SHORT REPORT
Giardiasis outbreak at a camp after installation of a slow-sand filtration water-treatment system

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SUMMARY

In July and August 2007, a giardiasis outbreak affected attendees of a private recreational camp in California. Twenty-six persons had laboratory-confirmed giardiasis; another 24 had giardiasis-like illness with no stool test. A retrospective cohort study determined that showering was associated with illness (adjusted odds ratio 3·1, 95% confidence interval 1·1–9·3). Two days before the outbreak began, the camp had installed a slow-sand water filtration system that included unsterilized sand. Review of historical water-quality data identified substantially elevated total coliform and turbidity levels in sand-filtered spring water used for showering during the suspected exposure period. Unfiltered spring water tested at the same time had acceptable coliform and turbidity levels, implicating the filtration system as the most likely contamination source. To prevent waterborne illness, slow-sand water filtration systems should use sterilized sand, and slow-sand-filtered water should not be used for any purpose where inadvertent ingestion could occur until testing confirms its potability.

Key words: *Giardia lamblis*, giardiasis, outbreaks, waterborne infections.

Giardiasis, an infection with the protozoon parasite *Giardia intestinalis* (also known as *G. lamblia* or *G. duodenalis*), is one of the most frequently diagnosed enteric parasitic infections in the USA [1], where an estimated 2 000 000 infections occur annually [2]. Transmission is by the faecal–oral route, resulting primarily from ingestion of *Giardia* cysts in contaminated water; however, foodborne and person-to-person transmission also occur. The incubation period ranges from 3 to 25 days, but is usually 1–2 weeks [3]. Symptoms can last 2–4 weeks and include diarrhoea, abdominal cramps, flatulence, greasy stools, nausea, and weight loss [1]. Symptoms, particularly diarrhoea, can be intermittent, and asymptomatic infections occur. Most reported giardiasis cases are sporadic, although outbreaks are occasionally reported [3]. Multiple factors can complicate and delay detection and control of giardiasis outbreaks, including an extended incubation period, low infectious dose [4], moderate chlorine resistance [5], and the ability of *Giardia* cysts to survive for weeks in cold water [6].

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We describe a giardiasis outbreak among attendees of camp A, a boys' scouting camp in central California. Each summer during June–August, camp A holds sessions lasting 4–7 days for scouts and their parents. Children and parents sleep in tents, and meals are prepared by staff in an indoor kitchen. The camp has an artificial lake, created by damming a river and used by scouts, parents, and staff for swimming and boating, and a natural spring from which water is piped to the kitchen, toilets, and showers. Before 2007, camp A also used spring water for drinking. However, in April 2007, camp A was placed on a boil-water order after routine testing by the local health department identified total coliforms (16 c.f.u./100 ml) in the spring water. During the 2007 summer season, imported tanks of potable water were used for cooking, and attendees drank bottled water. Signs were posted in camp A's kitchen and bathrooms stating that a boil-water order was in effect and that persons should not drink tap water or use it in the kitchen for washing hands or any other purpose. On 16 May 2007, a water engineer began installing a slow-sand filtration system and chlorinator for the spring water. On 6 July, physical installation was completed; the filtration system was put into operation; and the local health department began weekly testing of unfiltered and sand-filtered spring water for coliforms and turbidity. The boil-water order remained in effect after the filtration system was installed, but the system's effluent was used in the bathrooms for washing hands and showering.

We identified cases in parents, children, and staff who had attended camp A for at least one session during June–August 2007. We defined a confirmed case as giardiasis diagnosed by detection of *Giardia* cysts in stool samples, and a probable case as >4 days of self-reported diarrhoea (≥3 loose stools during a 24-h period) in a patient with no *Giardia* stool test. Cases with illness onset >7 days after that of another case in the same household were categorized as secondary cases and excluded from analyses.

To identify cases, we mailed letters to all households with members who had attended camp A during June–August 2007, notifying them of the outbreak and asking persons with giardiasis-like symptoms to contact their local health department. To identify confirmed cases, we tested 30 stool samples by using formalin ethyl acetate concentration and trichrome staining. Seven *Giardia*-positive stool samples were re-tested at CDC/DPD laboratory by using direct wet-mount, trichrome staining, and direct immunofluorescent antibody (IFA) assay, and were genotyped by using polymerase chain reaction (PCR) analysis of the triosephosphate isomerase (TPI) gene [7].

