Virus-Associated Outbreaks in Swimming Pools

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Throughout history, swimming for recreation has been popular in virtually all societies and countries. As the science of epidemiology and microbiology evolved, so did suspicions of risks of disease transmission associated with swimming in fresh or marine waters and, in recent history, swimming pools. Consequently, increasing numbers of outbreaks have been reported and documented. Compilation of the outbreaks and review of applicable pool problems emphasize that microbial health risks are indeed a concern to be reckoned with. Numerous epidemiological studies of bathers at marine and freshwater beaches have clearly demonstrated a risk of acquiring microbial illness such as gastroenteritis, respiratory infections, and hepatitis (Cabelli et al. 1983; Holmes 1989; Seyfried et al. 1985). Such illnesses, however, are rarely associated with well maintained and disinfectant-containing swimming pools. Enteric viruses, which are excreted in the feces and respiratory secretions, have been associated with swimming in contaminated pools and surface waters. This study reviews the current information on swimming pools associated with viral illnesses.

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Introduction

Viruses are obligate intercellular parasites. Consequently, their occurrence, growth, and transmission through the environment are dependent upon the invasion of living cells within a host organism. For this reason, viruses are usually very host specific, i.e., human viruses usually only infect humans. Human enteric viruses are those that infect the gastrointestinal tract of man and are excreted in the feces, and/or nasal or respiratory excretions. Man is the only known natural host. Enteric viruses are spread by the fecal–oral route, i.e., by ingestion of fecally contaminated water and food, or by touching contaminated surfaces (fomites). Enteric viruses are among the simplest living things being composed only of hereditary material (nucleic acid) and a protein coat. They are very small, only 25–80 nm in size. There are more than 140 different types of enteric viruses that are capable of causing a wide range of diseases (Table 1), but most infections only result in minor illness, such as fever, rash, or the "common cold". As few as 1–10 organisms ingested are believed capable of causing an infection. Only about half of the persons infected actually develop illness. The non–ill person, however, is still capable of excreting large amounts of virus into the environment. As many as 10^{10} viruses per gram of feces are excreted by infected individuals.

Although long suspected, transmission of viral infections in swimming pools has been difficult to

prove. Various studies have demonstrated the occurrence of viruses in swimming pools (Table 2), but epidemiological studies have been few because of the difficulty of isolating enteric viruses from clinical and environmental samples.

Virus group	Serotypes	Some diseases caused
Enteroviruses		
Poliovirus	3	Paralysis, aseptic meningitis
Coxsackie virus		
Coxsackie virus A	23	Herpangia, aseptic meningitis, respiratory illness, paralysis, fever
Coxsackie virus B	6	Pleurodynia, aseptic meningitis, pericarditis, myocarditis, congenital heart disease, anomalies, nephritis, fever
Echovirus	34	Respiratory infection, aseptic meningitis, diarrhea, pericarditis, myocarditis, fever, and rash
Hepatitis A virus (HAV)		Infectious hepatitis
Rotaviruses	5	Gastroenteritis
Adenoviruses	49	Respiratory disease, acute conjunctivitis, gastroenteritis
Norwalk agent (calcivirus)	many	Gastroenteritis
Astroviruses	5	Gastroenteritis

Table 1 – Some Human Enteric Viruses

Pool Type*	Location	Viruses Isolated	Chlorine (mg/l) Free	Total	Reference
S	Canada	parainfluenza 1	0.4	NR+	McLean 1963
W	Canada	coxsackie B1	0	NR	McLean 1973
W	New York	echo 3, 11	0	NR	Kelly 1961
S	Israel	echo 6, 7; coxsackie B6	NR ‡	NR‡	Marzouk *et al.* 1980
S	Georgia	adeno 4	0	0.5	D'Angelo *et al.* 1979
S	Germany	coxsackie B3	NR	0.3–0.5 ξ	Liebscher 1970
W	Soviet Union	coxsackie B1	NR	NR	Osherovich and Chasovnikova 1969
S	Texas	echo 7 polio 1 coxsackie B4	0–2.2	0–2.5	Keswick *et al.* 1981
W	Texas	coxsackie B3 polio 1	0	0–0.5	Keswick *et al.* 1981

* S = swimming pool; W = wading pool

\+ NR = not reported

‡ Three of six pools had residual chlorine

ξ Applied dose

Table 2 – Previous Reports of Viruses Isolated from Swimming and Wading Pools

OUTBREAKS

Adenovirus Type 3 (AV-3)

• An outbreak of pharyngo–conjunctival fever due to adenovirus type 3 occurred in two swimming teams shortly after exposure to unchlorinated water in 1966 in Seattle. Children swimming in the chlorinated water suffered no similar illness. The attack rate was 65 and 67% for each team. The manager regularly shut down the chlorinator when closing the pool at 10 p.m. to save the morning swimmers from eye irritation from chlorine which might interfere with their subsequent school work. The chlorinator was started again at 10 a.m. Each of 10 acutely ill swimmers in the morning group who were investigated within 10 days of onset of symptoms yielded adenovirus type 3 in the throat or conjunctival specimens or both.

The unique feature of this outbreak of pharyngoconjunctival fever was the sharp simultaneous epidemic among two morning swim teams, swimming at different hours, while the afternoon swimmers remained well. Yet the afternoon swimmers were in person–to–person contact with the swimmer who contracted the infection by swimming in the morning twice, and by two secondarily infected siblings of morning swimmers. It seems logical to associate the outbreak with the lack of chlorination of the pool in the morning.

The bacteriological analysis of the water samples after the outbreak revealed acceptable quality of the water. The coliform counts were negative even though the chlorine residuals were 0 at the time the samples were collected. Virus could not be isolated from the pool water.

