An outbreak of cryptosporidiosis associated with a swimming pool

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SUMMARY

In August 1988 an increase was noted in the number of cases of cryptosporidiosis identified by the microbiology laboratory at Doncaster Royal Infirmary. By 31 October, 67 cases had been reported. Preliminary investigations implicated the use of one of two swimming pools at a local sports centre and oocysts were identified in the pool water. Inspection of the pool revealed significant plumbing defects which had allowed ingress of sewage from the main sewer into the circulating pool water. Epidemiological investigation confirmed an association between head immersion and illness. The pools were closed when oocysts were identified in the water and extensive cleaning and repair work was undertaken. The pool water was retested for cryptosporidial oocysts and found to be negative before the pool reopened.

INTRODUCTION

The first documented case of human illness caused by the coccidian parasite, cryptosporidium, was reported in 1976 [1]. Initially, the diagnosis was made by the identification of cryptosporidial oocysts in intestinal biopsies, but since 1983 rapid and simpler methods to detect them in faeces have been introduced [2]. An increasing number of microbiology departments now screen some or all of the faecal samples submitted to them for these oocysts. The total number of reports of cases of cryptosporidiosis received by the Public Health Laboratory Service

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(PHLS) Communicable Disease Surveillance Centre (CDSC) from laboratories in England and Wales rose from 61 in 1983, the first year of reporting, to 7904 in 1989 [3].

Cryptosporidial infections have now been described world wide and several studies have reported a peak incidence in children aged 1–5 years [4]. In the immunocompetent, illness usually lasts under 3 weeks but in the immuno-suppressed, including those with AIDS, the organism can cause a protracted diarrhoeal illness [5]. Infection is transmitted by the faecal-oral route, and spread has been reported from person-to-person [6], animal-to-person [7, 8] and from the ingestion of contaminated water [3, 9-12]. The oocysts are resistant to many disinfectants including chlorine and at present filtration is the mainstay of their removal from potable and swimming pool water.

Investigation of the first reported swimming pool outbreak of cryptosporidiosis which followed recognized faecal contamination of the water revealed that one of the filters was inoperative [13]. We describe the first recognized English outbreak of cryptosporidiosis associated with a swimming pool which again highlights the need for regular and thorough maintenance of swimming pools.

THE OUTBREAK

The microbiology department at Doncaster Royal Infirmary (DRI) diagnosed a total of 79 cases of cryptosporidiosis between 1 June and 11 November 1988; 67 of them between 1 August and 31 October. Screening of all faecal samples for cryptosporidial oocysts had begun in 1984 and they had generally been found in between one and four samples a month.

Many of the early cases were children who lived in or to the north-east of Doncaster. All the cases were interviewed by Environmental Health Officers (EHO) who discovered that most had used the learner swimming pool at a particular sports centre (centre A). This was maintained at a higher temperature (32 °C) than the main pool (29 °C) and was very popular with families with young children. A systematic investigation of the outbreak was carried out in October and November 1988. Hypothesis generating interviews with eight cases confirmed a link with the swimming pool.

METHODS

A primary case of cryptosporidiosis was defined as a person with a laboratory confirmed diagnosis after 1 June 1988 in whose household no one else had had a gastrointestinal illness in the month preceding the onset of their illness, and who had not themselves been abroad in the month preceding their illness. Secondary household cases were defined as persons with a laboratory confirmed diagnosis of cryptosporidiosis in whose household someone else had had gastroenteritis in the month preceding the onset of their illness.

The microbiologists in neighbouring health districts were contacted to establish whether they had noticed a local increase in identifications. A case control study was carried out to determine whether cases were more likely than controls to have used either of the swimming pools at centre A and/or to have immersed their heads in the water there. Only cases diagnosed at DRI were included.

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Two controls within predetermined age bands, and of the same sex as the case for cases over 11 years, were sought for each primary case. One was a neighbourhood control, nominated by the case, who had not had gastroenteritis or travelled abroad in the preceding month. The other was a laboratory-based control selected from those who had had a faecal sample submitted to the microbiology laboratory at DRI within 3 weeks of their matched case. Patients were accepted as laboratory controls if they had had an acute gastrointestinal illness not attributable to cryptosporidiosis, were not a secondary household case of gastroenteritis, and had not travelled abroad in the month before the onset of their illness.