To enhance case finding and collect data for the epidemiological investigation, we developed a standardized survey, which was hand-delivered to camp A staff and administered by telephone to parents and scouts who had attended camp A during three consecutive camp sessions on 24–28 June, 5–8 July, or 9–12 July. The survey collected demographic data, clinical data, and data on exposures to recreational and drinking water and foods served at camp A. Using survey data, we conducted a descriptive analysis of all primary cases, and a retrospective cohort study to determine specific risk factors for illness. The cohort study assessed data from the subset of parents and children who attended the 5–8 July session attended by five of the six original patients. The study excluded persons with secondary illness, staff, and persons who reported gastrointestinal symptoms that did not meet the case definition. We used SAS® version 9.1 (SAS Institute Inc., USA) for analyses, and performed bivariate analyses for all variables and multivariable logistic regression analysis for variables identified as being significantly ($P<0.05$) associated with illness in bivariate analyses.

During a site visit on 14 August, we inspected the spring and the slow-sand filtration system, interviewed the water engineer regarding system installation and testing, and reviewed the local health department's records of total coliform, faecal coliform, and turbidity testing of unfiltered and sand-filtered spring water. We collected a 20-l sample of unfiltered spring water and a 500-ml sample of sand from the surface of the filtration system. The water sample was processed by using ultrafiltration and centrifugation [8]. The sand sample was eluted with Laureth-12, and the eluent was then centrifuged to pellet cysts. We also tested about 100 l each of sand-filtered spring water, water from the area of the lake most frequented by swimmers and boaters, and stagnant lake water using Envirochek® filters (Pall Corporation, USA) at a rate of 1–2 l/min. We tested turbidity and total and faecal coliform levels of unfiltered and sand-filtered spring-water samples, and used PCR and direct IFA (MeriFluor *Cryptosporidium/Giardia* Test; Meridian Bioscience Inc., USA) to test the water samples, sand sample, and Envirochek filters for *Giardia*. We tested the water distribution system for septic cross-contamination by
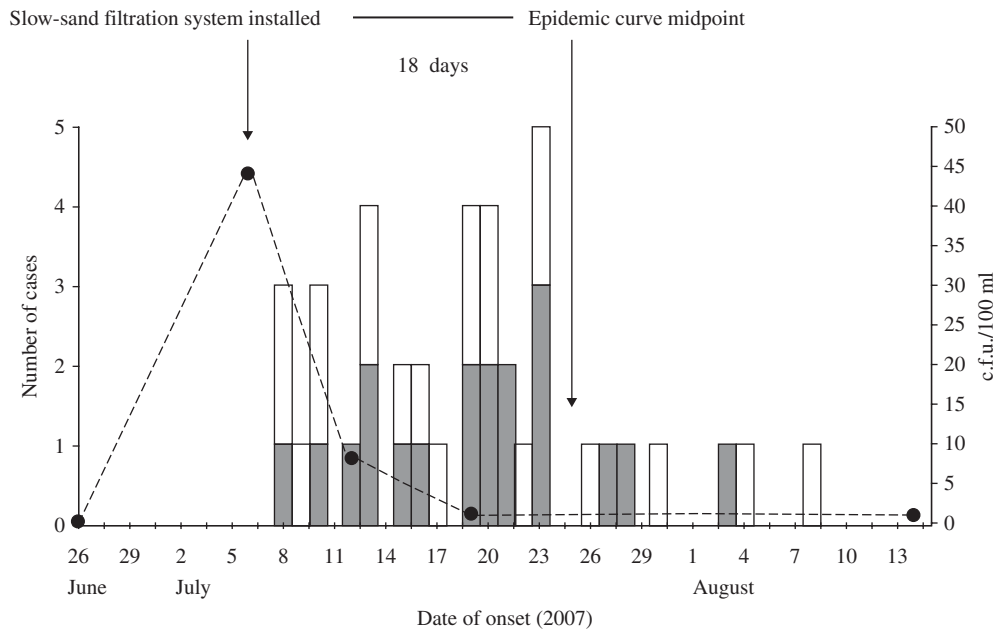
Fig. 1. Date of onset of cases (June–August 2007). ■, Confirmed case; □, probable case; - - -, total coliforms.

flushing about 500 ml fluorescein solution (created by mixing 6 g uranin, 6 g sodium hydroxide, and 500 ml deionized water) down the toilets and testing water samples from the showers for fluorescence 7 days later. A similar approach to evaluating potential hydraulic connections between septic systems and groundwater wells has been successfully used in other investigations [9, 10]. We also interviewed kitchen staff about food and beverage handling and preparation.