It could not be determined how the pool became contaminated, but the pool was without chlorination for 36 hours after back washing over the weekend before use by the children. In most outbreaks of pharyngo–conjunctival fever associated with swimming, chlorination has been reported to be satisfactory, although this does not appear to have been thoroughly investigated. The epidemic curve for both swim teams showed the same timing, which supported the hypothesis that the infection with adenovirus was derived from a common source and not by contact from child to child.

• In 1991 an outbreak of acute respiratory illness occurred in campers and counselors at a summer camp, followed by another outbreak during a second four–week session at the camp. Manifestation of the illness included pharyngitis, cough, fever to 40°C (104°F), headache, myalgia, malaise, and conjunctivitis.

During the first summer session, 226 persons (175 campers and 51 staff members, i.e., counselors, administrative staff, and infirmary personnel) visited the camp infirmary because of onset of symptoms of upper respiratory illness. During the second session, 369 campers and 86 staff members visited the infirmary with the same upper respiratory manifestations noted during the first session.

Every camper swam at least once during the four weeks [158 (90%) of 175] swam one or more times per day. The attack rate for campers who swam daily [74 (48%) of 153] did not differ significantly from that for campers who swam less than once per week [11 (65%) of 17]. The attack rate for staff who swam was higher than that for staff who did not swim [10 (77%) of 13, versus 13 (54%) of 24] and increased with increased frequency of swimming. The attack rate for non–swimmers was 54% (13 of 24); for infrequent swimmers (i.e., those who swam once per week or less) was 75% (six of eight); and for frequent swimmers (i.e., those who swam three or more times per week) was 80% (four of five). Of the 221 campers and staff members interviewed, 75 (41 campers and 34 staff members) reported whether they had shared a towel with another person. Towel sharing increased the rate for illness (11 of 12 who shared versus 31 of 63 who did not).

Of viral cultures (nasopharyngeal and throat swabs obtained from 25 ill persons), 19 grew adenovirus serotype 3. Bacterial analysis of grab samples of water obtained from the pond yielded 80 colonies per 100 ml of fecal coliforms, 200 colonies per 100 ml of Staphylococcus, and 9,000 colonies per 100 ml of Staphylococcus. A concentrated sample of pond water drawn approximately six feet below the surface yielded adenovirus serotype 3. Residual chlorine was not detectable.

Because of the turbidity of water in soil–bottom reservoirs, chlorination is ineffective. Turbid water contains organic molecules (e.g., humic and fulvic acids from plant decay) that react with chlorine. Viruses may attach or embed in suspended particles in turbid water, and these virus–containing particles precipitate into the sediment on the bottom where they may remain viable in the cooler temperatures. The virus containing particles may become re–suspended when the water is agitated by swimmers.

• An outbreak of illness due to adenovirus type 3 (AV–3) occurred in residents of a suburban community, Dekalb County, Georgia, from June 6 to July 24, 1977. Based on surveys, at least 105 cases occurred. This illness was primarily characterized by sore throat, fever, headache, and anorexia (loss of appetite). Conjunctivitis affected only 34 of 105 (32%) of the individuals contacted during two surveys. Frequent use of a private swimming pool in a community residential district was associated with the illness. The outbreak coincided with a temporary defect in the pool filtration system which probably prevented maintenance of proper chlorine levels in the pool water, suggesting that the infection was in fact spread by pool water.

A local pool service company was responsible for maintenance of the swimming pool. Chlorination of the pool water was done by the daily manual addition of calcium hypochlorite (65% by weight) and was monitored by twice–daily free–chlorine estimates using the orthotolidine method. Cyanuric acid was added as a chlorine stabilizer. No permanent records of chlorine determinations or pH had been kept prior to the outbreak. During the last week of June 1977, the pool water became increasingly turbid and then turned green, presumably due to the presence of algal overgrowth. Reports of ill swim team members began shortly thereafter.

On July 3, one of four valves regulating flow through the pool filter was found to be defective, causing an ineffective backwash cycle. The defective valve mechanism was repaired and the pool water hyper–chlorinated with approximately 13.6 kg of 65% calcium hypochlorite. Over the next 24–hour period, there was dramatic improvement in water clarity. Although chlorine demand purportedly improved following repairs, free–chlorine estimates as low as 0.2 mg/liter were documented as late as July 12.

Ages of the 54 ill persons (in the initial survey) ranged from one to 46 years, with a median of 12 years. The onset of illness was abrupt, with headache, fever, sore throat, and anorexia being the most common symptoms (Table 3).

Conjunctivitis was reported in 19 (35%) cases and lasted 2–7 days. It was unilateral in 6 (32%) and bilateral in 13 (68%). The overall duration of illness for the 37 who had recovered at the time of an interview ranged from 2–20 days with a median duration of six days. The 17 persons who were still sick when interviewed had been ill 1–13 days (median, 9 days).

Symptom	Number with symptom	% with symptom
Sore throat*	43	80
Fever*	42	78
Anorexia*	36	67
Headache*	35	65
Nausea	21	39
Chills	20	37
Cough	20	37
Earache	20	37
Conjunctivitis*	19	35
Runny/stuffy nose	19	35
Vomiting	18	33
Myalgia	16	30
Swollen glands	14	26
Sputum	9	17
Diarrhea	6	11
Rash	2	4

* Included in case definition

Table 3 – Symptoms in 54 Cases Due to Adenovirus Type 3 (Georgia 1977)

In this study, 19 persons had evidence of AV–3 infection by culture, serologic tests or both. In contrast to asymptomatic fecal shedding of AV–3, which has been described as persisting for months following AV–3 infection.

This outbreak is interesting from a clinical standpoint for two reasons: 1) conjunctivitis played a relatively minor role in the overall symptomatology, and 2) earaches were reported by an unexpectedly high proportion of ill persons (37%).

