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CASE REPORT

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Fatal chlorine gas exposure at a metal recycling facility: Case report

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At least four workers at a metal recycling facility were hospitalized and one died after exposure to chlorine gas when it was accidentally released from an intact, closed-valved cylinder being processed for scrap metal. This unintentional chlorine gas release marks at least the third such incident at a metal recycling facility in the United States since 2010. We describe the fatal case of the worker whose clinical course was consistent with acute respiratory distress syndrome (ARDS) following exposure to high concentrations of chlorine gas. This case report emphasizes the potential risk of chlorine gas exposure to metal recycling workers by accepting and processing intact, closed-valved containers. The metal recycling industry should take steps to increase awareness of this established risk to prevent future chlorine gas releases. Additionally, public health practitioners and clinicians should be aware that metal recycling workers are at risk for chlorine gas exposure.

KEYWORDS
acute respiratory distress syndrome, chlorine gas, metal recycling, post-traumatic stress disorder, reactive airway dysfunction syndrome

1 | INTRODUCTION

Occupational exposure to chlorine, although rare, occurs mostly due to unintentional events or human error in industries that use chlorine gas during production, such as pulp mills for bleaching. However, in 2010 at least 28 workers at two different metal recycling facilities in California were injured after exposure to chlorine gas when it was accidentally released from cylinders being processed for scrap metal. This report describes chlorine gas exposure from an intact, closed-valved cylinder being processed for scrap at a metal recycling facility in Washington State, resulting in at least four hospitalizations and one death. The two primary objectives of this report include detailing the clinical course of the fatal case of chlorine exposure to contribute to the current medical knowledge of this relatively rare occurrence, and elevating recognition of this specific hazard to the metal recycling industry as it marks at least the third such incident in the United States since 2010. The other three hospitalized workers were treated for varying lengths of time and discharged from the hospital; details of their clinical courses are not included in this report.

2 | CASE REPORT

The chlorine gas release occurred on the open air, ferrous (steel and iron) side of the metal recycling facility at approximately 9:40 am when an unlabeled metal cylinder being loaded by a material handler into a

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shear for processing burst at the seam, releasing a yellow gas that spread quickly. The material handler operator (hereafter patient), a 44-year-old male non-smoker with a history of diabetes, navigated through the thick yellow plume to escape the released gas; the amount of time he was exposed to the gas is unknown. Emergency response personnel arrived on the scene approximately 10-15 min after the gas release and removed the outer layer of the patient's clothing and decontaminated him onsite with water for roughly 15 min. The patient reported shortness of breath, coughing, chest pressure, and eye irritation. His oxygen saturation was 87% on ambient air and increased to 94% on a non-rebreather mask.

Emergency response personnel transported him to a local hospital; he arrived at the hospital a little more than 1 h after the gas release. Although alert, he had tachycardia and dyspnea on presentation and quickly developed hypoxic respiratory failure, necessitating mechanical ventilation. He was intubated roughly 20 min after arriving at the hospital. Due to poor oxygenation, he was placed in prone position and on an inhaled nitric oxide ventilation protocol. Chest radiographs showed bilateral infiltrates consistent with pulmonary edema and he was treated with the diuretic furosemide. Arterial blood gas measurements revealed concomitant respiratory and metabolic acidosis; intravenous sodium bicarbonate was administered. The patient's initial arterial partial pressure of oxygen to fraction of inspired oxygen (PaO_2/FiO2) ratio was 76; he was diagnosed with severe acute respiratory distress syndrome (ARDS).[1]

Initial reports from emergency response personnel relayed to the treating clinicians suggested the chemical released was arsenic trichloride rather than chlorine. After consultation with Poison Control, the chelating agent dimercaprol was ordered but was unavailable at the treating hospital or other local medical centers and had to be delivered from another city in Washington. Dimercaprol was administered when it arrived the second day of hospitalization. The patient also was treated with adjunctive therapies Vitamin B12, acetylcysteine, and folic acid for suspected arsenic trichloride exposure. In addition, Poison Control raised the possibility of arsenic trichloride's reacting to produce arsine gas, which can cause hemolysis. A peripheral blood smear was reviewed and reported to show schistocytes. Red blood cell/plasma exchange was initiated to treat suspected early hemolysis. The medical record indicated the fire department confirmed the gas was indeed chlorine and reported that to the hospital the evening of the incident. In addition, the patient's arsenic blood level was not elevated, although that result was not available until after the patient's death.

