Chlorine Gas Exposure Manifesting Acute Lung Injury

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Abstract

Unintentional exposure to chlorine at swimming pools is not uncommon and can occur through various exposure scenarios, such as chlorine leak from pipes or reservoirs, or inappropriate flushing of chlorination/sanitation lines. Although mixing bleach (sodium hypochlorite) with acids accounts for many household chlorine exposure accidents, such an exposure is rare at swimming pools. Clinical manifestations of chlorine inhalation can range from mild upper airway irritation to life-threatening toxic effects, such as pulmonary edema/acute respiratory distress syndrome. We reported an incident of chlorine exposure generated from mixing sodium hypochlorite and hydrochloric acid at a swimming pool. Among the 7 victims, 6 presented with dyspnea, dry cough, sore throat and eye irritation. These six patients were discharged within 1 day. A 15-year-old girl however developed hypoxemia and acute lung injury primarily involved right middle and lower lungs in addition to symptoms of airway irritation. She was discharged 5 days later after receiving both bronchodilator and intravenous steroid therapy. Her clinical manifestations were discussed along with a review of existing literature reports on chlorine inhalation. (J Intern Med Taiwan 2010; 21: 210-215)

Key Words： Chlorine, Lung injury, Sodium hypochlorite

Introduction

Chlorine is a yellowish-green gas with a pungent, irritating odor. It is intermediate water-soluble and can cause acute damage to both upper and lower respiratory tracts[1]. Its toxicity is thought to be mediated by the generation of hydrogen chloride upon contact with moist mucous membrane and by the formation of free radicals at the cellular level[1]. Toxic exposures to chlorine were first reported in 1915 when chlorine was used as a chemical warfare agent in Ypres, Belgium. In the 1920s, chlorine was introduced as a bleaching agent[2]. Chlorine was subsequently used in many industrial processes as well as household bleaching and water purification. Because chlorine is often transported and stored under pressure in pipes, trucks, or tanks, many mass

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poisonings have occurred following transportation or industrial accidents[1,3]. Decker and Koch reported the first case of acute chlorine gas exposure at a swimming pool in 1978[4]. Similar cases or incidents were not infrequently reported in the literature afterwards[5-11]. Clinical manifestations of such exposures could range from mild airway irritation to life-threatening toxic effects, such as acute respiratory distress syndrome (ARDS). We recently managed an incident of acute chlorine exposure that involved 7 persons at a swimming pool. While the patients' clinical features were largely similar to those described in previous reports, we observed unusual chest radiograph finding in one of the patients. We herein reported the case series and discussed various aspects of chlorine gas exposure.

Case Report

A nonsmoking, previously healthy 15-year-old girl was presented in the emergency room after an unintentional exposure to chlorine gas at a community swimming pool. She was exposed to chlorine gas for some 6 minutes in a shower room after while a swimming pool workers mixing sodium hypochlorite with hydrochloric acid outside the room. The exact concentration of inhaled chlorine was unknown.

On arrival, she manifested dyspnea, dry cough, throat and eye irritation, and chest discomfort. Her blood pressure was 100/68 mmHg, pulse 70/min, and respiratory rate 24/min. Physical examinations did not reveal rhonchi or rales; pulse oximetry however showed hypoxemia with oxygenation saturation of 86%. Arterial blood gas analysis revealed the following: pH 7.46, $PaCO_2$ 28.4 mmHg, and PaO_2 69 mmHg on room air. Chest radiograph demonstrated increased infiltrates over right middle and lower lungs (Fig. 1). Laboratory data, including complete blood cell count; serum sodium, potassium, glucose, cardiac enzymes, creatinine, blood urea nitrogen, and liver enzymes were all unremarkable. A complete electrocardiogram was also within normal limits.

