

CHILDREN'S HOSPITAL PEDIATRIC ENVIRONMENTAL  
HEALTH CENTER

## Reactive Airways Dysfunction and Systemic Complaints after Mass Exposure to Bromine

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Occasionally children are the victims of mass poisoning from an environmental contaminant that occurs due to an unexpected common point source of exposure. In many cases the contaminant is a widely used chemical generally considered to be safe. In the following case, members of a sports team visiting a community for an athletic event were exposed to chemicals while staying at a local motel. Bromine-based sanitizing agents and other chemicals such as hydrochloric acid, which were used in excess in the motel's swimming pool, may have accounted for symptoms experienced by the boy reported here and at least 16 other adolescents. Samples of pool water contained excess bromine (8.2  $\mu\text{g}/\text{mL}$ ; ideal pool bromine concentration is 2–4  $\mu\text{g}/\text{mL}$ ). Symptoms and signs attributable to bromine toxicity included irritative skin rashes; eye, nose, and throat irritation; bronchospasm; reduced exercise tolerance; fatigue; headache; gastrointestinal disturbances; and myalgias. While most of the victims recovered within a few days, the index case and several other adolescents had persistent or recurrent symptoms lasting weeks to months after the exposure. *Key words:* bromine, children, mucous membrane irritation, respiratory, swimming pool sanitizers. *Environ Health Perspect* 107:507–509 (1999). [Online 10 May 1999]  
<http://ehpnet1.niehs.nih.gov/docs/1999/107p507-509woolf/abstract.html>

### Case Report

A 12.5-year-old male seventh grade student stayed in a motel with at least 16 other members of a youth hockey team while attending a regional tournament. The players arrived on Friday, 8 March 1996, before the weekend events began. Soon after arriving, many of the hockey players, as well as their siblings and parents, visited the motel swimming pool. The families reportedly spent as many as 3 hr poolside or in the pool both before and after dinner. The bathers noticed at the time that the swimming pool was "foamy" and was covered with layers of a froth-like substance. The foam was so thick that some of the children were playing with it and throwing it at each other.

Later that same evening the patient complained of periorbital redness and skin irritation. His parents noted a periorbital rash, "bloodshot" eyes, and facial edema. The following day he had a generalized, erythematous, pruritic rash involving all parts of his body. These symptoms were accompanied by sore throat, headache, chills, cough, chest tightness, and difficulty breathing. He was seen in a local emergency department where he was provided symptomatic relief for what was felt to be a viral illness. The family later discovered that five children were seen in the emergency department the night before

for pruritic rash and severe eye redness and irritation. The local fire department was asked to investigate the motel swimming pool. An evaluation of the site quickly uncovered an error in how the pool had been sanitized by a new employee. Their analysis, which included retrieval of material safety data sheets on the chemicals used in the swimming pool maintenance, revealed the primary agent to be Sani-Brom (sodium bromide; Ameribrom Inc., New York, NY), a bromine-based disinfectant. Other agents were Brom-A-Gard (1-bromo-3-chloro-5,5-dimethylhydantoin; York Chemical Corporation, Irving, TX) and Dicalite (Grefco, Inc., Torrance, CA), a flux-calcined diatomaceous earth. Additional chemicals reportedly used to sanitize the pool were muriatic acid, xylene, and toluene. An analysis of the water revealed a pH of 8.8 and a bromine concentration of 8.2  $\mu\text{g}/\text{mL}$  (ideal is 2–4  $\mu\text{g}/\text{mL}$ ). The pool was ordered closed by the local fire chief.

The patient continued to have nausea, headache, and generalized itching. He saw his pediatrician 2 days later and was prescribed diphenhydramine, which offered no relief; he continued to complain of eye and mucous membrane irritation. He was referred to the Children's Hospital Pediatric

Environmental Health Center (PEHC) for further evaluation 3 weeks after the exposure. At the initial visit, ongoing symptomatic complaints consisted of intense itching, evanescent blotchy skin rashes, eye irritation, chest tightness, coughing, and headaches. In the interval between the exposure and the clinic visit, vague neck pains, generalized malaise, and reduced stamina with shortness of breath during soccer practice were also noted. There was no family history of reactive airway disease. Physical examination revealed a lean white male who was alert and cooperative. Conjunctivae were clear. A malar rash was noted. Chest auscultation disclosed no significant findings.

