Another Hot Tub Hazard: Toxicity Secondary to Bromine and Hydrobromic Acid Exposure

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and change the filters. This may be all that is necessary to prevent the buildup of M. avium complex in hot tubs. M. avium complex is resistant to chlorination and so it is found in domestic water. The high chlorine concentrations needed for sterilization may be unpleasant for hot tub use. Superheating the water, if possible, to 70°C for up to an hour may help. If our findings prove to occur more frequently in hot tubs, then a study of how to control the growth of M. avium complex must be done.

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Another Hot Tub Hazard*

Toxicity Secondary to Bromine and Hydrobromic Acid Exposure

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We describe the clinical course of two patients who developed acute pneumonitis following by reactive

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airways dysfunction syndrome after bathing in a hot tub. Additional findings were present and suggested that exposure to a corrosive agent was responsible. Bromine and hydrobromic acid generated from a widely used water disinfectant were implicated as the underlying cause. Physicians should be alert to the possibility that such exposures may initiate or exacerbate inflammatory pulmonary disease.

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Key words: bromine; hot tub; hydrobromic acid; RADS

Abbreviations: Br2=aqueous or gaseous bromine; HBr=hydrobromic acid; HOBr=hypobromous acid; HOCl=hypochlorous acid; RADS=reactive airways dysfunction syndrome

Hot tub bathing is a popular recreational activity. Although generally considered safe, public facilities usually have signs warning against hot tub use by individuals who are pregnant, take certain medicines, or have diabetes or cardiovascular disease. A variety of medical problems have been linked to hot tub bathing.1-3 Except for hypersensitivity pneumonitis due to the fungus, Cladosporium, however, pulmonary complications have not been described previously (to our knowledge).4 We describe two patients who developed signs and symptoms of acute bromine toxicity followed by reactive airways dysfunction syndrome (RADS) after bathing in a hot tub and discuss the pathogenesis and pathophysiology of their disease.

Case Reports

Case 1

A 42-year-old woman presented with burning eyes, chest, and throat, shortness of breath, and wheezing that began during a 10-min bath in a hot tub 2 h earlier. She reported a strong odor of “bleach” in the hot tub area, a glass-enclosed room measuring 8 x 11 feet, located inside a local health club. Black discoloration of her gold jewelry and green bathing suit was also noted. She had been in good health previously and did not smoke. Mild inspiratory stridor, inspiratory wheezing, and a bleach-like odor to the breath were noted on examination. Vital signs were normal and the eyes, nose, throat, and skin were unremarkable. Oxygen saturation was 98% on room air. Chest radiograph and results of routine laboratory analyses were unremarkable. She was treated with humidified oxygen and nebulized albuterol during a 24-h hospitalization.

She reported intermittent rectal bleeding and generalized alopecia during the following week and month, respectively. Dyspnea and chest burning continued to occur on exertion and exposure to cold air and strong odors. Symptoms respond to inhaled albuterol. Pulmonary function tests performed 3 and 10 months after exposure revealed normal results of spirometry and lung volumes with a strongly positive methacholine challenge. The provocative doses required to produce a 20% fall in FEV1 were 0.008 and 0.007 µmol, respectively. An α1-antitrypsin level was normal. Flexible sigmoidoscopy performed 13 months after her exposure revealed nonspecific proctitis.

Case 2

A 32-year-old woman presented with burning eyes, throat, and chest; hoarseness; nonproductive cough; and mild dyspnea that developed during a single 5-min bath in the same hot tub at the same time as patient 1. She, too, reported a strong acidic odor while bathing in the hot tub. She was previously healthy and did not smoke. Results of physical examination were normal. Oxygen saturation was 99% on room air. Chest radiograph and results of arterial blood gas analysis were normal. No treatment was administered. Cough, sore throat, and hoarseness persisted for 7 months. She continues to have chest burning with exertion. Pulmonary function tests performed 10 months after exposure revealed normal lung volumes and normal results of spirometry but a strongly positive methacholine challenge (provocative dose required to produce a 20% fall in FEV1, 0.447 µmol).

Discussion

Clinical manifestations and the temporal course of events suggested exposure to an irritant chemical in the hot tub water and atmosphere as the etiology of our patients’ signs and symptoms. Bleach (hypochlorous acid, HOCl) or a chlorine-based disinfectant was initially suspected. Investigation, however, revealed that a bromine-based agent was used in the hot tub in question.

Three brominating compounds are available for recreational hot tub, spa, and swimming pool sanitation: elemental bromine, sodium bromide/monopotassium persulfate, and bromochlorodimethylhydantoin. All three compounds generate aqueous hypobromous acid (HOBr), the primary bacterial agent, when dissolved in water.

