Series Editor: Lewis S. Nelson, MD

A Toxic Swimming Pool Hazard

A young child presents in acute respiratory distress after inhalation of chlorine gas. His case illustrates the potentially tragic result of chlorine's toxic effects on the pulmonary system.

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Case

A previously healthy 3-year-old boy is seen in a garage, playing with a cup of water over a sealed container of pool-chlorinating tablets. The tablets contain trichloro-s-triazinetrione (TST), also known as trichloroisocyanuric acid (TCCA) or tri-chlor. He appears outside moments later, coughing and with mild dyspnea. His mother notes a strong chlorine odor emanating from the garage. She takes him inside and gives him milk to drink. He vomits shortly thereafter. His respiratory symptoms progress and he becomes lethargic. He arrives in the ED 3 hours postexposure.

The child presents in obvious respiratory distress. He is afebrile; his other vital signs include a blood pressure of 91/51 mm Hg; heart rate, 92 beats/min; respiratory rate, 42 breaths/min; and Spo_2, 93% on room air. He is lethargic but opens his eyes to voice. His conjunctivae are erythematous. Diffuse wheezing and retractions are noted on the pulmonary exam. His skin color is normal with mildly increased turgor. Treatment is initiated with inhalational albuterol and ipratropium, intravenous methylprednisolone and magnesium sulfate, and subcutaneous epinephrine. The patient is intubated for impending respiratory failure. A chest radiograph reveals an infiltrate in the right lung field.

In the PICU, the following vital signs are noted: blood pressure, 95/59 mm Hg; heart rate, 149/min; Spo_2, 83% on 100% Fio_2. The ventilator is set for high-frequency, low-volume oscillations. A repeat chest radiograph reveals diffuse bilateral infiltrates (Figure 1). Laboratory data are significant for a white blood cell count of 21,000/μL; arterial blood gas analysis reveals a pH of 7.20; $Paco_2$, 45 mm Hg; and Pao_2, 60 mm Hg on 100% Fio_2. Overnight, his oxygen saturation continues to fall, and extracorporeal membrane oxygenation (ECMO) is initiated. Figure 2 shows a post-ECMO radiograph.

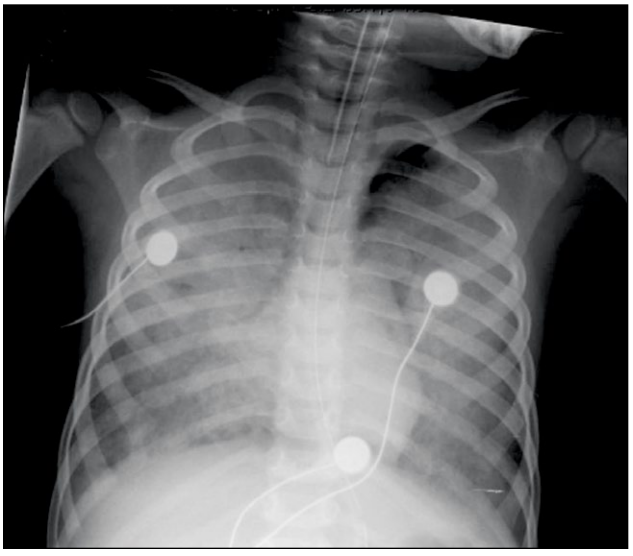

FIGURE 1. Initial chest radiograph showing diffuse bilateral infiltrates.

How does exposure to chlorine gas occur?

Chlorine (Cl_2) is a greenish-yellow gas with a characteristic odor. Chlorine gas is in common use in industrial settings (such as plastic manufacturing and food processing), and it finds more publicly accessible use as an antiseptic in water treatment systems, such as community swimming pools. It can be found in pressurized cylinders for transport and delivery, or in larger sealed containers for on-site use.

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Table. Commonly Available Pool-Chlorinating Products

Chemical name	Chemical formula	Common name	Form	Chlorine by weight	pH
Trichloro-s-triazinetrione	$Cl_3C_3N_3O_3$	Tri-chlor	Tablet	90%	3
Dichloro-s-triazinetrione	$Cl_2C_3N_3O_3$	Di-chlor	Granules	62%	7
Lithium hypochlorite	Li(OCl)		Granules	35%	11
Sodium hypochlorite	Na(OCl)		Liquid	12%	11
Calcium hypochlorite	$Ca(OCl)_2$	Cal-hypo	Tablet or granules	65%	12

For nonindustrial use, chlorine is typically generated from a precursor chemical, such as TST or a hypochlorite salt (Table). Wetting of a precursor liberates chlorine gas, which dissolves in the water to generate hypochlorous acid (HOCl) and other products, such as reactive oxygen species. If sufficient gas is rapidly liberated, it may escape immediate dissolution and result in ambient exposure to a nearby person. Pool chemical exposures are responsible for thousands of hospital visits each year.[1]

What are the signs and symptoms of chlorine gas inhalation?