A questionnaire was administered by telephone. It sought personal information on cases and controls, details of gastrointestinal illness, contact with other people with gastroenteritis, recent travel and detail of exposure to known or suspected sources of cryptosporidial infection including domestic and farm animals, untreated water, raw milk and swimming pools. One or two attempts were made by letter to contact cases and controls for whom a correct telephone number could not be found asking them to contact the District Health Authority or the Public Health Laboratory Service Communicable Disease Surveillance Centre.

A further study was undertaken to document the amount of gastrointestinal illness experienced by a cohort of approximately 180 children aged 9–10 years during September and October 1988. Half of the children swam with their school at centre A and half elsewhere. With the agreement of the head teachers, questionnaires designed for completion by parents were distributed via the school health services. Basic personal information, details of gastrointestinal illnesses experienced in those 2 months and details of all swimming pools used were requested.

The data for both studies were coded, entered and analysed using Epi Info [14]. The initial tests used were Yates corrected chi-squared or Fisher's exact two-tailed test for small numbers. An exact binomial probability test which takes account of the variable numbers of matched groups was then carried out on the significant associations [15].

Environmental and microbiological investigations

Results of routine tests on swimming pool water were obtained as well as information on recent plumbing problems. Samples of the water and filter sand from both pools at Centre A were tested for cryptosporidial oocysts at the Public Health Laboratory (PHL) Sheffield according to the method described by Chapman and colleagues [16]. This involved concentration of oocysts from pool water or sand washings by membrane filtration, with subsequent detection of oocysts by an enzyme immunoassay and by microscopic examination of smears stained by a modified Ziehl-Neelsen technique or a fluorescent antibody conjugate.

Once the results of these samples were known a detailed examination of the pool plumbing and water treatment system was organized. Samples taken during this examination included sand and water from each filter, sewage contaminated fluid from the duct around the learner pool, and sediment from the make-up water tank. All these samples were examined for cryptosporidial oocysts. Sand samples were shaken with 0.1 % Tween 80 in distilled water and allowed to stand for 60 sec.

The supernatant was decanted, allowed to stand overnight at 4 °C and then discarded. The sediment was divided between the Scottish Parasite Diagnostic Laboratories, Stobhill Hospital, Glasgow, Sheffield PHL and Yorkshire Water's Regional Microbiology Unit at York. The liquid samples were filtered through 142 mm nylon membrane filters with a pore size of $1.2 \,\mu$ m. The deposits were resuspended in filtrate and then centrifuged at 1000 g for 15 min, resuspended and divided for examination as before.

Samples were taken of all the bore hole water sources that could supply the centre and of the mains water entering the centre. These samples were filtered through Cuno filters according to the method of Smith and colleagues [17]. The filters were cut in three and each portion eluted twice in 0.1 % Tween 80 in distilled water. The eluent was centrifuged and then resuspended and divided as before. Some samples were examined by Yorkshire Water for bacteria and viruses. A cell culture immunofluorescence technique was used to detect rotavirus. Further pool water samples were taken and examined before the pool was re-opened.

At DRI, faecal samples were examined for cryptosporidial oocysts using modified Ziehl-Neelsen staining on a direct faecal smear. There had been no recent changes in the microbiological methods used or the staff undertaking the screening.

RESULTS

A total of 79 cases of cryptosporidiosis were identified by DRI between 1 June and 11 November 1989. No increase in identifications was reported by adjacent laboratories. Eight primary cases who had been interviewed using a hypothesis generating questionnaire and nine identified as secondary household cases were excluded from the case control study. Of the 62 remaining cases 54 (87%) could be contacted; none refused to be interviewed. A total of 43 neighbourhood nominated controls and 48 laboratory controls were also recruited and interviewed. Almost one quarter of the cases were possible secondary household cases and once these and other ineligible cases and controls were excluded, data from 32 primary cases, 19 matched neighbourhood nominated controls and 25 matched laboratory controls were available for analysis.

The ages of the primary cases ranged from 6 months to 70 years (median 6 years), 20 (63 %) were younger than 10 and a similar number of males and females were affected (Table 1). This age-sex distribution was similar to that for all cases. Eight cases, five of whom were primary, required hospital admission, most for less than 2 days. There were no deaths and no cases were known to be immunosuppressed. The week of onset of illness ranged from 20 June to 24 October (Fig. 1). No differences were observed between cases and controls in connection with holidays in the UK, recreational use of water outdoors, consumption of untreated water or milk, and contact with farms, farm animals or pets. Seventeen cases reported using the sports centre A. Eight had only used the learner pool, two only the main pool and seven both pools.