In this study, the higher attack rate among individuals with higher swimming frequency may have been a reflection of increased exposure to contaminated pool water, increased exposure to other ill persons at the pool, or both. If waterborne spread was the predominant mode of transmission, one might expect to see a significantly higher attack rate in swimmers than in those who merely visited the pool. Although individuals 20 years or older who visited and swam in the pool had a higher attack rate than those who just visited the pool, the difference was not significant.

Prior to the discovery of the defective swimming pool filtration system, the apparent overgrowth of algae, usually suppressed by recommended free–chlorine concentrations of 0.4–1.0 mg/liter, suggests that at the time of peak disease occurrence free–chlorine was likely at a suboptimal concentration. The survival of other bacteria and viruses, and AV–3 in particular, has additionally been shown to be affected by chlorine concentration, pH, and temperature.

When the investigation began, the defective filtration system had already been recognized and corrected, and the water hyper chlorinated. If pool water were important for transmission in this outbreak, inadequate chlorination after repairs, suggested by low free–chlorine estimates, might be cited as contributing to the occurrence of later cases. Alternatively, the onset of illness in these individuals might have merely been a reflection of the incubation period for naturally occurring AV–3 disease, estimated as having a median incubation of about 5–10 days.

Adenovirus Type 4 (AV–4)

• Infections with adenovirus type 4 rarely occur in the general population, but is frequently responsible for acute respiratory disease (ARD) among military recruits. During the summer of 1977, an outbreak of pharyngoconjunctival fever (PCF) occurred at a private recreational facility in Georgia. The lack of involvement of the lower respiratory tract and the

high percentage of patients with conjunctivitis (68%) distinguished this illness from the epidemic disease usually seen among recruits.

A total of 72 cases of PCF was identified. Adenovirus type 4 (AV–4) was recovered from conjunctival or pharyngeal swab specimens from 20 of 26 persons in the group of cases tested. AV–4 was also recovered from two concentrated samples of water obtained from the swimming pool at the facility of different dates. All persons affected had direct or indirect contact with the pool. A relation between the amount of time spent in the water and the attack rate was demonstrated. Investigation showed that inadequate amounts of chlorine had been added to the pool water. Frequently, levels of free chlorine were below the recommended level of 0.4 mg/liter. Breakpoint chlorination and closing of the pool for the summer stopped the spread of PCF.

Since no breakdown in the pool's filtration or chlorination system appears to have preceded this outbreak, it must be concluded that the consistently inadequate amount of chlorine added and the poor mixing of the chemical in this large pool were responsible for the survival of the virus in the water. Moreover, the relatively long incubation period of PCF and the continued shedding of adenoviruses by children after convalescence made reintroduction of the virus probable even if, by chance, effective levels of free chlorine had been reached.

Adenovirus Type 7 (AV–7)

• Acute conjunctivitis as a single disease entity has been produced by various adenoviruses and is a component of pharyngoconjunctival fever. Swimming pools have been presumed to be the source of many adenoviral epidemics, ostensibly because the chlorine–induced eye irritation may predispose the eye to infection. However, in many of these outbreaks the pool has been only a possible source of infection and it is not clear whether there is a indirect cause–and–effect relationship. The purpose here is to describe an epidemic occurring in swimmers and the events leading to the incrimination of the pool as the vehicle for spread of the epidemic.

On January 20, 1973, a Kansas community swimming club was limited in its ability to compete in a swimming meet because of a large number of acute conjunctivitis cases among the participants. One of the coaches notified the county health department that an epidemic situation existed. At this time it was estimated that approximately 20 persons from a team of 80 were affected, and it was believed that within the community only members of the community swimming team were involved.

Information from local physicians and hospitals indicated that the outbreak involved persons associated with the local junior high school. Consequently, a questionnaire was prepared and administered to as many individuals as possible who were known to have contact with this school during the epidemic period. From these data 44 cases of acute conjunctivitis were identified.

The original indication that the illness was limited to the community swim club was found to be erroneous. Other groups, including some school swim class members, were affected as well (Table 4). It was evident that only swimmers were at risk, since the gym and health classes had a zero attack rate. Furthermore, the risk was clearly greater for the evening swim groups which included 41 of the 44 cases. Two of the three–day cases occurred in persons who swam in the last hour of the day, and the third day case was the sister of a night case.

The only common factor was contact with the swimming pool, since there appeared to be no intergroup contact involving all of the patients. The first two cases occurred in boys known to have shared swimming goggles. However, so far as could be determined, no other vehicles (such as towels) were transferred from group to group. The swim classes used public school equipment; all other groups provided their own equipment.

Examination of the pool records revealed that in early December the filter sump pump had failed.

Group	Total persons in group	# ill in each group	Attack rate (%)
Gym class*	452	0	0.0
Health class*	116	0	0.0
Swim class	181	3	1.7
Diving team	4	0	0.0
Swim team	16	3	18.8
College class	17	3	17.7
Local swim club	91	30	33.0
Family open swim	14	5	33.3
Total	892	44	4.9

* No pool contact

Table 4 – Acute Conjunctivitis Distribution of Cases by Group

Manifestations	Number Persons	Percent (%)
Red or pink eyes	44	100.0
Swollen eyes	33	75.0
Painful eyes	31	70.5
Tearing	29	65.9
Exudate	23	52.3
Fever	23	52.3
Headache	20	45.5
Photophobia	16	36.4
Crusts	16	36.4
Lymphadenopathy	15	34.1
Cough	13	29.5
Nausea	10	22.7

Table 5 – Acute Conjunctivitis: Clinical Manifestations of 44 Patients

This equipment normally filtered the pool water every eight hours and automatically chlorinated the water by adding a set amount of hypochlorite stock solution each hour. When the sump pump failed, the diatomaceous filter became plugged and the chlorinating equipment was rendered inoperative. The amount of chlorine present in the pool water was determined each morning by the coach. When the level was low, the coach added by hand an amount of hypochlorite powder estimated to raise the chlorine content to a safe level, but no further additions or tests were made until the next morning. In general, the chlorine levels were lower during the epidemic period than previously.