During the first evening of hospitalization the patient developed hypotension. Vasopressor therapy was initiated and he responded with a mean arterial pressure maintained above 70 mmHg. Early in the morning of the second day of hospitalization, continuous renal replacement therapy was initiated for oliguria and refractory acidosis, but discontinued due to profound hypotension; the patient's mean arterial pressure dropped below 30 mmHg. Despite diuresis and improved urine output, his blood urea nitrogen (BUN) and creatinine continued to increase during the next several days of hospitalization (Table 1); the acute kidney injury was thought secondary to hypotension-related acute tubular necrosis. The patient's creatine kinase (CK) levels were elevated throughout hospitalization (Table 1).

The patient's cardiopulmonary status improved somewhat during the second and third days of hospitalization, though he remained critically ill. The patient's PaO_2/FiO_2 ratio increased during the second day of hospitalization (Table 1), and he was weaned from nitric oxide when his FiO_2 requirement decreased to 40% on the third day of hospitalization. Vasopressor therapy was weaned off by the fourth hospital day. However, he remained tachycardic and developed intermittent atrial fibrillation with rapid ventricular rate that responded to diltiazem. A cardiac panel ordered on the third day of hospitalization revealed an elevated troponin-I (Table 1).

On the fourth day of hospitalization, the patient experienced a precipitous decline in his oxygen saturation that was unresponsive to changes in ventilator settings. Endotracheal tube displacement was suspected and the tube was successfully replaced. Subsequently, he developed bradycardia followed by pulseless electrical activity. Cardiopulmonary resuscitation was initiated, but he responded poorly and was pronounced dead after further resuscitation efforts were deemed futile. Autopsy findings were consistent with ARDS with purulent bronchitis and pneumonia. Erythema and petechial hemorrhages were noted throughout the upper airway and bronchi; fluid exuded from the parenchyma. Microscopic examination of the lungs demonstrated altered hyaline membranes and hypertrophy of type II pneumocytes. Cardiomegaly was also present as all four heart chambers were globally enlarged.

3 | DISCUSSION

The acceptance and processing of an intact, closed-valved, unlabeled cylinder led to the release of chlorine gas, exposure of facility employees, at least four hospitalizations, and one death. The unintentional chlorine gas release at this metal recycling facility marks at least the third such incident in the United States since 2010, and the first involving a fatality. Public health practitioners and clinicians should be aware that metal recycling workers are at risk for chlorine gas exposure. The metal recycling industry should take steps to increase awareness of this established risk to prevent future chlorine gas releases.

Occupational exposure to high concentrations of chlorine gas is rare, and most commonly due to workplace unintentional events or human error. In 2005, 72 persons were hospitalized and 9 died after a train transporting 60 tons of chlorine gas derailed in South Carolina.[2] Additionally, emergency personnel responding to such incidents may be exposed to the hazards without sufficient respiratory protection.[3] Industries such as pulp and paper mills that use chlorine gas as part of production processes typically educate employees on chlorine safety and install chlorine detectors for added protection.[4] Although chlorine gas exposure is not expected in the metal recycling industry, three separate incidents resulting in at least 32 workers hospitalized and one

TABLE 1 Select laboratory results from patient, hospital admittance (day 1) to death (day 4)