A statistically significant association between illness and head immersion at centre A was found using both laboratory based controls (P = 0.008) and neighbourhood controls (P = 0.002) (Table 2), although an overall significant

 Table 1. Case control study. Age-sex distribution in 32 primary non-overseas travel

 associated cases of cryptosporidiosis

Age (years)	Male	Female	Total
≤ 1	2	2	4
2-4	4	4	8
5-9	5	3	8
10-19	2	0	2
20-39	4	2	6
≥ 40	1	3	4
Total	18	14	32

Table 2. Case control study. Head immersion in any pool at centre A

Head immersion	Case	Laboratory-control
Yes	8	1
No	10	18

Case – Laboratory control: Matche
dP=0.008,Odds Ratio 14.40, 95% Confidence Limits 1.46–673.41

Head immersion	Case	${f Neighbourhood}$ -control
Yes	13	4
No	11	21

Case – Neighbourhood control: Matche
dP=0.002,Odds Ratio $6.2,\,95\,\%$ Confidence Limit
s1.41-31.36

 Table 3. Schools study. Number of children reporting gastrointestinal illness by school

School	Number in class(es)	Number of completed questionnaires*	Number with definite illness reported†
1	28	16	2
2	30	17	5
3	30	28	13
4	30	27	4
5	30	22	6
6	29	28	1
Total	177	138	31

* 1 completed questionnaire did not have a school stated.

† 15 returned questionnaires did not indicate a definite yes or no to illness.

association between the use of the pools at centre A and illness was not demonstrated.

Schools study

In all 139/177 (79%) questionnaires were returned. Thirty-one children were definitely reported to have had gastroenteritis but none had had stool samples taken (Table 3). The data suggested an association between illness and swimming with the school at centre A (P = 0.035) (Table 4). Attendance at another local

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Table 4. Schools study. Attendance at centres A and B^*

	Numbers of definite cases	Numbers of definite non cases
Attended centre A	16	30
Attended centre B	10	54

P = 0.035, Odds Ratio 2.88, 95% Confidence Limits 1.06-8.00.

* 114 children were reported to have gone swimming with the school and reported a definite yes or no to illness. Four children were excluded as they did not specify which centre they attended with the school.

Table 5. Reported illness and association with School 3

	Attended School 3	Did not attend school 3
Case	13	18
Non-case	12	81

6 5 Number of cases 4 3 2 11 18 25 12 19 26 5 3 10 17 24 31 7 20 27 4 1 8 15 22 29 14 Aug Jun Jul Sep Oct Nov Week beginning

P = 0.001, Odds ratio 4.88, 95% Confidence Limits 1.71–13.75.

Fig. 1. Week of onset of illness in 32 primary, non-travel associated cases of cryptosporidiosis.

swimming pool (centre B) was shown to have a protective effect in that children who swam there were significantly less likely to be ill. A statistically significant association (P = 0.001) was also shown between school 3 and illness with 13/25 children reporting illness, 11 of whom had swum at centre A (Table 5).

Environmental results

Two problems had been reported to the pool management at centre A in the months before the pools were closed; an unusual smell in the changing rooms and difficulties in controlling the levels of free chlorine residuals. Despite investigation, no cause had been found for either problem and the results of routine water tests had shown satisfactory results. After the learner pool had been closed about 30 cm of liquid sewage was found in the deep end. When the manhole distal to the main changing area was lifted, approximately 30 cm of solid sewage were found above, but not blocking the main sewer. (Fig. 2, manhole cover 1).

The learner pool was surrounded by a concrete deck under which was a dry duct

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area the depth of the swimming pool and several feet wide. Access to the area was via a man-hole cover on the deck. Pipes running along the base of the duct returned water from the pool scum channels to the filter house under negative pressure. Water from the pool deck drained through surface water drains into risers which entered pipes attached to the underside of the deck. These pipes discharged into the main sewers at the far end of the site (Fig. 2). This duct area was separated from the main sewer by a concrete wall, although it was traversed by a U trap between the sewer and the surface water collection pipes.