All additional cases were detected by survey. Of the 316 scouts, parents, and staff at camp A during the 24–28 June, 5–8 July, and 9–12 July camp sessions, 255 (81%) responded, including 84% (228/271) of parents and scouts and 60% (27/45) of staff. Response rates for scouts and parents attending the 24–28 June, 5–8 July, and 9–12 July camp sessions were 74% (32/43), 86% (122/142), and 86% (74/86), respectively. Of the 30 stool tests performed, 26 were positive for *Giardia*. All seven positive specimens retested at DPD laboratory were positive for *Giardia* by wet-mount and trichrome staining, and two were positive by PCR. The two PCR-positive specimens produced TPI sequences of assemblage B that were primarily to the WB6 and S7 subtypes (GenBank accession numbers AY368167 and AY228634), with only one nucleotide substitution in the 530-bp region analysed.

In addition to the 26 confirmed cases, we identified 24 probable cases, for a total of 50 cases. Four were secondary cases, all of which were laboratory-confirmed, occurred in a single household, and were epidemiologically linked to an index case from that household. Of the 46 primary cases, 34 (74%) occurred in parents, eight (17%) in scouts, and four (9%) in staff. Attack rates for parents, scouts, and staff were 37% (34/92), 6% (8/136), and 15% (4/27), respectively ($P < 0.0001$, χ^2). Attack rates for scouts and parents attending the 24–28 June, 5–8 July, and 9–12 July camp sessions were 0% (0/32), 28% (34/122), and 11% (8/74), respectively (staff attended all three sessions). Eighty-three per cent of ill scouts and parents attended camp A during 5–8 July.

Forty patients reported an illness onset date; onset dates ranged from 8 July to 8 August. Midpoint of the epidemic curve was 24 July (Fig. 1). Three ill staff reported onset dates, which were 22 July, 27 July, and 28 July. The range of patients' age was 7–55 years (median 43 years). Thirty-seven (80%) of the 46 patients were male. The most commonly reported symptoms were diarrhoea (42/46, 91%, of patients), flatulence (34/46, 74%), abdominal cramps (27/46, 59%), and nausea (23/46, 50%). The range of reported duration of diarrhoea was 1–42 days (median 15 days). In all, 25 (54%) patients reported consulting a healthcare provider for their illness. No patients were hospitalized or died.

The retrospective cohort study included 90 respondents. Thirty-eight were scouts, and 52 were parents; 34 were ill, including 20 with laboratory-confirmed

giardiasis, and 56 were well. In bivariate analyses, showering [risk ratio (RR) 1·8, 95% confidence interval (CI) 1·3–2·5] and eating green salad (RR 1·7, 95% CI, 1·3–2·2) were significantly associated with illness, and parents were more likely to become ill than scouts (RR 1·8, 95% CI 1·3–2·5). Other exposures, including swimming in the lake, were not associated with illness. Four respondents reported having drunk tap water; one became ill. On multivariate analysis, only showering remained statistically significant [adjusted odds ratio (aOR) 3·1, 95% CI 1·1–9·3]. Patients reported using all showers at camp A, and no dose–response association was identified between number of showers and likelihood of illness. In all, 73% of parents vs. 26% of scouts reported showering during the 5–8 July camp session.

The slow-sand filtration system consisted of multiple tanks with smooth, vertical walls about 2·5 m high. The spring was protected by a cement box. Spring water was first pumped directly from the box through an enclosed pipe into the filtration system, and then travelled through enclosed pipes to the chlorinator before entering the distribution system. Sand for the filtration system was supplied by a company from Wisconsin, USA, and was unsterilized. The water engineer reported that spring water had been allowed to run through the filtration system for 45 days before the system was put into operation. The chlorinator effluent reportedly had a residual chlorine level range of 1·5–2·0 mg/l during this 45-day period, and a level of 1·0 mg/l after the filtration system became operational. The filtration system was not pilot-tested before installation.