Laboratory test	Day 1 time point A[b]	Day 1 time point B[b]	Day 2 time point A	Day 2 time point B	Day 3 time point A	Day 3 time point B	Day 4 time point A	Day 4 before death
O_2 saturation[a] (%) (95-99)	88	79	99	98	97	91	97	63
pH[a] (7.35-7.45)	7.20	6.99	7.34	7.43	7.44	7.42	7.37	7.03
Arterial partial pressure of oxygen (PaO_2)[a] (mmHg) (80-100)	69	76	213	238	83	59	93	48
Fraction of inspired oxygen (FiO2) (%)	-	100	93	78	55	40	90	100
PaO_2/FiO_2[c]	-	76	229	305	151	148	103	48
Lactate (mmol/L) (0.4-2.0)	6.6	8.9	4.4	-	1.8	-	1.2	10.3
Hematocrit (HCT) (%) (41-53)	55.3	44.8	41.2	41.2	26.4	-	19.7	-
Hemoglobin (HGB) (g/dL) (13.5-17.5)	17.6	14.8	14.6	14.6	9.9	-	7.1	-
Blood urea nitrogen (BUN) (mg/dL) (7-18)	17	-	22	23	25	26	31	39
Creatinine (mg/dL) (0.8-1.3)	1.5	-	2.8	3.2	3.6	3.9	4.6	5.0
Troponin-I (ng/mL) (0.00-0.06)	-	-	-	-	0.99	0.73	0.34	-
Creatine kinase (CK) (units/L) (39-308)			3044	6919	9460	10705	9430	-

[a]Arterial blood gas measurements.
[b]Time points A and B are sequential time points during each of the patient's four calendar days of hospitalization from admission to death.
[c]Calculated from arterial blood gas measurements: normal (>300); mild acute respiratory distress syndrome (ARDS) (200-300); moderate ARDS (100-200); severe ARDS (<100)[1].

death since 2010 collectively indicate that workers in this industry are at risk. In response to the two chlorine gas releases at metal recycling facilities in California that occurred in 2010, the California Department of Public Health distributed a Chemical Release Alert to raise industry awareness of this potential occupational hazard.[5] The Chemical Release Alert likely increased industry awareness in California, as to our knowledge no chlorine gas releases involving injuries at metal recycling facilities have been reported since 2010. The Chemical Release Alert may not have been widely distributed outside of California. This chlorine gas release occurred in Washington State, 5 years following the Alert, emphasizing the importance of continued, industry-wide messaging to reduce the risk of this occupational hazard.

The health status of the surviving affected employees at this metal recycling facility was not assessed. Reactive airway dysfunction syndrome (RADS) has been documented previously following exposure to chlorine gas.[6] RADS typically includes persistent asthma-like respiratory symptoms, particularly cough, shortness of breath, or wheeze, and evidence of airflow limitation on spirometry, or bronchial hyperresponsiveness on non-specific bronchoprovocation challenge testing. Most patients with RADS improve over time, although many remain symptomatic for years.[7] Post-traumatic stress disorder (PTSD) symptoms are also possible following exposure to chlorine gas. Twenty-two percent of employees reported symptoms consistent with PTSD following an unintentional chlorine gas release at a poultry processing plant in 2011,[6] and nearly half of respondents reported PTSD symptoms following a train derailment and chlorine gas exposure in South Carolina in 2005.[2] PTSD symptoms have the potential to last many years, with approximately one-third of individuals diagnosed with PTSD displaying symptoms for up to 6 years in one study.[8]

According to the medical records, emergency response personnel confirmed the gas released from the cylinder was chlorine and reported the information to the hospital treating the deceased worker the same day of the release. However, the confirmation that chlorine gas was released from the cylinder, rather than arsenic trichloride, may not have been disseminated to all members of the medical team during his hospitalization. Lack of timely chemical identification and communication of pertinent information from emergency response personnel to healthcare providers have delayed treatment decisions in previous unintentional gas releases.[9] Effective coordination and communication between emergency response personnel and health-care providers and within these groups is paramount to reduce morbidity and mortality following unintentional gas releases.

The initial chlorine concentration is not known. Given his initial clinical signs and rapid development of respiratory failure, the deceased employee's chlorine exposure was likely >50 parts per million (ppm) (Table 2).[6] The amount of time he spent navigating through the yellow plume of chlorine gas is unknown. Although chlorine is approximately twice as dense as air and therefore tends to settle, weather conditions, as well as piles of scrap metal serving as physical barriers, would probably have affected the movement, and dissipation of the chlorine plume. Reports of eye and upper respiratory irritation in employees located near the road separating the ferrous and non-ferrous sides of the facility suggest a concentration of at least 5 ppm at that distance. During previous chlorine gas releases, workers attempting to escape chlorine plumes may have inadvertently increased their exposure by moving through the plume rather than staying upwind up the plume.[10] Developing evacuation plans and training employees to stay upwind if at all possible when evacuating from a chemical release could also decrease morbidity and mortality. Additionally, metal recycling facilities could consider strategically placed escape gas masks in the case of unintentional chemical gas releases.