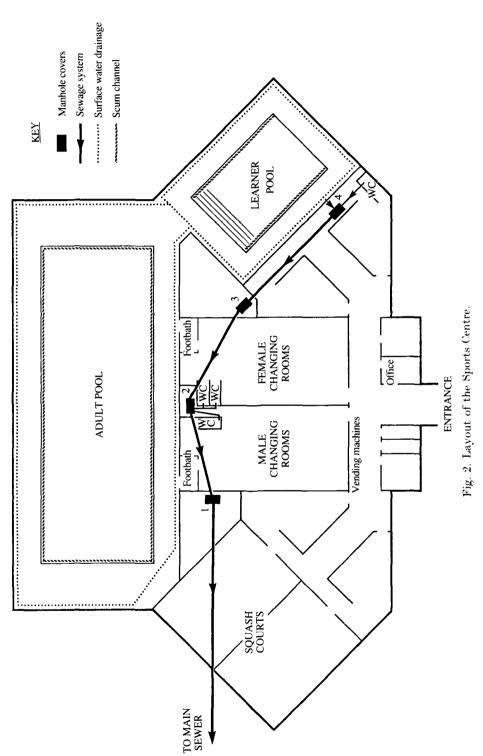
When the dry duct area was inspected sewage and water had flooded it to a depth of several inches. The surface water collection pipes were found lying on the bottom of the duct area and the U trap had rotated through 180°. Water from the pool deck had run through the risers and accumulated in the duct. Sewage contamination of the duct occurred when the sewer was blocked and sewage had apparently flowed down the U trap into the duct through the holes in the surface water collection pipes where the risers entered. This was confirmed when the block in the sewer was recreated and water flowed into the duct. Disconnected jubilee clips on the pipes draining water from the scum channels enabled the sewage contaminated fluid to be sucked into the circulating pool water under negative pressure.

Microbiological results

The first learner pool samples were found to contain cryptosporidial oocysts both on conventional microscopy and on enzyme immunoassay (EIA), at a concentration of 50/litre. Samples of the main pool water were consistently negative. However the first sand sample from the learner pool filter was found to contain cryptosporidial oocysts by both methods and samples from the main pool filters by the EIA. Cryptosporidial oocysts were identified in the learner pool filter water and in the sewage contaminated fluid from the dry duct at a concentration of 80 oocvsts/litre. Rotavirus was also detected in these two samples. The counts were 0.5 fluorescent foci per litre (FF/l) from the learner pool filtrate, and 3.8 FF/l from the sewage contaminated fluid. The mains water to the sports centre was negative for cryptosporidial oocvsts. One of the repeat samples after cleaning and disinfection was found to contain oocysts at a concentration of 0.6 oocyst/litre. This sample had been taken from a dead leg attached to the filter which had not been adequately flushed through during disinfection. The sampling process had flushed out the contamination and further repeat samples were consistently negative.

Control measures

The learner pool was closed and emptied immediately the results of the first water tests were available. The main pool was closed soon after because the tests had identified oocysts in the filter sand and there was concern about low level contamination of the water. A specialist company was employed to decontaminate the entire system. The U trap between the sewer and the surface water pipes was removed and the surface water was re-routed to enter the circulating pool water in the filter house. The EHOs advised affected families on good hygiene practices during their home visits to prevent secondary cases.



DISCUSSION

The statistically significant association between head immersion at centre A and illness suggested that swallowing of contaminated water was important in causing illness. The microbiological and environmental evidence to associate the use of a specific pool with illness was strong although epidemiological results failed to show a statistically significant association between use of the pool and illness. No other associations were shown.

The majority of cases were willing and able to nominate one or two neighbourhood controls but for two main reasons an eligible control could not be found for each. A number of controls had had a gastrointestinal illness themselves in the month before their matched case's illness which confirmed anecdotal evidence of widespread mild gastrointestinal illness in the community during that period. Others had recently travelled abroad and this fact also affected the recruitment of laboratory controls; a particular problem of outbreak investigations involving the peak holiday period. A potential laboratory control was identified for all but one case but about a fifth could not be contacted. The large number of children with cryptosporidiosis made the identification of further possible age and sex matched laboratory controls impractical. Consequently, the final statistical analysis was performed on a data set much smaller than expected.

