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Chloramine Intoxication in a Five-Month Pregnant Patient: A Case Report

Niels Oversteyns, Hans De Puydt, Stefanie Vandervelden

Department of Emergency Medicine, Ziekenhuis Aan de Stroom Network of Hospitals (ZAS) (Augustinus Campus), Antwerp, Belgium
Email: niels_ovrstns@hotmail.com

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Abstract

Chloramine inhalation resulting from the accidental mixing of bleach and ammonia is a potentially life-threatening intoxication, as it may lead to severe respiratory compromise. The majority of case reports on intoxication with chlorine-containing substances focus exclusively on chlorine inhalation, and no cases involved pregnant patients. We present a case of a 27-year-old, five-month pregnant biologic female who experienced acute respiratory distress due to non-cardiogenic pulmonary edema after cleaning her bathroom with bleach in combination with ammonia. Initial management included removal from exposure, supplemental oxygen, non-invasive ventilation via Continuous Positive Airway Pressure (CPAP) and methylprednisolone IV, resulting in rapid clinical improvement. Fetal monitoring remained reassuring throughout hospitalization. The patient was observed for 24 hours and discharged in stable condition. Management of chloramine intoxication is primarily supportive, with limited evidence for bronchodilators, corticosteroids, or nebulized sodium bicarbonate. In pregnant patients, maternal stabilization and continuous fetal monitoring are essential. Observation for 24 hours is advised, with follow-up to monitor for potential chronic pulmonary sequelae.

Keywords

Chloramine Intoxication, Acute Non-Cardiogenic Pulmonary Edema, Pregnancy

1. Introduction

Chloramine gas can be generated by mixing bleach, which contains sodium hypochlorite, with ammonia. Similar to chlorine gas, inhaling chloramine can cause a range of respiratory symptoms, from mild mucosal irritation to acute respiratory

failure. In severe cases, exposure can be fatal. Both chlorine and chloramine have historically been used as chemical warfare agents. Most incidents involving chlorine-containing substances occur due to the improper use of household cleaning products or the accidental mixing of incompatible agents [1] [2]. While chlorine gas typically forms when bleach is combined with an acidic cleaner (e.g., drain cleaner), chloramine gas arises from mixing bleach with ammonia, producing mono-, di-, and trichloramine compounds (NH_2Cl, $NHCl_2$, and NCl_3). These compounds are highly irritating and react with moisture in the airways to form hydrochloric acid and reactive oxygen species, leading to epithelial injury and inflammation [3]. Despite the widespread domestic use of these products, case reports of chloramine intoxication remain rare, and data on its effects during pregnancy are lacking. A comprehensive review of the extant literature in the PubMed database revealed that this is the first reported case of chloramine poisoning in a pregnant patient resulting from the accidental mixing of bleach and ammonia.

2. Case Description

A 27-year-old biologic female (G3P2A0) who was five months pregnant presented to our emergency department with dyspnea and chest pain. Approximately 15 minutes after accidentally using a mixture of bleach (sodium hypochlorite) and ammonia to clean her bathroom with the window and door closed, she developed symptoms. Thirty minutes after the onset of symptoms, she arrived at the emergency department in respiratory distress. Her past medical history was unremarkable aside from active tobacco use. She reported no allergies, was not taking any chronic medications and had no significant comorbidities.

On initial assessment, her airway was patent. However, she exhibited overt respiratory distress, with a respiratory rate of 40 breaths per minute, an oxygen saturation of 86% on ambient air and bilateral wheezing and crackles on auscultation. Her heart rate was 104 beats per minute (sinus tachycardia), her blood pressure was 142/81 mmHg and her capillary refill time was two seconds. No cardiac murmurs were audible. She was alert and neurologically intact. Her temperature was 36.6˚C. Physical examination confirmed a gravid uterus consistent with approximately five months of gestation. There was no rash, conjunctivitis or peripheral edema.

The patient was immediately transferred to a resuscitation room. Oxygen was initially administered via a non-rebreather mask at a flow rate of 15 liters per minute. Bronchodilator therapy (aerosolized salbutamol and ipratropium bromide) was initiated. Two peripheral intravenous lines were established. Initial arterial blood gas analysis revealed the following: pH: 7.4; pCO_2: 29 mmHg; pO_2: 67 mmHg; HCO_3^-: 18 mmol/L, lactate: 1.1 mmol/L. An electrocardiogram (ECG) showed sinus tachycardia at 110 beats per minute without arrhythmia or signs of acute ischaemia. Point-of-care ultrasound (POCUS) performed by a clinician with expertise in point-of-care ultrasound revealed bilateral lung sliding, diffuse B-lines without pleural effusion, a hyperdynamic left ventricle without evidence of pericardial

effusion, right ventricular dilation, or D-shaping. Given the pregnancy and a clear clinical picture confirmed by POCUS, no chest X-ray was performed.