Laboratory evaluation included a chest X ray, electrocardiogram, pulmonary function tests (Table 1), complete blood count, renal function tests, and serum immunoglobulins. The chest X ray and electrocardiogram were normal. Pulmonary function tests, including a cold-air challenge test, were performed. During this test, the patient developed chest discomfort associated with a mild decline in pulmonary function. This was immediately relieved by albuterol nebulization therapy; repeat spirometric performance returned to baseline. Laboratory findings included a normal blood count [hemoglobin 12.9 g/dL; hematocrit 36.2%; total white blood cell count 8,590 (57% neutrophils, 33% lymphocytes, 5% monocytes, 3% eosinophils)]; and 261,000 platelets/ $\text{mm}^3$ . The urinalysis was normal. The blood urea nitrogen (BUN) concentration was 14 mg/dL, with 0.5 mg/dL serum creatinine. Serum immunoglobulins were as follows: IgG, 1,018 mg/dL; IgA 136 mg/dL; IgM, 101 mg/dL; and IgE, 69 U/mL (all within normal limits).

The assessment was that the child had the new onset of reversible bronchospasm.

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Received 25 March 1999; accepted 26 March 1999.

**Table 1.** Pulmonary function test results.

Date	27 March 1996	29 May 1996
TLC <sup>a</sup> (L)	3.34	3.03
Percent predicted	93	81
RV (L)	0.82	0.86
Percent predicted	99	71
FVC (L)	2.52	2.42
Percent predicted	96	88
FEV <sub>1</sub> (L)	2.35	2.18
Percent predicted	105	94
FEV <sub>1</sub> /FVC	0.93	0.90
PFR (L/min)	6.08	6.33
Percent predicted	103	104
MMFR (L/min)	3.41	2.77
Percent predicted	132	103

Abbreviations: FEV<sub>1</sub>, forced expiratory volume in 1 sec; FVC, forced vital capacity; MMFR, maximum midflow rate; PFR, peak flow rate; RV, residual volume; TLC, total lung capacity.

<sup>a</sup>TLCs were determined with a body plethysmograph.

Pulmonary consultants recommended treatment with inhaled bronchodilators and corticosteroids (albuterol and flunisolide). Nedocromil sodium was later added to his maintenance therapy. Pruritus and rash gradually resolved over the subsequent 2 weeks, but chest pain and tightness, shortness of breath, and fatigue persisted. He continued to use bronchodilators regularly and had fewer respiratory problems and better exercise tolerance by 8 weeks after the exposure. When seen in follow-up 8 weeks postexposure, repeat pulmonary function tests revealed improved performance and no abnormal response to a cold air challenge. Most of his other complaints had resolved, although he continued to complain of headaches, which were responsive to acetaminophen. Further evaluation revealed a frontal sinusitis for which he was prescribed a steroid nasal spray. He used the spray intermittently for the next 30 months, but continued to have four to five sinus headaches monthly. At the last evaluation in the PEHC, these had resolved and he was functioning well in the 9th grade, obtaining good grades and engaging in a range of sports activities, with normal exercise tolerance and no apparent residual effects.

Overall, 14 adolescents were evaluated in the PEHC within 1 month of the index patient for illness that developed immediately after swimming in the motel swimming pool. The most common complaints were eye/mucous membrane irritation and rashes that developed within 24 hr of swimming in the pool and persistent complaints of pruritus, throat and mucous membrane irritation, eye irritation, photophobia, gastrointestinal disturbances, cough and chest pain, headache, myalgias, fatigue, and reduced exercise tolerance. While some patients had

resolution of symptoms within days of the incident, others had complaints lasting weeks to months thereafter.