The specific adverse clinical effects of chlorine exposure are related to the concentration (ppm) of chlorine gas and the duration of exposure. Following acute high-concentration exposure, irritation of the eyes, nose, and throat may occur within minutes. With increasing duration of exposure, patients can develop chest tightness, dyspnea, cough, laryngitis, and wheezing. Acute lower-concentration exposures typically result in a delay in the onset of effects, even as late as 24 hours. In this setting, upper respiratory effects may begin nearly simultaneously with pulmonary effects. Patients may experience vomiting from upper airway irritation and altered mental status from hypoxia. Severely affected patients may develop acute lung injury (chemical pneumonitis) that can progress to acute respiratory distress syndrome (ARDS). Death following acute inhalational exposures is rare, with one report of a train derailment that spilled tons of liquid chlorine in a small town producing a mortality greater than 1%.[2] Chronic low-concentration exposures, which are often occupation related, can result in reactive airways disease, which may improve when exposure is terminated. Since chlorine gas has a higher density than air (which is primarily nitrogen), small children, due to their shorter stature, may suffer greater exposure than adults.

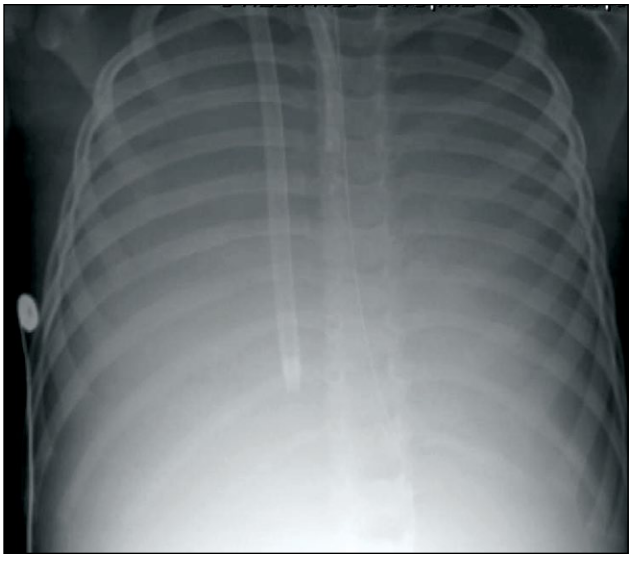

FIGURE 2. Chest radiograph obtained after initiation of ECMO.

The toxic effects of chlorine on the pulmonary system are related both to acid formation, as explained above, and to oxidative stress. In the latter situation, cytotoxic free radicals are responsible. This injury generates inflammatory mediators, which activate an inflammatory response, further increasing pulmonary damage.

What therapeutic options are available to treat pulmonary injury due to chlorine gas inhalation?

Care is primarily supportive and begins with supplemental oxygen. The goal is to maintain oxygen saturation above 90%. Nebulized β_2-adrenergic agonists are provided for symptomatic patients. The use of nebulized dilute sodium bicarbonate solution (2.1% to 4.2%; typical clinically available sodium bicarbonate solu-

tions are 8.4%) may neutralize the newly formed hydrochloric acid (HCl) and reduce pulmonary injury.[3]

Although there are case reports of oral or intravenous corticosteroid administration,[4] and animal studies suggesting a beneficial effect of corticosteroid use,[5] no controlled human studies exist. Aggravation of pulmonary superinfection in some patients may be a risk of use.

Endotracheal intubation should be performed as clinically indicated, and conventional ventilator management may be sufficient. For patients with ARDS, high-frequency ventilation (HFV)—a technique that provides very rapid breaths (up to 900 per minute) at very low tidal volumes—may be helpful. The result is continuous positive pressure maintaining the alveoli open, while minimizing further injury to the airway. HFV appears to improve overall oxygenation and mortality in patients with ARDS.[6]

In cases where gas exchange is severely disrupted, even advanced ventilation strategies may not be adequate to oxygenate the blood. When efforts to improve oxygen delivery come at the cost of increasing lung injury, ECMO has been used successfully.[6] With ECMO, venous catheters are placed and blood is shunted through an external device that both oxygenates and removes carbon dioxide. Bypassing the lungs decreases the risk of further ventilator-induced lung trauma.

Case Conclusion

During the second day, the patient's respiratory parameters worsened, and he became increasingly hypoxic despite aggressive medical management, including ECMO. As a result, the parents made the difficult decision to withdraw care. **EM**

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