Chlorine is a common element found worldwide regardless of location or social status. From industrialized nations to third-world enclaves, chlorine is used in diverse applications such as polymer production, water and sewage treatment, bleaching applications, and chemical weapons. In western Afghanistan, liquid chlorine is used in wastewater treatment facilities before the discharge of the waste.

We present a case of a patient with multiple chlorine gas exposures, complicated by an austere medical environment, and a relative lack of resources. Although this chlorine exposure was a result of a workplace mishap, similar exposures are expected if chlorine is used as a chemical weapon. Nurse anesthetists must be prepared for chemical casualties because of the prevalence of chlorine in American society.

Case Summary

A 45-year-old, 100-kg man presented to the Role II medical facility in western Afghanistan following a workplace exposure to liquid chlorine. He opened a 323-L (85-gal) drum of liquid chlorine at a wastewater treatment plant and was met with a cloud of pressurized chlorine gas that quickly enveloped his head and neck area. He wore no mask, respiratory filter, or breathing apparatus. The patient was found by his coworkers a short time following the exposure while he attempted to drive a forklift away from the site; he was disoriented, coughing, and appeared short of breath.

On presentation to the emergency medical tent (EMT), the patient appeared uncomfortable, tachypneic (respiratory rate, 32/min) and dyspneic (able to speak 1-to 3-word sentences only, interrupted by paroxysmal coughs). He was unable to inhale deeply because of coughing but denied pleuritic chest pain. His vital signs on presentation were as follows: temperature of 36.7°C (98.0°F), pulse of 82/min, blood pressure of 112/69 mm Hg, respiratory rate of 32/min and labored, and oxygen saturation at 100% (15 L via a nonrebreather mask) and 90% in room air. On physical examination, he had bilateral expiratory wheezing without crackles or rales; no visible conjunctival hyperemia, mild lacrimation, and rhinorrhea without epistaxis. A nonproductive cough occurred every 4 to 5 seconds.

The patient’s medical history showed a chlorine gas exposure in 1991 at a previous job site. Although no medical records were available regarding this incident, the patient reported that he was maintained on a regimen of mechanical ventilation therapy for 90 days following this exposure. Additionally, he sustained a second major chlorine gas exposure in Kirkuk, Iraq, in 2007 prompting a return to the United States for convalescence. His surgical history was notable for a tracheostomy in 1991. Allergies included iodine and shellfish. He used no outpatient medications or tobacco products. His laboratory, radiologic, and pulmo-
nary data on presentation were as follows: arterial blood gas, 15 L of oxygen by mask; pH, 7.39; PCO₂, 43.4 mm Hg; PO₂, 101 mm Hg; base excess, +1 mEq/L; bicarbonate, 26.2 mEq/L; and oxygen saturation, 98%.

A 12-lead electrocardiogram demonstrated normal sinus rhythm at 70/min, PR interval of 154 ms, QRS duration of 72 ms, QTc of 406 ms, no ST-segment elevation or depression, and no T-wave inversion. A chest radiograph obtained 3 hours after admission revealed no changes. There was no new development of cardiac enlargement, no evidence of pulmonary vascular prominence or pleural effusions, and no adenopathy noted.

Peak expiratory flow (TruZone Peak Flow Meter, Monaghan Medical Corp) for the 190.5-cm (75-in) patient; the predicted normal flow is 651 L/min. The patient produced a flow rate of less than 250 L/min after 3 attempts. The patient was admitted to the medical facility and was administered oxygen and albuterol, 2.5 mg, with ipratropium by nebulizer. No racemic epinephrine was given because the facility does not stock this product. His respiratory rate slowed to 20/min within 15 minutes of the albuterol treatment, and his cough had nearly resolved within 30 minutes of admission. At no time during the admission did he exhibit altered mental status, gastrointestinal upset, or blepharospasm or lacrimation. Ninety minutes after presentation, the patient was resting comfortably and speaking in full, nonlabored sentences. His peak flow rates had improved to 360 L/min. His flow rates would continue to improve to more than 500 L/min in the 3 hours following admission, and with the additional doses of albuterol via a metered dose inhaler every 30 to 45 minutes. A chest radiograph repeated 3 hours after admission demonstrated no changes, notably, no development of increased pulmonary vascular markings or effusion.

As the patient had normal vital signs, acceptable peak flow rates, and resolution of his subjective symptoms and wheezing on auscultation, he was discharged to his living quarters 4 hours following presentation to the medical facility. He was advised against further chlorine exposure and was instructed on the use of an albuterol inhaler, 4 puffs with a spacer, as needed for wheezing or chest tightness. He was instructed to follow up with the Role II facility surgeon in 72 hours or earlier if needed.