The clinical picture was variable, with temperatures ranging from 37 to 39°C (99 to 103°F) (Table 5). One patient was ill enough to require hospital admission. The average duration of illness was not precisely determined, but most persons were well again within 5 days.

Conjunctival and pharyngeal swabs were taken from eight acutely involved patients for viral and bacterial identification. The adenoviruses were identified as belonging to the adenovirus 7 group. Of the eight persons cultured, four were found to have the virus present in both eyes and throat, while two had the virus isolated from the throat only.

The specimens of pool water collected for virus and bacterial isolation were negative but these had been taken after the pool had been super–chlorinated on January 23.

Adenoviruses as a group are known to produce a wide variety of illnesses depending on the host response. The symptomatology found in the persons involved in this epidemic (Table 5) demonstrated the range of clinical manifestations produced by a single infecting agent, adenovirus type 7.

Type 7 adenovirus was first isolated from the phayrnyx in 1955. Since that time it has been found in mild ocular diseases as sporadic infection and in epidemics of pharyngo–conjunctival fever, but most often in respiratory diseases both in sporadic illness and epidemics. Serious respiratory illnesses have been observed, and gastroenteritis with nausea, vomiting and diarrhea has been prominent in some cases. Skin manifestations, especially rash, have occurred and central nervous system disease has been reported with headache and/or meningism a common complaint.

In this outbreak, even though eye signs and symptoms predominated, a variety of other complaints were also noted. All of the manifestations listed have been found in cases of adenovirus type 7 infections.

The transmission of adenoviruses by swimming pool water has been suspected in the past but the addition of chlorine, which inactivates adenovirus type 3 should prevent this. However, in this epidemic, similar to the epidemic reported by Foy *et al.* (1968), the water was inadequately chlorinated because of equipment failure.

The day swimmers in the school classes were largely protected because the coach had added hypochlorite powder to the pool when the morning residual chlorine level was low. However, the extra chlorine was gradually inactivated by organic debris from the hair, skin, and bodies of the day swimmers so that the pool was left essentially un–chlorinated for the late afternoon and evening swimmers, as shown by the morning tests of the chlorine level.

It matters little whether the adenovirus type 7 was continuously present in the water or whether it was reinoculated on subsequent days as increasing numbers of persons entered the prodromal period just prior to swimming. The epidemic was easily controlled by raising the pool's chlorine level above 0.3 ppm.

• In July 1982, an outbreak of pharyngitis caused by adenovirus type 7a occurred among children in a small town in western Oklahoma. Predominant symptoms were fever and sore throat (by case definition), headache (83%), abdominal pain (64%), and conjunctivitis (51%). At least 77 persons were identified whose symptoms met case definitions for illness. Onsets of the illness peaked during the week of July 5 to 12 and the outbreak resolved within three weeks.

During the second week of July, 12 persons who had been seen in a local clinic with an illness consisting of fever, sore throat, nausea, and vomiting were reported to the Oklahoma State Department of Health. All were less than 18 years of age. Interviews revealed only three exposures that were common to at least one third of them: swimming at a local community pool, attendance at a softball game, and visiting a local lake recreation area.

As a result of the initial information about the outbreak, an epidemiologic investigation was begun by members of the health department. For the epidemiologic surveys, a case was defined as an illness characterized by fever (37%), sore throat, and at least one other symptom beginning on or after July 1.

A systematic telephone survey of the town revealed that persons who had swum at the swimming pool were more likely to be ill than those who had not. A second survey of families with season passes to the pool showed that among swimmers, illness was directly related to the average number of hours exposed to the pool each week. In addition, swimmers who reported swallowing pool water were more likely to be ill (29 of 56) than persons who did not (10 of 41).

Throat–swab specimens from five of seven ill persons grew adenovirus 7a compared with one of 12 well persons. The pool chlorinator had reportedly malfunctioned during early July. Of the 77 persons who met the case definition, 12 were from the initially reported group, 18 from the first survey, and 47 from the second survey. Sixty–four cases occurred in persons who swam at the community swimming pool. Incidence peaked in the second week of July and had virtually subsided by the last week of July. Predominant symptoms were fever and sore throat (by definition), headache (83%), abdominal pain (64%), conjunctivitis (51%), diarrhea (49%), nausea or vomiting (54%), cough (43%), earache (28%), and rash (10%). Two persons who met the case definition were hospitalized with dehydration from persistent vomiting.

The pool was equipped with an automatic chlorination and pH regulator system which was not operational between July 1 and July 19. During that time, the pool was chlorinated by hand. No permanent records of chlorine testing were kept, but pool personnel said that chlorine levels had ranged from 0.5 to 1.5 ppm, with the lowest levels occurring during peak hours of use. After July 19, when the automatic chlorinator was repaired, the chlorine level in the pool water was continuously monitored and adjusted.

The symptoms of ill persons in this outbreak were consistent with adenovirus infection. In addition, adenovirus 7a was cultured from significantly more ill persons than well persons, implicating that virus as the etiologic agent. In the two surveys in this investigation, exposure at a community swimming pool was associated with illness, and the attack rate for illness increased in relation to length of time spent at the swimming pool. Persons who reported swallowing pool water were significantly more likely to have been ill. It is possible that swallowing water was a marker for more intense exposure to the swimming pool water.

Based on the attack rate for illness from the sample survey, it was estimated that approximately 400 cases of adenovirus 7a infection occurred in the affected community in July. More than two thirds of these had exposure to the swimming pool.