In the present incident, 1-bromo-3-chloro-5,5-dimethylhydantoin (“Brom Tabs,” “Brominating Tablets”) was identified as the brominating agent. When added to pool water, this compound hydrolyzes to HOBr and HOCI.5 Bromide ions are subsequently generated when HOBr reacts with microorganisms. In an acidic pH, as occurs at high concentrations, bromide also exists as hydrobromic acid (HBr). Unless the pH is kept relatively neutral (eg, by adding potassium carbonate), these two compounds readily react to form aqueous bromine (Br2). The subsequent liberation of Br2 (and possibly HBr) vapors increases as water temperatures increase. Chlorine gas is not generated because HOCl reacts with bromide ions resulting in the reformation of HOBr.5

Pool water bromination is achieved similarly with the sodium bromide/monopotassium persulfate method. When these compounds are added to water, bromide ions are oxidized by monopotassium persulfate to HOBr.5 Subsequent reactions are the same. Aqueous Br2 concentrations between 2 and 4 ppm are recommended for residential spas and 4 to 6 ppm for commercial spas.5 Water pH should be between 7.2 and 7.6 optimally. Br2 and HBr liquids and their vapors are skin and mucous membrane irritants. The extent of tissue injury is dependent on their concentration, duration of contact, and water content of exposed tissue. Pathologic effects include edema, inflammation, and necrosis. Exposure to atmospheric concentrations of 10 ppm of Br2 is considered immediately dangerous to life and 0.3 ppm is the current short-term (15-min) exposure limit.6 Human volunteers could not tolerate a Br2 concentration of 0.9 ppm for greater than 5 min.7 Br2 is toxic to three times more corrosive than HBr. Nasal irritation occurs at HBr vapor concentrations of 5 to 6 ppm.8

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Like chlorine, the major cytotoxic effects of Br₂ are presumed to be secondary to the oxidizing effects of nascent oxygen. Br₂ reacts rapidly with tissue water to form HBr and HOBr. HOBr may subsequently break down into HBr and oxygen free radicals. The corrosive action of the acids generated and reaction of Br₂ and these acids with cellular proteins to form bromamine and thiol radicals may contribute to toxicity. Greater water solubility, and, thus, tissue permeability may explain why bromine is more toxic than chlorine.

Clinical manifestations usually begin immediately after vapor inhalation but may be delayed for up to 10 h. Generally, low-concentration exposures will result in eye, nose, and upper airway injury and higher concentrations will additionally result in lower airway damage. Sequelae are infrequent and usually consist of subjective complaints without objective findings to correlate with symptoms.

One patient developed chemical pneumonitis and a restrictive pattern of pulmonary dysfunction following short-term exposure to a mixture of HBr and phosphorus tribromide. Persistent bronchial spasm has been observed in animals exposed to Br₂ vapors but not in humans.

Both patients described fulfill the criteria for RADS. The abrupt onset of irritant symptoms within minutes of a single bath suggest high-concentration irritant exposure. Circumstances strongly implicate exposure to bromine and HBr as the precipitating event. Allergic asthma (hypersensitivity pneumonitis) was not considered likely because it is typically insidious in onset, requiring repetitive exposures to elicit an inflammatory reaction. Even with acute hypersensitivity pneumonitis, symptoms do not develop until 4 to 6 h after exposure. In addition, patients with hypersensitivity pneumonitis typically have constitutional symptoms, abnormal chest radiographs, and restrictive pulmonary disease, findings that were not seen in our patients.

The pathophysiologic state of RADS has not been fully elucidated. Nonspecific bronchial hyperresponsiveness is operative and may be mediated by structural airway abnormalities, immunogenic inflammation, and dysregulation of neurogenic inflammation. Bronchial biopsy specimens from patients with RADS have revealed epithelial destruction, squamous cell hyperplasia, subepithelial basement membrane and connective tissue thickening, and variable nonspecific inflammatory cell response (lymphocytes and plasma cells). Structural bronchial damage is closely linked to heightened airway responsiveness. RADS has been reported following inhalation of chlorine gas and pulmonary toxic reactions have resulted from the inhalation of vapors generated by swimming pool chlorinating tablets. Given the widespread use of bromine compounds as bathing water disinfectants, the lack of previously reported cases of toxic reactions from brominating agents is surprising, particularly in the setting of hot tub use, where high water temperatures would be expected to promote the liberation of toxic vapors. This may reflect greater safety of bromine-containing disinfectants as compared with chlorinated ones. The greater water solubility of bromine allows for less vapor liberation from aqueous solutions. Alternatively, similar cases may have occurred but gone unrecognized or unreported.

We suspect that the bromine concentration and pH of the hot tub water in which our patients bathed were higher and lower, respectively, than recommended. The jewelry and bathing suit discoloration noted by patient 1 supports this contention. We were unable, however, to obtain any information from the health club management in regards to these parameters. Although not previously described, alopecia and proctitis are also consistent with the known toxicity (ie, corrosive effects) of bromine and its halogen acid and exposure by way of immersion.