Forty-eight hours later the patient presented again to the Role II facility following a repeated inhalation of chlorine gas. He was working at the wastewater treatment plant, supervising his employees when an inadvertent vent of chlorine gas filled the workspace. On arrival 20 minutes after the exposure, he was tachypneic (respiratory rate, 40/min, and labored) with pronounced lacrimation and salivation as well as moderate conjunctival injection. He was leaning forward and using accessory muscles of respiration and he could produce 1- to 2-word sentences, interrupted by a cough productive of yellow mucus but without obvious hemoptysis. Peak flow measurements were unobtainable because of paroxysmal coughing. His vital signs on presentation were as follows: temperature, 36.9°C (98.5°F); pulse, 77/min; blood pressure, 138/77 mm Hg; respiratory rate, 40/min and labored, oxygen saturation, 98% (15 L via a nonrebreather mask) and 92% on room air. His physical findings included bilateral expiratory wheezing without crackles or rales, conjunctiva with visible hyperemia and lacrimation coupled with mild blepharospasm, and rhinorrhea (clear effluent) without epistaxis. A nonproductive cough occurred every 1 to 3 seconds.

Arterial blood gas (10 L of oxygen by mask) analysis demonstrated the following values: pH, 7.42; PCO₂, 42 mm Hg; PO₂, 60 mm Hg; base excess, −1 mEq/L; bicarbonate, 24 mEq/L; and oxygen saturation, 90%.

A chest radiograph obtained with a portable machine showed no cardiac enlargement, increased pulmonary vascular congestion compared with the study 48 hours previously, no pleural effusions, and no adenopathy.

In the EMT the patient was placed on a regimen of 10 L/min of oxygen by mask and given albuterol, 2.5 mg, via nebulizer. Peak flow measurements remained at less than 300 L/min following the β-agonist treatment, and the patient appeared fatigued from the work of breathing.

A plan of care was developed by the Certified Registered Nurse Anesthetist (CRNA) and the surgeon of the military surgical facility, with the patient’s consent. The patient was prepared for elective intubation and planned sevoflurane inhalation therapy, and he was moved to the operating room (tent). Following induction with 100 μg of fentanyl, 100 mg of lidocaine, 250 mg of propofol, and 120 mg of succinylcholine, a videolaryngoscope (GlideScope Ranger, Verathon Medical Inc) was used for laryngoscopic evaluation of the hypopharynx and endotracheal intubation. Direct videolaryngoscopy allowed the identification of diffuse mucosal edema of the hypopharynx and glottis with patchy mucosal sloughing and bleeding. The false vocal cords were profoundly edematous; the true vocal cords remained opposed following pharmacologic induction, preventing passage of an 8.0-mm endotracheal tube. After ensuring patient sedation, the anesthesia provider made 2 additional attempts at intubation, which were met with resistance to advancing the endotracheal tube with first a 7.5-mm endotracheal tube and then a 7.0-mm endotracheal tube. The third attempt was successful using the passage of a gum elastic bougie (15 French, SunMed) through the glottic opening and the placement of a 7.0-mm endotracheal tube over the bougie. Proper placement of the airway was confirmed with end-tidal carbon dioxide (ETCO₂) monitoring as well as by bilateral auscultation.

The initial ventilator settings were as follows: assist control rate, 12; tidal volume, 500 mL; positive end-expiratory pressure (PEEP), 5 cm H₂O; fraction of inspired...
Discussion

As chlorine gas enters the alveoli, it dissolves in the epithelial lung lining fluid. This fluid covers the apical surfaces of airway and distal epithelial cells. The chlorine then reacts with biological molecules, such as antioxidants and alveolar water. As the concentrations of antioxidants are decreased, the chlorine undergoes hydrolysis to generate hypochlorous and hydrochloric acids. These inorganic acids may contribute to the initiation and propagation of lung injury.

Chlorine is a common element found in the manufacturing, service, and household industries throughout the world. Its commonality belies its serious toxicologic and environmental threats. Chlorine is a common gas involved in toxic inhalation injury, either from accidental means or intended attack from chemical munitions. In this instance, a chlorine gas exposure occurred at a wastewater treatment plant in western Afghanistan. Unfortunately, the patient wore no personal protective equipment near the liquid chlorine drums despite 2 previous serious chlorine gas exposures. The material safety data sheet for liquid chlorine recommends splash-proof goggles and a face shield coupled with a respirator approved by the National Institute for Occupational Safety and Health and the Mine Safety and Health Administration (NIOSH/MSHA) while working in the vicinity of liquid chlorine. Because most municipalities in the United States and Canada use chlorine in water treatment facilities, it is reasonable for one to expect chlorine-exposed patients at small community hospitals in North America.