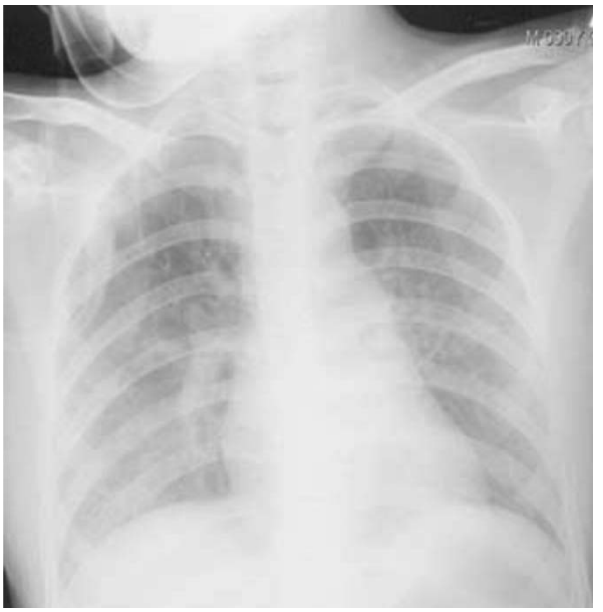

Fig. 1.Chest radiograph on presentation showing increased infiltrates over right middle and lower lung fields.

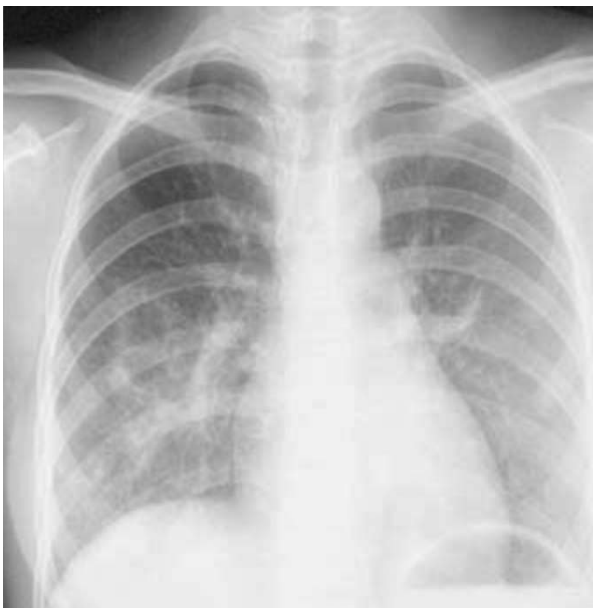

Fig. 2.Chest radiograph on day 4 revealing persistent infiltrates over right middle and lower fields, and some infiltrates over left perihilar region.

She was treated with 100% oxygen, intravenous fluid and corticosteroid, and inhaled β_2 agonist every 6 hour. Repeated chest radiograph

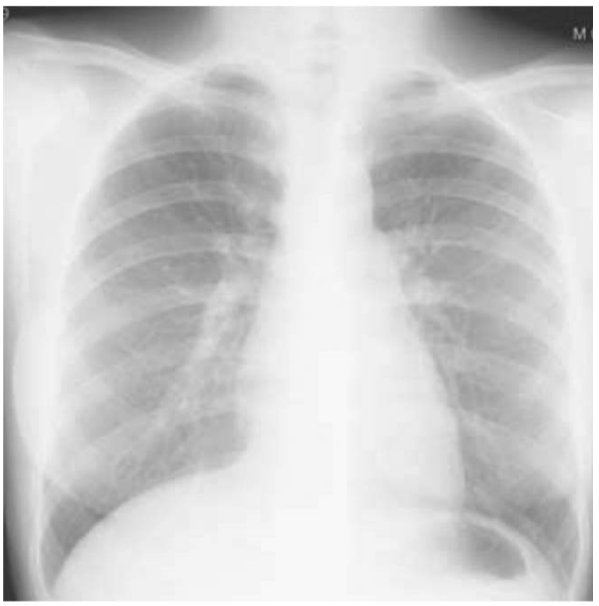

Fig. 3. Follow-up chest radiograph 8 days post-exposure demonstrating normal findings in both lungs.

performed later on the same day showed no interval change. She was hospitalized due to persistent dyspnea and received supplementary oxygen, oral prednisolone 10 mg and inhaled budesonide 2 puff every 8 hour for 4 days. Computerized tomography (CT) scan of chest was not performed as the patient declined the study.

While her clinical symptoms gradually improved, the follow-up chest radiograph on day 4 still showed increased infiltrations over right lung (Fig. 2). In addition, some infiltrates over left perihilar region were noted. A repeated arterial blood gas analysis on the same day however revealed pH 7.38, $PaCO_2$ 46.3 mmHg, PaO_2 98.7 mmHg, and HCO_3 26.6 mmol/L with room air breathing, which indicated delayed resolution of chest radiograph abnormalities that lagged behind clinical improvement. The patient was discharged on 5th hospital day. A follow-up chest radiograph was normal 8 days post-exposure (Fig. 3).