Hepatitis A Virus

• The first hepatitis A (HAV) outbreak associated with a swimming pool was reported by Solt *et al.* (1994). In September 1979, in a small town in the north–eastern region of Hungary, a hepatitis epidemic occurred with an abrupt onset. In the middle of September, 31 children were hospitalized with clinical manifestations of hepatitis. On the basis of the epidemiological investigations, the common sources of infection such as raw and uncooked food, milk, and milk products, ice cream, drinking water, soft drinks, etc., were excluded. Person–to–person transmission which could have had a role in the outbreak was also excluded. All of the patients were visitors of a summer camp site in August, and all of them had reported bathing or swimming at a health spa, predominantly in the "large" swimming pool. All were local citizens. In order to identify all hepatitis cases associated with the outbreak, the investigators organized a country-wide surveillance. They found 25 additional cases in five counties and in Budapest. Of the total 56 cases, 70% were boys, in age from 5 to 17 years. Using an experimental hepatitis A antigen to detect antibody in the patient's serum, the authors determined that the causative agent was hepatitis A virus.

The town has a health resort (18,000 sq) that is a camp site with three different pools of thermal water. The amount of the water supplied by the cold and thermal springs was not sufficient for regular refilling of the large swimming basin (3200 m^3). Four days were required to completely fill the swimming pool. During that time the visitors, especially the children, used the half–filled pools, which were operated without any treatment and disinfection procedures.

Patients with hepatitis were found only in organized groups staying there between August 1–8 (13, 6, and 15%). Exact times of swimming pool use were known in the case of 36 patients. Nine were children who reported swimming only on one occasion. It was established that the time of exposure was between August 2 and August 4. On August 1, the large swimming pool was emptied, and refilling began the following day. At that time the resort and the thermal spa were overcrowded. Overcrowding and bad hygienic conditions might have played a role in fecal contamination of the large swimming pool.

• The first known outbreak of hepatitis A (HAV) in the United States was associated with the use of a swimming pool and was reported by Mahoney *et al.* in 1992. On September 19, 1989, Louisiana officials were notified that three children from two families had hepatitis A virus. The families shared no common exposures except for a weekend camping trip to

Organism	Number Affected	Illness and Symptoms	Attack Rate	Duration of Illness	Probable Cause
Adenovirus Serotype 3 (AV–3)	26	Pharyngo–conjunctival fever. Majority of infected, pharyngitis, and conjunctivitis. Symptoms in adults were milder. Diarrhea was more common among children infected at home.	85% and 67% for each swim team.	2 to 8 days	Contamination of pool water due to inadequate chlorination. Chlorinator shut–off during early morning swim practice; thus there was exposure to unchlorinated water due to intermittent chlorination.
Adenovirus Serotype 3 (AV–3)	595 (681 visited camp infirmary)	Pharyngo–conjunctival fever. Illness included pharyn-gitis, cough, fever to 40°C, headache, myalgia, malaise, and conjunctivitis.	52%	Not specified	Inadequate chlorination. A concentrated sample of pond water drawn six feet below the surface yielded adenovirus serotype 3.
Adenovirus Serotype 3 (AV–3)	105	Conjunctivitis. Reddened conjunctivae affected only 3 – 4 out of 105 cases that occurred. Other swimmers developed sore throat, fever, headache, and anorexia (loss of appetite).	18%	2 to 7 days	Outbreak coincided with temporary defect in pool filtration system.
Adenovirus type 4 (Samples taken on day 1 and day 15 were positive for adenovirus type 4).	72	Pharyngo–conjunctival fever. Children who regularly used the pool and swim team members experienced fever and pharyngitis. Identified cases also showed signs of illness and symptoms that included sore throat, runny nose, chills, vomiting, earache, nausea, cough, swollen glands, and diarrhea.	17%	Not specified.	AV–4 was recovered from water specimens taken from the implicated pool.
Adenovirus Type 7	44	Acute Conjunctivitis Mainly limited to the eye (red or pink eye, swollen, painful, tearing). In some cases, fever, headache, coughing, nausea, earache, sore throat, vomiting, stomachache, diarrhea, and dizziness.	49% (18.8% for Swim team)	5.7 days	Failure of pool filter and chlorinator to maintain adequate chlorination level.

Table 6 – Swimming Pool Outbreaks Caused By Viral Infection

	Disinfection Rate	Pool Description	Year / Locale	Remarks
	Analysis of water samples after the outbreak revealed acceptable quality.	Public outdoor swimming pool, 75x35 ft.; depth of water, 3 to 10 ft.; Water volume: 111,500 gal plus 950 gal. in make–up tank. Entire water volume recirculated once every 8 hr.	1966 in Seattle	Epidemic occurred among two morning swim teams, while afternoon swim team remained well. In ten of 2 affected families, transmission to other members was demonstrated; secondary attack rate for children was 63%, for adults 19%. REF: Foy *et al.* (1968)
	Residual chlorine was not detectable. The pond was turbid and plants grew in the bottom of it.	One–acre man–made pond, with a maximum depth of 10 ft. Well water pumped into the pond. Water overflowed through a spillway into an adjacent river. An automatic chlorination system treated the water before it entered the pond.	1991 in North Carolina	Nearly 700 persons swam each day in the pond–type swimming facility. At the end of the second session, the pond was drained. REF: Anonymous. Medical News (1992); McMillan *et al.* (1992)
	Monitored twice daily but no records kept, prior to outbreak. Calcium hypochlorite added manually, twice daily.	Private recreation center swimming pool. Pool size not specified.	1977 in Dekalb County, GA.	Pool water had become turbid and then turned green due to algal overgrowth. Reports of ill swim team members began shortly afterwards. One of 4 flow regulating valves defective causing a backwash cycle. REF: Martone *et al.* (1980)
	Calcium hypochlorite was added by hand once or twice a day to maintain levels of free chlorine in excess of 0.4 mg/l of pool water.	Private outdoor recreational pool, with capacity of 412,500 liters. Pool water was circulated and cleansed by a rapid, pressure, sand–filter system.	1977 in Georgia.	This is probably the largest outbreak of Pharyngoconjuctival fever caused Adenovirus Type 4. REF: DeAngelo *et al.* (1979)
	Inadequate chlorination due to sump pump failure. This caused filter to become clogged, rendering the chlorinator inoperative.	Local junior high swimming pool, automatically chlorinated.	1977 in Kansas	Occurred to a Kansas community swim team. Transmission of adenoviruses by swimming pool water has been suspected in the past. The water was inadequately chlorinated because of equipment failure. REF: Caldwell *et al.* (1974)

Table 6 – Swimming Pool Outbreaks Caused By Viral Infection (cont.)