In western Afghanistan, a US Army Role II facility provides some primary care as well as limited surgical and critical care. This concentrated medical team provides rapid medical care for short periods with a limited holding capability. Its function is most similar to small rural healthcare facilities around the world. Despite the capabilities of the facility, one must be prepared to provide rapid assessment, stabilization, and immediate treatment before transfer to a higher level of care.

When the airway is exposed to chlorine gas, there are complex changes in both the upper and lower airways. Upper airway changes include increased mucus production, increased airway resistance, cough, and increased lacrimation. Lower airway changes are important to consider in clinical planning with large amounts of inhaled chlorine. These large inhalation injuries become evident as there is damage to the lung unit. The destruction occurs as the chlorine interacts with the epithelial cells, cilia, surfactant, proteins, and lipids. The need for early and controlled intubation and medication management must be considered related to these airway changes on exposure to chlorine gas.

Potent inhaled anesthetics have been used previously in the treatment of asthma. The similar pathophysi-
ogy includes bronchoconstriction, airway irritation, and increased airway resistance, which makes asthma and chlorine inhalation similar pathologies, and therefore treatment options are similar. Therapies for these pathologic conditions commonly include β-agonists and phosphodiesterase-4 inhibitors, both causing effects on cyclic adenosine monophosphate (cAMP). The benefits of cAMP levels include decreasing lung inflammation, dampening of the inflammation process, reducing neutrophil adhesion and chemotaxis, and bronchodilation of smooth muscle relaxation. Sevoflurane is known to have bronchodilation properties, therefore decreasing airway resistance. This smooth muscle relaxation mechanism works through changes at the voltage-dependent Ca++ channel activity and cAMP levels. Integration of early intubation and the use of sevoflurane to the plan of care was based primarily on the large amount of chlorine the patient inhaled. Worsening symptoms and limited resources made the use of sevoflurane an integral part of the patient care and may have promoted the best outcomes.

Potential treatments for patients who inhaled chlorine gas have been researched for many years, permitting the use of multiple therapies. In a best-case scenario, where all medications and treatments are available, it is critical for the anesthesiology provider to consider β-agonists, vitamin C, free radical scavengers (eg, N-acetyl-L-cysteine, deferoxamine), and parenteral sodium nitrate preparations as adjuncts for overall patient care. Treatment pathways involving multiple strategies to offset the acute and chronic damage of chlorine exposure may result in more rapid patient recovery and/or mitigation of the sequelae of chlorine gas inhalation. Future research into the effects of chlorine inhalation may offer workers at high risk of inhalation injury (military or industrial employees) pharmacologic prophylaxis to offset inadvertent damage due to chlorine gas damage. Corticosteroids have been studied for years in the treatment of lung injury because of their anti-inflammatory properties. They have a controversial role in the treatment of chlorine injuries during the acute inflammatory stage; however, research in this field for inhalation injury has been disappointing, and further studies are needed before any definitive recommendations.

Conclusion
All practitioners should be prepared for rapid intervention because of the numerous applications of chlorine in daily living worldwide. Patients who sustain chlorine inhalation injuries likely will present in various stages of distress. In similar fashion to patients with reactive airway disease, patients with chlorine inhalation injury will require prompt oxygen therapy and administration of an inhaled β-agonist. In select patients, advanced therapies should be considered, including administration of free radical scavenging agents and administration of volatile anesthetic agents. We believe that sevoflurane has a role in the treatment of moderate to severe injury by chlorine gas inhalation. In this case, sevoflurane proved beneficial in bronchodilation and improvement of gas exchange as manifested by lowered peak airway pressures and improvement in oxygen saturation levels.

As with patients who sustain thermal injury to the airway, those with chlorine inhalation injury may be a challenge to perform direct laryngoscopy and endotracheal intubation. Adjuncts such as video laryngoscopy and gum elastic bougies may lessen the need for surgical airway placement in patients who manifest airway mucosal slough and edema. Prompt airway intervention and therapy can be safely performed in less-than-ideal locations, from a tent in western Afghanistan to community hospitals around the world.

REFERENCES

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