Six other persons were also exposed to chlorine gas at the swimming pool but probably had a shorter period of exposure. All of them were sent to the emergency room with dyspnea, dry cough, and throat and eye irritation. Physical examinations and routine laboratory workup were unyielding. They were treated with supplemental oxygen, intravenous fluid and an inhaled β_2 agonist therapy. On the next day, all of them were asymptomatic and did not require oxygen therapy. All were discharged on that day.

Discussion

Many people use swimming pools for exercise, recreation, sports, and even rehabilitation therapy. Chlorination is the primary measure employed in disinfecting community pools. Unintentional inhalation of chlorine at swimming pools is thus not uncommon and can occur through various exposure scenarios, such as open chlorine canisters, chlorine leak from pipes or reservoirs, inappropriate flushing of chlorination/sanitation lines, release of vapors from solid chlorine compounds, or mixing bleach (sodium hypochlorite, HOCl) with acids[1,4-11]. Mixing sodium hypochlorite with acids is a rare cause of chlorine exposure at swimming pools, however it does account for many household chlorine exposures[12].

Chlorine, once inhaled, dissolves in water and generates hydrochloric acid upon contacting moist mucous membrane. Toxicity of chlorine however is not limited to the effects attributable to hydrochloric acid because chlorine is approximately 20 times more toxic to the respiratory tract than hydrochloric acid[2]. Chlorine is a highly irritant gas with intermediate water solubility. Therefore, it can damage large airways as well as small airways and lung parenchyma. Toxicity following chlorine gas exposure appears to get worsened with longer duration and higher concentration of exposure[7]. With considerable consistency around the world, chlorine gas has a time-weighted average exposure standard of 0.5-1 ppm. However, one recent study showed that at the level of 0.5 ppm chlorine exposure could

Table. The severity of acute effects associated with approximately 1 hour of chlorine exposure[15]

Minimal effects 1 hour at 0.5-2 ppm	Significant effects 1 hour at 2-20 ppm	Severe effects 1 hour at ＞20 ppm	Very severe effects 1 hour exposure at ＞34 ppm
Strong odor, slight irritation of nose/throat/eyes	Burning of eyes or throat, some cough and choking sensation	Sensation of suffocation, chest pain, dyspnea, nausea, vomiting, horseness	Pulmonary edema, sudden death bronchospasm (closeure of larynyx)
1-hr minimal MEG 0.5 ppm (1.5mg/m^3)	1-hr significant MEG 2 ppm (5.8mg/m^3)	1-hr severe MEG 20 ppm (58mg/m^3)	Lethality has been reported after 1 hour 34-51 ppm

result in nasal irritation in individuals with seasonal allergic rhinitis[13]. The fatal dose ranges from 50 to 2,000 ppm[9]. One study with pigs demonstrated that exposure of 100-140 ppm for 10 mins, 5 of 6 animals died within 6 hours[14]. The severity of acute effects associated with approximately 1 hour of exposure is generalized below in conjunction with the military exposure guidelines (MEGs) provided in USACHPPM Technical Guide (TG) 230 (Table)[15]. The recommended criteria for treatment of acute chlorine inhalation was included in the table (from minimal effects to very severe effects). In our case, there was no enough data about the concentration of chlorine gas.

The basic mechanism of toxicity is related to the solubility of chlorine in water, with chlorine forming hydrochloric and hypochlorous acids, which subsequently undergo ionization. This reaction occurs in moist environments such as eyes, nasal mucosa, and respiratory epithelium. Injury begins with edema of the upper airway and lung parenchyma, followed by development of a celluar exudates in alveoli. As injury progresses, severe edema, hemorrhage, and destruction of the bronchiolar mucosa can develop[16]. Reported respiratory injuries following acute chlorine exposure include rhinitis, tracheobronchitis, pneumonitis, and pulmonary edema, diffuse bronchiolitis, acute respiratory distress syndrome[9,11,17]. Although the underlying pathophysiology of chlorine gas inhalation is obscure, the immediate reaction, with signs of bronchoconstriction, pulmonary vasoconstriction and hypoxia, suggests local release of stored mediators leading to mismatch of ventilation and perfusion. This notion is supported by the very early and sharp decline of lung compliance, which reflects air trapping with hyperinflation secondary to increased expiratory resistance. It is possible that flooding of the pulmonary parenchyma also contributed to arterial deoxygenation later in the course of injury, as indicated by increased pulmonary wet to dry ratios[18].