Organism	# Affected	Illness and Symptoms	Attack Rate	Duration	Probable Cause
Adenovirus type 7a	77	Pharyngitis. Predominately fever, sore throat, headache, abdominal pain, conjunctivitis, and diarrhea. Two persons were hospitalized with hydration and persistent vomiting.	25%	3 weeks	Swimming pool exposure was identified as the risk factor. Pool chlorinator had reportedly malfunctioned. Persons reporting swallowing pool water were more likely to have been ill. Attack rate increased with length of time in pool.

Organism	# Affected	Illness and Symptoms	Attack Rate	Probable Cause
Hepatitis A Virus (HAV)	56	Hepatitus A Clinical manifestations of hepatitus.	Highest rate was children, 5–9%.	Causative agent was probably HAV. Fecal contamination of the large swimming pool played a significant role in the outbreak.
Hepatitis A Virus (HAV)	20	Hepatitis A Acute hepatitis or jaundice. A case was defined as acute Hepatitis A.	6.4% (Highest for 5 – 9 yr. old)	The pools may have been contaminated either by fecal contamination by one of the swimmers or by cross–connection while the sewage during routine pool maintenance. There was evidence of diapered children in the pool. Pools may have been contaminated on a hot afternoon when many people were swimming. Highest attack rates were found for those who used the Jacuzzi.
Norwalk virus	229	Norwalk gastroenteritis. Vomiting (86%) and cramping (70%), but more than half of the persons involved also reported having nausea, diarrhea, and headache. Similar illness frequently followed in household members (29%) of families with primary cases.	32% (Those who swallowed water, 71%)	Contaminated pool water during period when pool chlorinator was inadvertently unconnected. Person–to–person spread of illness followed. The infectious agent responsible for all cases was Norwalk agent or a closely related virus.
Norwalk virus (assumed). Symptoms suggest infection w/ Norwalk agent, rotavirus, or some other similar virus.	48	Gastroenteritis. Symptoms included nausea, vomiting, abdominal cramps, diarrhea, fever, headache, gas, and dizziness.	27%	Lack of adequate chlorination. Records showed a lapse of chlorine addition to the spa, resulting from chlorine residuals of 0.0 mg/l.. Similar findings were observed for the swimming pool on three separate occasions.

Table 6 – Swimming Pool Outbreaks Caused By Viral Infection (cont.)

	Disinfection Rate	Pool Description	Year/Loc.	Remarks
	Automatic chlorinator system malfunctioned. Pool was chlorinated by hand. No records kept, but pool personnel said that chlorine levels had ranged from 0.5 to 1.5 ppm, with lowest levels at peak hours of use.	Community swimming pool equipped with an automatic chlorination and pH regulator system.	1921 in Cordell, OK	Of approximately 400 cases of infection that occurred in Cordell (population 4500), more than two–thirds had exposure to the swimming pool. However, the outbreak resolved within 3 weeks upon proper operation of the chlorination system. REF: Turner *et al.* (1987)
	Disinfection Rate	**Pool Description**	**Year/Loc.**	**Remarks**
	No maintenance during 4 days it took to fill the large pool. Visitors used the half–filled pool.	Campsite with 3 separate pools of thermal water supplied by hot and cold springs.	1979 in Northeastern Hungary.	All patients were visitors who had used the thermal spa and predominately the large pool. REF: Solt *et al.* (1994)
	Not determined. Increased water temperature may have depleted available free chlorine.	Public section of campground contained two adult sized pools, a non–heated Jacuzzi, and a wading pool that all campers could use. The members–only section had a private pool of their own.	1989 at a campground in Louisiana.	The adult pool filtering system had a design flaw that could cause a cross–connection between a sewage line and pool water intake. A simple air gap on the sewage line draining the pool could prevent inadvertent cross–connection. Among 822 campers, 20 developed hepatitis A during same weekend. REF: Mahoney *et al.* (1991)
	Inadequate chlorination or none at all. How the swimming pool became contaminated remained uncertain.	Outdoor swimming pool that had been filled 2 days before, after being drained during the previous winter.	1977 in Kettering, OH.	Four classes of school children held an all–day picnic at a nearby church. Next day two more classes arrived, but subsequent outings were canceled when illness was reported. Primary cases involved 112 students and teachers, as well as 117 family members. No more cases were reported after the pool was drained, cleaned, and reopened.
	No problem in the swimming pool's chlorination system were found. Motel records revealed the chlorine tablets were not added to the spa. Main pool indicated free chlorine residual of 0.0 mg/l.	Motel swimming pool, wading pool, and spa. The swimming pool shared the same filtration system. The spa was 7 ft. In diameter and 3 ft. deep, with a hydrotherapy pump. Chlorine tablets were added daily.	1987 in Williston, ND.	All infected persons stayed in the same motel to participate in a hockey tournament. Contaminated pool water indicated the whirlpool spa was the culprit. The difficulty of enforcing (and the lack of) regulations pertaining to number of persons allowed to use a pool of a given size at a given time, swimming pool– and spa–related gastroenteritis may be an unrecognized, relatively common public health problem. REF: Holmes *et al.* (1989)

Table 6 – Swimming Pool Outbreaks Caused By Viral Infection (cont.)