An abnormally high bromine concentration or abnormally low pH in the hot tub water would also have resulted in greater production and liberation of Br₂ and HBr vapors, and, hence, respiratory tract exposure. The fact that the hot tub was located in a small room rather than outdoors or in a larger (eg, poolside) indoor area may have created a situation similar to that of smoke inhalation, where exposure in an enclosed space is well known to increase the risk of respiratory tract injury. Since both of our patients reported that no one else was present in the hot tub at the same time as they were, we were unable to determine whether other people used the hot tub in question and were similarly affected. We suspect that when our patients reported their symptoms to the health club management at the time of exposure, the hot tub area was closed to use until the situation could be investigated and corrected.

The known chemistry of brominating compounds, their reported use in the hot tub in question, the temporal relationship between exposure and toxic effects, clinical signs and symptoms that were consistent with the known toxicity of halogen gases and their acids, and the absence of alternative explanations strongly implicate Br₂ and HBr as the cause of our patients’ illnesses. Bromine poisoning should be added to the list of potential hot tub hazards. As with chlorine gas exposure, inhalation of Br₂ and HBr vapors may cause RADS. Patients with new-onset RADS or exacerbation of preexisting pulmonary disease should be questioned about hot tub use and exposure to halogen gases or vapors as a precipitating factor. Further study is necessary to determine the incidence of poisoning resulting from such exposures.

REFERENCES

Neurocardiogenic Syncope and Prinzmetal’s Angina Associated With Bronchogenic Carcinoma*

Paolo Angelini, MD; and Paul Y. Holey, MD

A clinical case is presented illustrating a previously unreported association of (1) neurocardiogenic syncope of new onset in a 57-year-old man, (2) Prinzmetal’s angina, and (3) bronchogenic carcinoma of the lung. Initiation of aggressive chemotherapy resulted in immediate suppression of both cardiac manifestations. This newly described paraneoplastic syndrome is discussed.

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Key words: bronchogenic carcinoma; neurocardiogenic syncope; Prinzmetal’s angina

Neurocardiogenic syncope is a well-defined cardiovascular condition; its cause, however, is still poorly understood.1,2 Although several pathophysiologic interpretations regarding its cause have been proposed, various mechanisms may contribute to the cause in different patients or even simultaneously in one patient.2-4 We present an unusual case of neurocardiogenic syncope, which was associated with a malignant form of Prinzmetal’s angina. In this patient, the causative factor, bronchogenic carcinoma, could be substantiated by empirical and clinical proof. Treatment of carcinoma by chemotherapy was indeed sufficient for prompt elimination of the cardiovascular symptoms.

Case Report

A 67-year-old Hispanic man, who was a long-time cigarette smoker (2 packs per day), was well until February 1995 when he began to experience episodes of syncope. Initially, he related the syncope to standing from the supine to the erect position; eventually, he began to experience these episodes even during bed rest. On March 30, 1995, he was admitted to a hospital in Mexico City because of prolonged rest, accompanied by near syncope. After the ECG showed anterior ST-T segment changes, he was immediately taken to the catheterization laboratory; a tentative diagnosis was made of acute anterior myocardial infarction. During the catheterization procedure, the patient was sedated and the pain disappeared. At that time, coronary angiography revealed a normal coronary flow pattern, with the appearance of a left anterior descending coronary artery lesion that showed about 50% diameter narrowing. While the patient was being prepared for balloon angioplasty, he again complained of chest pain and developed severe hypotension and bradycardia. New coronary angiograms clearly revealed an uneven flow pattern with slow flow limited to the left anterior descending coronary artery territory, without discrete narrowing. The ECG showed anterior T-wave changes and mild ST elevation, with advanced atrioventricular block. Intracoronary nitroglycerin and temporary ventricular pacing alleviated the bradycardia and ischemia. The new angiograms revealed no fixed stenosis and brisk flow in all the coronary branches. The procedure was thus aborted. Because of recurrent episodes of syncope and atrioventricular block with long periods of asystole, the patient eventually received a permanent atrioventricular sequential pacemaker. Medications were changed to dipryamide (100 mg, 3 times a day), fludrocortisone (0.1 mg daily), famotidine (40 mg daily), and sedatives. The patient continued to be bedridden because of recurrent dizziness and chest pain until his admission to St. Luke’s Episcopal Hospital on May 5, 1995. At that time, physical examination revealed an anxious, diaphoretic, acutely ill patient. No significant abnormalities were discovered at the time of physical examination, which included a detailed neurologic examination. BP was 110/70 mm Hg. ECGs obtained over the next several days (Fig 1) showed sinus or electronic pacemaker atrial rhythm or intermittent atrial fibrillation, with normally conducted QRS complex and transient T-wave changes that correlated with chest pain. A chest x-ray film was read as normal, with possible linear atelectasis in the left perihilar region.

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