## Discussion

Swimming pools, spas, and hot tubs are easily subject to contamination. It is only through their regular sanitation by disinfectant solutions that they can be kept safe for use. State and local public health regulations provide guidance for the maintenance of safe and comfortable conditions with respect to water chemistry, clarity, temperature, lighting, bather load, filtration and circulation standards, bacterial counts, and supervision (1). Swimming pools and all forms of hydrotherapy should be routinely sanitized so that bacteria and other pathogens are not transmitted between people using them. To accomplish this, chemical oxidizers such as chlorine, ozone, or bromine are commonly added in concentrations sufficient to be antimicrobial, but without human toxicity, particularly irritation, due to their own irritative properties. In the case of chlorine, for example, granular chlorine and muriatic acid are added to swimming pools in a quantity that achieves a free chlorine concentration of 1–2 ppm. Other compounds, e.g., cyanuric acid, may also be added to stabilize free chlorine, preventing its dissipation in sunlight (1). The ratio of free and combined chlorine, accomplished by buffering water to neutral pH, must be maintained for maximal bactericidal action.

Recently, bromine and related chemicals have replaced chlorine as swimming pool sanitizers in some outdoor pools because of their lower cost and greater effectiveness against *Pseudomonas*. However, bromine has greater solubility than chlorine and may also be more irritating to the eyes, throat, and skin (2). When used to sanitize pools, the recommended concentration of chlorine and bromine is 1.0–1.5 µg/mL at pH 7.2–7.8 (1,3). Although ozone also has suitable antimicrobial action, it is usually an inadequate sanitizer when used alone in hydrotherapy pools and requires the addition of halogenated compounds to suppress a variety of bacteria (1).

Both halogens, chlorine and bromine, are widespread in the earth's crust. Bromine is found naturally in seawater, in concentrations of approximately 65 ppm (4), and can also be present in smaller amounts in well water. Chlorine is a yellowish-green liquid that is quite volatile. Bromine is a reddish-brown volatile liquid. Whereas it has no known physiologic use, serum bromine concentrations measured in 30 nonoccupationally exposed adults ranged from 6 to 12.5 mg/L (4). Another study of adults

found bromine concentrations in serum of 3.2–5.6 mg/L, with concentrations in urine of 0.3–7.0 mg/L in 10 adults (5). There are no reference values for bromine concentrations in the serum or urine of a pediatric population.

Like chlorine, bromine is a versatile element with potent oxidizing properties. It has found great utility for industrial and commercial purposes. Industrially bromine is used in the manufacture of petrochemicals (including the gasoline additive dibromomethane), fire retardants, and agricultural chemicals. It is also used in the manufacture of paper, dyes, and photographic film (6). In the occupational setting, bromine has a threshold limit value (TLV) of 0.1 ppm and a short-term exposure limit (STEL) of 0.3 ppm.

Bromine compounds are potent chemical irritants. Documented occupational exposures consistently identify eye and nose inflammation, sore throat, chest tightness, bronchospasm, blepharospasm, and dermatitis as common health consequences. In high concentrations bromine can be corrosive to the skin; inhalation may lead to epistaxis, dizziness, headaches, upper airway edema, and pulmonary edema. Pneumomediastinum, complicated by forceful coughing and transient respiratory obstruction, has been reported (7). Carel (8) reported the death of a truck driver whose truck was loaded with bottles filled with bromine; the driver became trapped after his cab overturned in a motor vehicle crash. The exploding bottles enveloped the truck in a cloud of bromine gas, and the victim died within 3 hr of exposure. Six people who attempted rescue without proper protective equipment suffered a variety of health effects including skin burns, respiratory complaints, and headaches.