Occasionally, pulmonary dysfunction may develop subacutely or in multiple stages. Given the fact that both upper and lower respiratory tract irritation/dysfunction can be present, and the onset of toxic manifestations may vary greatly, inhalation of chlorine gas can pose diagnostic and therapeutic challenges. Late complications such as occupational asthma, reactive airway dysfunction syndrome, increased airway responsiveness, and decreased residual volume have been described[19,20]. Exposure to chlorine gas generated from sodium hypochlorite at swimming pools is uncommon. A search of the case registry database of the Taiwan National Poison Control Center did not identify such cases between 1986 and 2008. Parimon et al. had previously reported a 23-year-old man who developed diffuse brochiolitis after such an exposure[9]. In our index case, mild hypoxemia immediately developed after exposure to chlorine for 6 minutes. Initial chest radiograph revealed pneumonitis-like opacities over right middle and lower lungs. Her clinical manifestations improved 3 days later, yet

her chest radiograph abnormalities did not resolve until 8 days post-exposure. While the patient's clinical manifestations were largely similar to those observed in previously reported cases of chlorine exposure, the finding of acute lung injury that primarily involved right lung was uncommon and might be attributable to the higher amount of ventilation of right lung[21]. Other alternative causes, such as aspiration, were unlikely to explain the patient's findings.

Although chest radiographs revealed the presence of acute lung injury in the patient, the exact extent of injury was unknown because she declined chest CT scan, which is a more sensitive tool to detect inhalation injury of lung. The lack of CT image findings is the main limitation of this report.

Current treatment of acute chlorine exposure is symptomatic and supportive, including antitussive medications, inhaled and/or intravenous bronchodilators for bronchospasm, and supplemental oxygen. In animal models, treatment with systemic or inhaled corticosteroids immediately following high-level chlorine exposure had resulted in improved pulmonary and cardiovascular function; however the mortality rate was unaffected[22]. In another animal study with ventilated pigs, Wang et al. reported that the timing of corticosteroid inhalation might be an important factor in the management of chlorine inhalation[18]. Treatment with inhaled budesonide immediately or 30 minutes after chlorine lung injury had a significant beneficial effect, but treatment delayed for 60 minutes was not ineffective compared with a control group[10]. Treatment of acute chlorine injury with aerosolized terbutaline followed by aerosolized budesonide improved lung function. Combined treatment was more effective than treatment with either drug alone[23]. In previous animal study, inhaled budesonide 5 mg and intravenous betamethasone 5 mg were given 30 minutes after completion of chlorine gas exposure. These drugs were given every 1-2 hour for 15 hours and then every 4 hour for 8 hours (total 23 hours). The doses of inhaled budesonide and intravenous betamethasone were chosen as recommended for treatment of toxic gas exposure by the Swedish Poison Information Center[24]. In a previous literature, inhaled budesonide and neubulized sodium bicarbonate treatment could prevent extended hospital stay by accelerating symptomatic and functional recovery[25]. Although our case did show much improvement after receiving both β_2 agonist and steroid therapy, the efficacy of such treatment in human with chlorine poisonings has not been confirmed and needs further evaluation.

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氯氣暴露導致急性肺部傷害

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摘 要

在游泳池意外暴露氯氣並非太罕見，且可能因多種狀況產生，譬如氯氣由管路或儲存槽外洩、或不當的沖洗氯化或消毒管線。雖然混合漂白水 (過氯化鈉)及酸液導致之氯氣暴露並非少見的家庭意外事件，但類似的暴露很少發生在遊泳池。吸入氯氣後的臨床表徵可以從輕微的上呼吸道刺激到危及生命的毒性作用，如肺水腫或急性呼吸窘迫症候群。吾人謹報告一起在游泳池發生的因混合過氯化鈉與鹽酸導致的氯氣意外暴露事件。在七名受害者中，有六人呈現呼吸困難、乾咳、及喉嚨與眼睛刺激症狀，經治療後在一天內出院。然而一名15歲的女孩除了產生呼吸道刺激的症狀外，另有缺氧及主要影響右側中、下肺之急性肺傷害。經給予支氣管擴張劑及靜脈注射類固醇後，她於五天後出院。吾人謹討論她的臨床表徵，並回顧現有與氯氣吸入有關之文獻。