The results from the schools study again confirmed anecdotal reports of mild gastrointestinal illness in the community during the early autumn. As none of the children had had faecal samples examined no pathogen could be identified but a viral aetiology seems likely. Rotavirus was identified in two samples, the learner pool filtrate and the sewage contamination fluid and it is possible that some of these mild illnesses were due to rotavirus.

The likely involvement of centre A in the outbreak was recognized early on but it was initially suggested that a bather excreting oocysts might have contaminated the water. However, after further investigation it became clear there had been serious problems with the plumbing, particularly backflow from the sewers into the duct around the learner pool. Intercurrent defects in the pipes from the scum channel had allowed sewage contaminated fluid to enter the pool circulation. The original source of the oocysts remains unknown although it is most likely that they were excreted by someone who used the toilets. Oocysts were not identified in large volumes of the mains water supplying the sports centre. The infective dose of oocysts for humans is unknown but is probably small, possibly under 10 oocysts [3], and the concentration of oocysts detected in the pool water samples that were tested was 50 oocysts/litre.

The oocysts are resistant to many disinfectants and optimally functioning filters which are believed to remove most oocysts are the mainstay of their removal [18, 19]. The presence of corroded distribution rings in the main pool filters and the uneven sand surface suggested that these filters may not have been functioning at their maximum efficiency which might have decreased the proportion of oocysts removed. Chapman and Rush have investigated the passage of oocysts through a laboratory model of a sand filter. The results indicated that oocysts do not pass easily through the filter although a quantitative result was not available. Some disintegration of the oocysts may occur during this process [20].

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It is impossible to know when the faults in the plumbing developed although the reports of an odd smell in the changing room during the summer may have coincided with the blocking of the sewer and the flow of sewage into the duct. Regular inspections of the duct area should have allowed earlier recognition of the faults. In retrospect the difficulty experienced in controlling the free chlorine residual levels when the testing point was proximal to the filters could also be explained by sewage contamination of the pool water.

A number of outbreaks of cryptosporidiosis have now been documented where there was epidemiological and/or laboratory evidence of waterborne spread and in some there was evidence of contamination of the potable water supply [3, 9, 11, 12]. In one instance this was thought to have occurred because well water had become contaminated with sewage [9], and in some others an unusually large number of oocysts were likely to have been present in the water reaching the treatment works. In two of these outbreaks a review of the water treatment processes identified factors that were likely to have increased the number of oocysts in the treated water [3, 12]. A further outbreak was associated with post treatment contamination of potable water [11], and another with consumption of untreated surface water [10]. Investigation of the first reported swimming pool associated outbreak of cryptosporidiosis revealed that one of three filters was inoperative [13]. Inadequate pool maintenance was felt to be a contributory factor in that outbreak in common with the outbreak described here and outbreaks of other illnesses associated with swimming pool contact. In one reported outbreak of swimming pool associated giardiasis, turbid water and low free chlorine residuals were reported in a pool used by infant and toddler swim classes [21]. In another, chlorine levels were not recorded on the day a child had a faecal accident in the pool and the following day the chlorine level was zero [22]. Outbreaks of adenovirus infections have also been reported in association with swimming pools and in one, the same type of adenovirus was recovered from cases and the pool water [23]. Investigation of all these outbreaks highlighted the need for adequate daily pool maintenance.

In the outbreak described in this paper, sewage contamination of the pool water occurred because of several plumbing defects principally a break in the surface water collection pipe. Additional operational problems were identified later. The detailed investigation of the outbreak was facilitated by close liaison between all those involved. The recommendations for future action resulting from the investigation included regular detailed inspection of the entire pool and treatment systems, and the need for staff to be sensitive to reports of unusual phenomena. The Pool Water Treatment Advisory Group produced a Code of Practice for action in the event of an identified faecal accident in a pool [24]. Department of the Environment guidance on the treatment and quality of swimming pool water is also available [25] and the Standing Committee of Analysts has recently published the current provisional recommendations for the isolation and identification of cryptosporidial oocysts in water [26]. In addition, the report of the Department of the Environment and Department of Health's Group of Experts on Cryptosporidium in Water Supplies outlines action to be taken when a waterborne outbreak of cryptosporidiosis is recognized [3].

This is the second documented outbreak of cryptosporidiosis associated with a

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swimming pool. While there were exceptional circumstances, exposure to sewage contaminated water should be considered in the investigation of cases of cryptosporidiosis. Swimming is a popular activity in the UK and a potential public health hazard if pools are poorly maintained.

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