The fetal heart rate was 135 beats per minute. Laboratory results were unremarkable, showing no signs of infection and indicating normal cardiac enzyme levels and preserved renal function.

A differential diagnosis of acute pulmonary edema was considered due to the presence of acute respiratory distress in combination with a bilateral B-line pattern on POCUS. Given the temporal correlation between the onset of symptoms and exposure to chloramine gas, we proposed a diagnosis of acute non-cardiogenic pulmonary edema secondary to chloramine inhalation. Other differential diagnoses, such as primary cardiogenic pulmonary edema, were considered less probable due to the absence of a known medical history of cardiac disease in an otherwise healthy female patient, the absence of acute ECG abnormalities, and a POCUS examination demonstrating normal left ventricular function on visual estimation. The likelihood of bilateral pneumonia was considered low due to the absence of symptoms such as productive cough or fever. The patient's history indicated that alternative etiologies for acute non-cardiogenic pulmonary edema, including neurological conditions, drowning, drug-induced causes, aspiration, or transfusion-related events, were considered unlikely.

Non-invasive ventilation was initiated using continuous positive airway pressure (CPAP) with positive end expiratory pressure (PEEP) at 8 cm H_2O and FiO_2 at 100 %. Additionally, intravenous methylprednisolone (40 mg) and furosemide (20 mg) were administered. Over time, the FiO_2 requirement was gradually reduced to 40 %. After two hours of CPAP use in our emergency department, non-invasive ventilation was discontinued and oxygen was administered via a nasal cannula at a flow rate of 2.5 L/min. The patient reported significant improvement in her symptoms and expressed a wish to be discharged. Our reassuring fetal findings were confirmed by a bedside obstetric transabdominal ultrasound examination.

Following a multidisciplinary consultation, she was admitted to the pulmonology ward for further monitoring. Over the subsequent 24 hours, pulmonary function testing and ongoing fetal surveillance revealed no abnormalities. She was discharged in a stable condition with no recurrence of symptoms. The patient gave birth to a healthy child. At the nine-month follow-up, the patient reported complete resolution of symptoms. Pulmonary function testing was not repeated.

3. Discussion

Our patient exhibited features consistent with severe, chlorine-related inhalation injury. These features included the rapid onset of dyspnea and hypoxemia, as well as the presence of bilateral B-lines on ultrasound, despite the absence of underlying cardiac pathology. These features are suggestive of acute, non-cardiogenic pulmonary edema. The following discussion addresses the epidemiology, clinical spectrum, management strategies (including current controversies), and potential fetal implications in the context of the available literature.

3.1. Epidemiology

In a recent retrospective analysis of the U.S. National Poison Data System, Atalla *et al.* documented 75,186 cases of exposure to chlorine and chloramine gas between 2015 and 2022. The vast majority (96.6%) were unintentional. Of all reported exposures, 16.7% required evaluation in the emergency department, 10.9% resulted in moderate to severe clinical symptoms, 2% required hospitalization, and 0.03% were fatal [1].

3.2. Clinical Presentation

The clinical manifestations of chlorine-containing substances range from mild to life-threatening. They depend on the concentration of the gas, the duration of exposure, and patient-specific factors, such as age, preexisting pulmonary disease, and tobacco use [1] [2]. Active smoking has been demonstrated to enhance a patient's vulnerability to severe respiratory symptoms subsequent to exposure to irritant gases. Moreover, it has been associated with an augmented risk of developing persistent airway obstruction following chlorine inhalation [4]. Mild exposure typically causes irritation of the upper airways, presenting as a burning sensation in the oropharynx, cough, and hoarseness. This is often accompanied by ocular irritation (conjunctival injection), as well as nausea, vomiting, and headache. More severe symptoms usually develop within six hours and may include dyspnea, a persistent cough, excessive sputum production, hemoptysis, or chest pain. These symptoms can be caused by laryngeal edema, toxic pneumonitis, acute non-cardiogenic pulmonary edema, or acute respiratory distress syndrome (ARDS) and generally occur at exposures of 40 ppm - 60 ppm. Concentrations above 400 ppm can be fatal within 30 minutes. Direct chemical injury may also result in severe corneal erosion [1] [2] [5] [6]. Long-term exposure to chlorine gas carries a risk of developing pulmonary fibrosis or reactive airway disease [5]. In our patient, however, no long-term sequelae have been observed nine months after the inhalation incident.