TABLE 2 Chlorine gas concentration and acute health effects[4]

Concentration	Effect on human health
1-3 ppm	Mild mucous membrane irritation
>5 ppm	Eye irritation
>15 ppm	Throat irritation
15-30 ppm	Cough, choking, burning
>50 ppm	Chemical pneumonitis
430 ppm	Death after 30 min exposure
>1000 ppm	Death within minutes

The clinical course of the deceased employee is consistent with previous case reports of acute lung injury and ARDS following exposure to high concentrations of chlorine gas. While there are no widely accepted medical treatment guidelines for acute chlorine exposure, mostly due to the rare nature of the occurrence, some studies suggest that inhaled bicarbonate and glucocorticoids, including systemic corticosteroids and inhaled agents such as budesonide, may be beneficial, but these findings are largely anecdotal.[11] This patient received intravenous bicarbonate due to his profound concomitant respiratory and metabolic acidosis, which corrected following treatment. The initial uncertainty surrounding the chemical involved in the release (arsenic trichloride vs chlorine) could have influenced the treatment decision not to use glucocorticoids.

Hemolysis was suspected shortly after admission to the hospital. Treating clinicians consulted with a Poison Control toxicologist and discussed the possibility that arsenic trichloride reacted to produce arsine gas. Arsine (AsH_3) gas exposure can cause of rapid destruction of red blood cells, hypoxia, and renal failure,[12] and has been successfully treated with red blood cell/plasma exchange.[13] However, the gas was proven to be chlorine, which is not known to directly cause acute hemolysis. Interestingly, there is increasing evidence that hemolysis could occur in the airway space of patients with acute lung injury/ARDS, releasing hemoglobin and heme as a proinflammatory mechanism.[14] His autopsy findings including pulmonary edema, bronchial erythema, and cardiomegaly were similar to findings from those who died following chlorine gas exposure from a train derailment in South Carolina in 2005.[15] Recent studies suggest that chlorine inhalation can cause cardiotoxicity directly likely due to oxidative stress in part, rather than solely secondary to respiratory effects like hypoxia.[16–18] Cardiac involvement during the clinical course of the patient was demonstrated by tachyarrhythmia, elevated troponin I, cardiac arrest, and cardiomegaly. The attribution of these effects as due directly to cardiotoxicity of chlorine rather than as secondary to other conditions such as underlying cardiac disease in a patient with diabetes, ARDS, acute kidney injury, or profound hypotension is unclear; however, the fact that his oxygen saturation began to improve suggests that the cardiac abnormalities were unlikely solely secondary to the respiratory effects, and that direct cardiotoxicity of chlorine was plausible.

Steel is the most recycled material worldwide. Ferrous metal recycling activities contributed more than 150 000 jobs, more than $8 billion in wages, and nearly $2 billion in tax revenue in 2016.[19] As the metal recycling industry continues to grow, the risk of processing intact, closed-valved containers that are unlabeled should be explicit within the industry, and such containers should be treated as potential hazardous materials, with the appropriate authorities notified before processing. The threat of radioactive materials is controlled during the process of metal recycling as radioactive detectors monitor incoming scrap metal. Administrative controls to identify intact, closed-valved containers brought to metal recycling facilities before processing begins may further protect employees.

Chlorine and other noxious chemical gas releases from intact, closed-valved containers at metal recycling facilities are preventable. The metal recycling industry should take steps to increase awareness of the potential risks of processing materials that may contain hazardous substances, and provide regular training to employees on health and safety protocols, and emergency plans. Additionally, public health practitioners and clinicians should be aware that metal recycling workers are at risk for chlorine gas exposure.

AUTHORS' CONTRIBUTION

All authors contributed to acquisition and interpretation of the data, drafting or revising the work, as well as approving the final version to be published.

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ETHICS APPROVAL AND INFORMED CONSENT

As the person described in the report is deceased, the report is not covered by provisions of human subject research protection..

DISCLOSURE (AUTHORS)

The authors declare no conflicts of interest.

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Rodney Ehrlich declares that he has no conflict of interest in the review and publication decision regarding this article.

DISCLAIMER

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention.

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