In addition, the level of chlorination (2 ppm) found during inspection of the pool was sufficient to inactivate other known enteric pathogens.

Resistance of *Cryptosporidium* to chlorination,[13] an inadequately maintained filtration system, repeated and often prolonged exposure, and the apparent low infectious dose of *Cryptosporidium*[14] may have contributed to ongoing transmission in this outbreak. Continued pool use and possible ongoing contamination of the pool by infected persons, many of whom continued to swim despite their illness, also could have helped sustain transmission. The increased attack rate observed for longer water contact may be related to a greater inoculum associated with swallowed water. The scuba class, which presented a high likelihood of swallowing large quantities of water, had the highest attack rate (100%), and the only patients hospitalized were from this group. The elementary school children, who might also be expected to swallow water, had much less water exposure than the scuba group. Because attempts to recover *Cryptosporidium* from the pool water were not made and patients were not tested for viral pathogens, our findings must be interpreted with caution.

Outbreaks of *Giardia intestinalis* infection and adenovirus types 4 and 3 associated with inadequately maintained swimming pools have been reported.[8,15–17] A well-maintained, fine-grade diatomaceous earth filtration system is probably effective in removing the small *Cryptosporidium* (4–6) oocysts.[18] Whether the commonly used rapid sand filters are able to remove oocysts is unknown.

Because chlorine is ineffective against *Cryptosporidium*, closure of a pool until relatively complete filtration has occurred should be considered as an additional measure for pool disinfection following fecal contamination for pools with diatomaceous earth filtration systems. Complete draining of the pool and cleaning of filter media may be necessary for pools with rapid sand filters. Although this outbreak followed a fecal accident, reliable information on the person who defecated in the pool was unavailable, and testing was not possible. Therefore, the causal role of the fecal accident in the occurrence of the outbreak is only suggestive.

Swimming-related cryptosporidiosis may be more common than previously recognized.[19] Public health authorities should have a high index of suspicion for *Cryptosporidium* as a possible etiologic agent during outbreaks of gastroenteritis associated with recreational water contact.

Although typically a self-limited infection in the immunocompetent host,[1] *Cryptosporidium* infection, for which there is no known effective therapy, may cause an unremitting diarrhea with significant morbidity and mortality in the immunocompromised patient.[20] There are an estimated 15 000 public swimming pools and 500 000 in-ground private pools in Los Angeles County. This is also an area of relatively high AIDS incidence and HIV seroprevalence.[21] The convergence of these factors creates the potential for swimming-related exposure of *Cryptosporidium* among highly susceptible persons at high risk of potentially life-threatening illness. □

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