According to the local health department's records, on 26 June 2007, the turbidity of the spring water was 0·1 nephelometric turbidity units (NTU). On 6 July, when the filtration system was operational, total coliform and turbidity levels of the unfiltered spring water were 4 c.f.u./100 ml and 0·05 NTU, respectively, vs. 44 c.f.u./100 ml and 2·0 NTU for sand-filtered, unchlorinated spring water (Fig. 1). On 14 August, total coliform and turbidity levels for the unfiltered spring water were 4 c.f.u./100 ml and 0·20 NTU, respectively, vs. 1 c.f.u./100 ml and 0·16 NTU for the sand-filtered, unchlorinated spring water. Level of faecal coliforms was <1 c.f.u./100 ml for both sand-filtered and unfiltered spring water. Samples of unfiltered and sand-filtered spring water, lake water, and sand from the filtration system were all negative for *Giardia*, and fluorescent dye tests for septic cross-contamination were also negative.

An industrial sanitizer was used to clean cooking equipment, and salad ingredients were purchased pre-cut from commercial sources and not re-cut or washed at camp A. Kitchen staff denied illness, reported wearing gloves when handling food, and denied washing food with unboiled water.

In our investigation, several data sources supported the hypothesis that a giardiasis outbreak at camp A was caused by a point-source exposure beginning during 5–8 July. Clinical characteristics and stool test results indicated that *Giardia* was the aetiological agent, 83% of ill scouts and parents attended only the 5–8 July camp session, and the attack rate for this session was 28%, vs. 0% for the preceding session and 11% for the subsequent session. Furthermore, the epidemic curve midpoint was about 2·5 weeks after the 5–8 July session, an interval that is consistent with the incubation period for *Giardia* [3].

On 6 July, camp A had finished installing a slow-sand filtration system and chlorinator for its spring water. Slow-sand filtration systems are non-pressurized systems whose treatment performance relies on a biological filter created by a complex bio-film layer, or schmutzdecke, that forms on top of a sand matrix [11]. Water enters the system and filters by gravity first through the schmutzdecke and then through the underlying sand. After a filtration system is installed, a period of ≤8 weeks is required for a mature schmutzdecke, capable of biological filtration, to develop. In addition, pilot testing for slow-sand filtration systems, which camp A did not perform, is critical for evaluating likely treatment efficacy for specific source waters.

Elevated levels of total coliforms and turbidity measured in sand-filtered vs. unfiltered spring water on 6 July indicated that the water became contaminated after it left the spring. This water was pumped directly from the cement spring box through enclosed pipes into the filtration system; therefore, it was unlikely to have been contaminated by surface runoff, particularly because no rainfall was recorded at the nearest weather station in June or July [12]. Contamination by wildlife was also unlikely because the pipes were enclosed and the tank's high walls would be difficult for animals to scale. Therefore, the most likely source of contamination was the unsterilized sand from the filtration system itself. It is plausible that the sand was contaminating the spring water not only with coliforms, but also with *Giardia*. The lower attack rate during the 9–12 July camp session supports this hypothesis by indicating a persistent, but

decreasing, source of *Giardia*, as might have occurred if spring water had continued to gradually wash contaminants out of the sand after the filtration system was put into operation. If the sand had been obtained from a stream or lake bed, it might well have been contaminated with coliforms and *Giardia*. However, because sand and water specimens were not tested for *Giardia* until 14 August, well after the outbreak had ended, this hypothesis cannot be confirmed.

The retrospective cohort study identified a statistically significant association between showering and illness, and sand-filtered spring water had been used for showering from 6 July onwards. Unintentional ingestion of *Giardia*-contaminated water while showering is a reasonable mechanism for the outbreak. Although the treated water reportedly had a residual chlorine level of 1·0 mg/l, *Giardia* is moderately chlorine-resistant, and at least one large outbreak of giardiasis associated with an unfiltered, chlorinated water supply has been previously reported [13].

Although the bivariate analysis implicated salad consumption, detailed interviews of kitchen staff did not identify a plausible means for salad consumption to have caused illness. It is more likely that consuming salad served as a proxy for being a parent, as 98 % of parents reported having consumed salad, and parents were substantially more likely to experience illness than scouts, perhaps because parents showered more frequently at camp.

The investigation was subject to at least two limitations. First, only 60 % of staff responded to the survey, so the true attack rate among staff is unknown. Second, because the survey did not include a complete menu, we cannot eliminate the possibility of an association between illness and exposure to a food or beverage about which data were not obtained.

Our findings highlight the importance of using sterile sand in slow-sand water filtration systems, and verifying filtration adequacy through pre-installation pilot testing and post-installation performance testing. Until testing has consistently confirmed the safety of slow-sand-filtered water, this water should not be consumed or used for bathing, showering, or any other purpose by which inadvertent ingestion of contaminated water might occur.

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DECLARATION OF INTEREST

None.

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