3.3. Therapy

There is no specific antidote for inhaling chlorine or chloramine, and treatment is primarily supportive. Initial management focuses on immediate removal from the source of exposure and administration of supplemental oxygen [7].

Non-invasive ventilation is recommended for managing acute pulmonary edema, and its effectiveness has been demonstrated in case reports of chlorine and chloramine inhalation [8] [9]. In our case, we observed clear clinical improvement with reduced oxygen requirements and fewer respiratory symptoms approximately two hours after initiating CPAP.

Although **bronchodilators** are commonly administered to manage bronchospasm associated with gas exposure, evidence supporting their efficacy in cases of chlorine or chloramine inhalation is limited [7]. In our case, no improvement was observed following a single trial of bronchodilator therapy (salbutamol-ipratropium

bromide). We hypothesize that the audible wheezing was due to pulmonary edema rather than to reversible bronchospasm.

Corticosteroids are sometimes used empirically to treat inhalation injury, as they can reduce inflammation and edema. However, their use in acute chlorine or chloramine injury is controversial because there are no randomized trials demonstrating a reduction in mortality or long-term morbidity. Some argue that steroids might delay epithelial repair, impair alveolar re-epithelialization, or hinder the differentiation of alveolar type II cells into type I cells. Others cite the risks of immunosuppression and hyperglycemia [7]. The use of methylprednisolone in this pregnant patient appears to be safe based on the current literature, given the absence of direct evidence linking antenatal systemic corticosteroid exposure to adverse pregnancy outcomes such as preterm birth, low birth weight, or preeclampsia. Furthermore, the potential adverse effects of corticosteroids are associated with the duration of exposure; in our case, this was limited to a single dose [10].

Given the presence of pulmonary edema with hypoxia, furosemide was administered intravenously at a dose of 20 milligrams. Although no fetal harm is reported with **diuretics**, its use was not retrospectively justified [11]. Diuretics are not the standard therapy for acute non-cardiogenic pulmonary edema because elevated preload and left atrial pressure, which are key determinants of cardiogenic edema, are absent. Their use may induce hypotension; therefore, caution is essential when using diuretics in pregnant patients to avoid hypovolemia and uteroplacental perfusion compromise [12].

Although we did not administer nebulized **sodium bicarbonate** in this case, it has been recommended as an additional treatment for inhaling chlorine gas. Sodium bicarbonate could potentially neutralize hydrochloric acid in the airways. One randomized controlled trial (RCT) involving 44 patients with reactive airway dysfunction syndrome (RADS) following chlorine exposure showed significant improvements in forced expiratory volume in one second (FEV_1) at 120 and 240 minutes compared with placebo [13]. However, an umbrella review published in 2022 concluded that the evidence remains limited and that no firm clinical recommendations can be made [7].

3.4. Observation and Follow-Up

Patients with moderate exposure are frequently monitored for a minimum of 24 hours, given the potential for delayed onset of respiratory deterioration. Longitudinal follow-up is also recommended to monitor for late sequelae, such as airway hyperreactivity, RADS, or restrictive lung disease [7].

3.5. Fetal Effects

To date, no studies have specifically examined the effects of elemental chloramine gas inhalation during human pregnancy. Nevertheless, a large retrospective cohort study including 109,182 mother-infant singleton pairs in Shanghai did not identify any clear link between maternal exposure to elevated chloramine levels in

tap water and adverse outcomes for the newborn [14]. In this case, continuous fetal monitoring provided reassurance, and no adverse fetal outcomes were observed during hospitalization or follow-up. Given the absence of a known teratogenic risk and the maternal benefits of optimal respiratory support, managing pregnant patients similarly to non-pregnant patients seems reasonable, with additional fetal surveillance and obstetric input.

4. Conclusion

Acute chloramine inhalation from mixing household cleaning products (e.g., bleach and ammonia) can manifest as acute non-cardiogenic pulmonary edema in a patient who is five months pregnant. The appropriate initial management includes early removal from the source of exposure, supplemental oxygen, and non-invasive ventilation (e.g., CPAP). For pregnant patients, the priority should be maternal stabilization and fetal monitoring. There is limited high-quality evidence supporting the use of bronchodilators, corticosteroids, or nebulized sodium bicarbonate. Observation for at least 24 hours is recommended in cases of moderate exposure. Long-term follow-up may reveal chronic pulmonary diseases.

Authors' Contributions

Niels Oversteyns: Conceptualization and writing. Hans De Puydt: Writing. Stefanie Vandervelden: Writing.

Consent Statement

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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