Organism	# Affected	Illness and Symptoms	Attack Rate	Probable Cause
*Non–polio Enterovirus (suspected, not identified; viral studies not available)	26	Enteroviral infection. Symptoms consisted of diarrhea, nausea, fever, headache, stomach ache, and malaise (discomfort). No one was hospitalized, and all symptoms resolved an average of 5.7 days after onset of the illness.	41%	Lack of disinfectant due to a faulty gas chlorination system. Pool personnel had manually chlorinated the pool. However, the pool remained closed until adequate repairs were made. Strong association between swimming in the wading pool and illness suggests that an infectious agent associated with the wading pool caused the outbreak.

Table 6 – Swimming Pool Outbreaks Caused By Viral Infection (cont.)

a commercial campground during early August. The campground was open to the public and also had a membership clientele. The public section of the campground contained two adult–size pools, a non–heated Jacuzzi, and a wading pool that all campers could use. There was also a members–only pool in another section of the campground.

All case–patients reported swimming in one or more of the campground's five swimming pools. Among children under 18 who swam in any one of the pools, the attack rate was five percent. The highest attack rates were found for those who swam in the Jacuzzi pool (7.7%) and those who swam in one or more of the three adult pools (5.3%).

All campsites received running water from a single source. The campground water was supplied from a well and was not chlorinated; it was tested and found to be free of fecal coliforms on September 21 and 27. The pools may have been contaminated either by fecal contamination from one of the swimmers or by cross–connection with the sewage. Several people reported that children wearing diapers were allowed to swim in the pools on the weekend in question. The management at the campground reported that fecal contamination of the pools by swimmers was not uncommon. There was no specific pool maintenance policy for personnel to follow when this type of contamination occurred. It was not determined if this had occurred on the weekend in question.

The unusual design of the plumbing suggests that contamination could have occurred via mixture of raw sewage with pool water during routine pool maintenance procedures. A simple air gap on the sewage line draining the pool could have prevented this type of cross–connection.

Norwalk Virus

• Kappus *et al.* (1982) investigated an outbreak of Norwalk gastroenteritis associated with swimming in a pool and secondary person–to–person transmission. In early June 1977, after successive all–day outings attended by groups of elementary classes from a school near Dayton, Ohio, many of the children and their adult chaperons developed illness characterized predominantly by vomiting and diarrhea. Four classes held an all–day picnic on the grounds of a nearby church, which included swimming in a pool that had been filled two days before, after being drained the previous winter.

This outbreak of acute gastroenteritis typically lasted 24 hours or less and was characterized by vomiting (86%) and cramping (70%), but more than half of the persons involved also reported having nausea, diarrhea, and headache. Similar illness frequently followed in household members (29%) of families with primary cases. Investigation revealed that 70% of the children and teachers who swam in a pool June 1 (4 classrooms) and 55% of those who swam during a similar outing June 2 (2 classrooms) had the onset of acute illness from 12–48 hours later. None of the children who attended the outings but did not swim had a similar illness. The evidence suggested that the primary outbreak was caused by contaminated water in the pool and that person–to–person spread of illness followed. Results of a microbiologic study of pool water were negative for bacterial and viral pathogens. Throat washings, stool specimens, and paired blood samples studied for evidence of pathogens were negative initially, but subsequent serologic studies suggested that infection by Norwalk virus was the cause of the outbreak. The pool chlorinator which was inadvertently unconnected at the time of the school visits was reconnected and an underground leak in the water supply pipes was corrected. No more cases were reported after the pool was drained, cleaned, and reopened.

The attack rate in swimmers from June 1 (70%) was not significantly higher (55%) for those who swam on June 2. The overall attack rate for swimmers who reported swallowing water was significantly higher (71%) for those who did not swallow (50%).

	Disinfection Rate	Pool Description	Year/Loc.	Remarks
	Pool readings were zero for free chlorine during 5 days of the onset and a low of 0.1 mf/l for one day. Attack rate for swimmers who used the outdoor wading pool was 62% as compared to those who did not (12%).	An Olympic size indoor pool and an outdoor wading pool. Both equipped with high–rate sand filters and automatic–feed gas chlorination disinfection systems. The huge indoor pool capacity was 250,000 gal.	1987 in Longmont, CO	The main indoor pool had been operating properly. The outdoor wading pool had to be closed due to violations of water quality deficiencies of extremely high turbidity, temperature of 33.3°C (maximum allowable 28.9°C, 84° F), pH od <6.8 (maximum allowable, 7.2), and an inoperative flow meter which prevented calculation of pool water "turnover". REF: Lenaway *et al.* (1989)

Table 6 – Swimming Pool Outbreaks Caused By Viral Infection (cont.)

A case of acute illness was defined to be with either vomiting or diarrhea or with at least two of the following: fever, abdominal cramping, or nausea. A representative case history might involve a child waking in the night with explosive vomiting. Frequently, fever and abdominal cramping developed simultaneously or soon after the vomiting began. Typically, the symptoms would diminish over a period of several hours with complete recovery the next day. Many cases were less incapacitating, perhaps only involving fever and mild abdominal cramping. None of those affected were hospitalized, and all were able to resume normal activity within a few days. Symptoms were similar in school and household cases. According to the case definition, there were 112 cases in the fourth and fifth level home rooms; 117 family members also met case definition.

How the swimming pool became contaminated remains uncertain. The infectious inoculum may have aspirated into the pool from the standpipe when the pool was filled, or may have been deposited by an infected swimmer who did not report being ill.