There are several reports of illness in adults after mass exposures to bromine. Morabia et al. (9) describe the consequences of a liquid bromine spill in a chemical plant in Geneva, Switzerland, in 1984, in which part of the gaseous cloud escaped through the ventilation system and wafted over the adjacent neighborhood at ambient levels of 0.2–0.5 ppm. They reported the symptoms of 91 patients who were seen at local emergency departments. Most had eye and upper airway irritation, cough, expectoration, and/or headache lasting only for a few hours; none of the patients had persistent symptoms when followed up more than 4 weeks after the incident (9).

In a mass exposure at a public school where the ventilating and air conditioning systems were sprayed with Microban disinfectant (a quarternary ammonium complex combined with 0.04% bromine), 44



exposed staff members reported more symptoms of eye and throat irritation, nausea, dizziness, headaches, cough, and runny nose than did 15 unexposed staff (10). Interestingly, although the toxic exposure occurred in a public elementary school, children were not included in the survey, and their complaints were not reported in the analysis of the adverse health effects attributable to the disinfectant.

The skin-irritating effect of the bromine compound 1-bromo-3-chloro-5,5-dimethylhydantoin, which is frequently used in swimming pools, has been described as "spa pool dermatitis" (or "bromine rash"). The pruritic rash tends to be eczematoid and can be exacerbated by repeated bathing in brominated water. In select individuals, it appears to be an allergic contact sensitivity with a positive skin patch test (11).

Bromine and inorganic brominated compounds have also been shown to be capable of causing lingering health effects. In one report of six cases of bromine exposure, persistent complaints of health effects were evident 6–8 weeks after the exposure, despite scant physical or laboratory findings. Patients continued to complain of eye irritation, respiratory complaints (chest pain, cough, expectoration, shortness of breath), nervous system complaints (headache, dizziness, fatigue, memory disturbances), gastrointestinal complaints (abdominal pain, diarrhea, constipation), pruritis, and rashes (8). In another case of occupational exposure to inorganic bromine compounds (including hydrogen bromide) in a laboratory explosion, the victim developed a

chemical pneumonitis that incompletely resolved; follow-up 9 months later revealed continued shortness of breath and dyspnea on exertion, as well as subnormal pulmonary function test results, with abnormalities in total lung capacity and diffusing capacity (2).

In the swimming pool-related exposures reported here, the children and adolescents were more affected than the parents, indicating disproportionate toxicity. This exemplifies the principle of unique susceptibilities in children as compared with adults after environmental exposures. The risk of exaggerated toxicity in children results from physiologic factors including smaller body mass (resulting in a higher milligram per kilogram exposure). In the case of inhalational exposures, the dose absorbed is accentuated by the higher resting ventilatory rate of young children. After the disaster in Bhopal, India, involving the inhalation of a toxic cloud of methyl isocyanate, there were at least 119 documented childhood deaths and 1,337 pediatric admissions to the local hospital for primarily respiratory, gastrointestinal, and nervous system complaints (12).

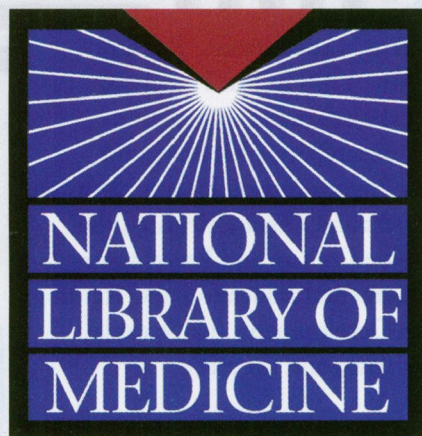
The constellation of health complaints experienced by this child and his teammates was similar to the profile of toxic effects that brominated compounds have been reported to cause. In previously reported cases of acute bromine exposure, complaints of "not feeling right" persisted for months after the acute event, even though no abnormalities were discernible on physical examination or laboratory testing. Likewise in the case described here,

reports of persistent, evanescent rashes, respiratory complaints, the new onset of allergies, behavioral changes, and a loss of exercise tolerance only gradually resolved after 12–24 months of follow-up.

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