• Epidemic gastroenteritis associated with swimming pool use is not commonly reported in the medical literature. It is likely then, that swimming pool–related outbreaks of gastroenteritis suffer even greater under–reporting for the following reasons: persons involved in an outbreak may be from diverse geographical areas, public health authorities may not look beyond food and water supplies as potential sources of gastroenteritis, and the public is not aware of the potential for the transmission of gastrointestinal disease–causing agents by swimming pools. Such an outbreak identified by the North Dakota Department of Health was believed to be caused by a pool and/or hot tub at a motel. The health department had become aware of the outbreak because a number of pee–wee hockey teams from various towns stayed at the same motel. Of the 48 persons identified as ill, 70% developed diarrhea and 85% vomited. All of the ill persons had visited the pool at the motel. Duration of the illness ranged from 12 to 48 hours. Incubation for the outbreak was estimated to range from 24 to 56 hours. The symptoms, incubation period, and duration are very suggestive of a Norwalk virus–like etiology. Bacterial gastroenteritis typically have longer incubation periods and longer duration of symptoms.

The swimming pool was 20 x 40 ft, with an average depth of 5.5 ft. An adjacent wading pool was 6 ft in diameter with a depth of 1.5 ft. These two pools were served by the same continuous chlorination and filtration systems. Filtration was accomplished with a pressure–type diatomaceous earth filter with a turnover rate of six hours. The spa was 7 feet in diameter and 3 feet deep, with a hydrotherapy air pump providing agitation. The spa used a basket–chlorine tablet method for disinfection. Chlorine tablets must be added daily to this system. Filtration was accomplished by a high rate sand filter with a turnover rate of 30 minutes. At the time of the investigative inspection on February 3, the swimming pool and wading pool both had free chlorine residuals of 0.7 mg/l, pH readings of 7.7, and water temperature readings of 35.9°C (96.7°F), the spa had a free chlorine residual of 0.0 mg/l, a pH reading of 7.5, and a water temperature of 38.9°C (102°F).

Free chlorine residual and pH were tested and recorded by motel employees each morning and evening. A review of the motel records revealed that chlorine tablets were not added to the spa on January 30. Recorded free chlorine residual levels in the spa were 0.0 mg/l from the evening of January 30 to the evening of February 1. Chlorine tablets were added to the spa on January 31 and February 1–3. The main pool had a recorded free chlorine residual of 0.0 mg/l on the morning of January 30 and the evening of the 31st.

The swimming pool records also revealed free chlorine residuals of 0.0 mg/l at several testing times on January 30, 31, and February 1. However, no problems in the swimming pool's chlorination system were

discovered during the pool inspection and previous monthly bacteriological testing was well within acceptable limits.

Assuming there was not a failure in the swimming pool's chlorination system, pool overloading could be hypothesized as a contributing factor. Anecdotal reports indicated extremely heavy pool use, especially Saturday evening. Evidence of gross bacterial contamination of the swimming pool and spa are the Standard Plate Counts of greater than 6000 per ml. Coliform contamination of the spa also was confirmed. Of note, these samples were drawn on February 3, two days after the last period of high use. The extent of the contamination during periods of high use probably was much greater.

Enteroviruses

Enteroviral disease associated with swimming pools have been only suggestive and few studies have been reported in the literature. An outbreak of aseptic meningitis in children in Berlin where echovirus 30 was isolated from most of these cases. It is believed that the water in outdoor swimming pools was the source of the virus for these children. Another report from Germany also incriminates an open–air swimming pool in an outbreak of aseptic meningitis and encephalitis due to coxsackie virus B3. The same virus was isolated from 20% of the water samples from the pool. Application of 0.3–0.5 mg/l of chlorine to the pool water was insufficient in meeting the hygienic standards.

• On June 24, 1987, the Boulder County Health Department received phone calls from concerned parents regarding an illness among children who had participated in a swim class at a local municipal pool in Longmont, CO, prompting an investigation. Of the 63 children in the swim class, 26 became ill. A case was defined as a child with fever greater than 38.3°C (101°F), and at least one of the following: malaise, headache, stomach ache, nausea, or diarrhea. All symptoms resolved three to seven days after onset. None of those affected were hospitalized and all were able to resume normal activity within a few days. All of the children reported swimming in the main indoor pool in conjunction with the scheduled swim class. However, the attack rate for swimmers who used the outdoor wading pool was 62% compared to 12% for those who did not use the wading pool. The Longmont municipal pool complex houses an indoor 50,000–gallon Olympic–size pool and an outdoor 20,000–gallon wading pool. Both pools are equipped with high–rate sand filters and automatic feed gas–chlorination disinfection systems. Pool personnel manually monitored the pool chemistry and performed manual adjustments to the chlorination system as necessary.

Inspection of the community pool complex showed the main indoor pool to be operating properly. The outdoor wading pool, however, had to be closed. Violations included water quality deficiencies of extremely high turbidity, temperature of 33.3°C (92°F) [maximum allowed 28.9°C (84°F)], pH of <6.8 (minimum allowed, 7.2), and an inoperative flow meter which prevented the calculation of pool water "turnover." Review of the daily water chemistry log for the period June 8–19 indicated "0" free chlorine readings for five days, no readings taken for six days, and a low disinfection level of 0.1 mg/l free chlorine on one day. The lack of chlorine disinfectant was due to a faulty gas chlorination system. Pool personnel had manually chlorinated the pool; on the day of inspection, the level was >3 mg/l. The pool remained closed until adequate repairs were made.

The lack of chlorination and the strong association between swimming in the wading pool and illness suggest that an infectious agent associated with the wading pool caused the outbreak. The clinical manifestations and course of the illness, estimated incubation time, potential for a fecal–oral route of transmission, and the exclusion of likely bacterial pathogens all suggest a non–polio enterovirus as a likely etiological agent.

Discussion

The source of virus in recreational pools is swimmers, and in wading pools is almost exclusively children. Viruses can enter the pool through fecal material and nasal and pharyngal secretions (Keswick *et al.* 1981).

The association between use of swimming pools and illness has been long recognized. When maintained at proper levels, chlorine can inactivate or control most pathogens in pool water.

The total of 11 documented outbreaks reported herein involves 1,745 cases of viral infection; at least one–half reached epidemic proportions. The outbreaks are summarized in Table 6. Prevention requires adequate disinfection of pool water.

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