

Myocardial Injury Due to Inhalation of A Mixture of Sodium Hypochlorite and Hydrochloric Acid

Sodyum Hipoklorit ve Hidroklorik Asit Karışımının İnhalasyonuna Bağlı Gelişen Miyokard Hasarı

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ÖZ

Amaç

Sodyum hipoklorit (çamaşır suyu) ve hidroklorik asit (tuz ruhu) ülkemizde temizlik amacı ile sık olarak kullanılır. Bu iki sıvının karıştırılması sonucunda zehirli klor gazı ortaya çıkmaktadır. Klor gazının inhalasyonu, mukoz membranlarda iritasyon, pulmoner gaz değişiminin engellenmesi ve pulmoner ödem nedeniyle ciddi solunum sıkıntısına neden olabilir. Nadiren kardiyovasküler sisteme de zarar verebilir.

Olgu

Bilinen kalp veya solunum hastalığı teşhisi konmamış 81 yaşında bir erkek hasta acil servise klor gazı maruziyeti nedeniyle lakrimasyon ve burun akıntısı şikayeti ile başvurdu. Aktif kardiyak şikayeti olmayan hastada troponin pozitifliği saptandı. Takibe alınan hastada takiplerinde troponin değerinde artış olması ve akut koroner sendrom dışlanamaması nedeni ile hastaya yapılan koroner anjiyografide koroner arterler plaklı olarak izlendi. Troponin yüksekliği sodyum hipoklorit ve hidroklorik asit karışımının inhalasyonuna bağlıdır.

Sonuç

Klor gazına maruz kalma genellikle solunum sistemine zarar vermektedir. Bunun yanında, aritmi, akut koroner sendrom, kalp yetmezliği ve hatta ölüm gibi kardiyovasküler etkiler de görülebilir. Acil tıp doktorları klor gazına maruz kalan hastalarda kardiyovasküler etkilere dikkat etmeli ve kalp hastalığı öyküsü olmasa bile kardiyak hasarın gelişebileceğini düşünmelidir.

Anahtar Kelimeler: Sodyum hipoklorit; hidroklorik asit; troponin; miyokard hasarı

ABSTRACT

Aim

Sodium hypochlorite (bleach) and hydrochloric acid are chemicals, commonly used in household cleaning and can be dangerous when mixed with each other. As a result of the mixture, chlorine gas is released which is poisonous. Inhalation of chlorine gas may cause severe respiratory distress due to irritation of mucous membranes, impaired pulmonary gas exchange, and pulmonary edema. Rarely, it also damages the cardiovascular system.

Case

An 81-year-old male patient, who had not been previously diagnosed with heart or respiratory disease, presented to the emergency department with a complaint of lacrimation and nasal discharge due to chlorine gas exposure. He had troponin positivity. Coronary angiography was performed due to the subsequently troponin increase. Coronary angiography revealed plaques. The cause of troponin increase was associated with the inhalation of chlorine gas.

Conclusion

Exposure to chlorine gas is commonly manifested by damage to the respiratory system. However, cardiovascular effects such as arrhythmia, acute coronary syndrome, heart failure, or even death may also occur. Emergency medicine physicians should pay attention to these cardiovascular effects and consider that patients exposed to chlorine gas may experience heart damage even if they do not have a history of heart disease.

Keywords: Sodium hypochlorite; hydrochloric acid; troponin; myocardial injury

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Introduction

Chemicals are not only used in manufacturing or industrial fields, but they have also become a part of our daily life, particularly in household cleaning. However, chemicals may have negative consequences as a result of unconscious use. Sodium hypochlorite (bleach) and hydrochloric acid are two of these chemicals widely used household cleaning. Indeed, there is a perception that mixing sodium hypochlorite (bleach) and hydrochloric acid enables better cleaning in our region. As a result of the mixture, chlorine gas is released which is poisonous (1-2). In emergency departments, chlorine gas intoxication is the second inhaled poisoning after carbon monoxide intoxication (3).

Chlorine gas inhalation poisoning is mostly accidental. It may occur as a result of the preparation of swimming pool water, military exposures, industrial exposures, misuse of cleaning products and chemical terrorism (4-5).

Inhalation of chlorine gas mainly damages the respiratory system, while damage to other organ systems is rare (6). Since chlorine is water-soluble, it primarily damages the upper and lower respiratory tracts. In developed or developing countries, using the mixture of sodium hypochlorite with hydrochloric acid can cause mild mucosal irritation, acute lung injury, reactive airway dysfunction syndrome or even death (1).

Chlorine gas has well known cardiac effects such as sinus tachycardia, sinus bradycardia, extrasystole, myocardial infarction, and cardiac arrest (2,6,7).

Here, we presented a rare case of non-cardiac troponin elevation due to the inhalation of a mixture of sodium hypochlorite and hydrochloric acid.

Case Report

An 81-year-old male patient with no history of cardiac or respiratory disease admitted to the emergency department with complaints of lacrimation and nasal discharge after exposure to chlorine gas released by a mixture of sodium hypochlorite and hydrochloric acid for approximately 10 minutes. He had no other complaints. He had no history of medication use. His vital signs followed as; blood pressure of 108/72 mm Hg, heart rate of 98 beats/min, SPO₂ of 98%, temperature of 36,9°C. Respiratory and cardiovascular system examinations were normal. Chest X-Ray was obtained as normal. ECG was in sinus rhythm and there was no acute ischemic change (Figure 1).

Laboratory investigations revealed; creatinine 0,92mg/dL (0,72-1,25mg/dL), white blood cell count 7,78 K/ul (4,0-10,0 K/ul), C-reactive protein 1,1 mg/L (0-5mg/L), creatine kinase (CK)-MB 3,74ng/mL (0-5 ng/mL), troponin I 0,125ng/mL (0,02-0,06 ng/mL). Results of initial arterial blood gas analyses were; pH 7,28 (7,35-7,45), PO₂ 98 mm

Hg(80-100 mm Hg), PCO₂ 57,3 mm Hg(35-45 mm Hg), SO₂ 96,5%(95-98%) HCO₃ 23,6 mmol/L(22-26 mmol/L).



Figure-1: ECG

Echocardiography revealed a normal left ventricular ejection fraction with stage 1 diastolic dysfunction (without segmental or regional wall motion abnormality).

To prevent possible bronchospasm due to chlorine gas exposure, 2L/min nasal oxygen, 250 microgram inhaler ipratropium bromide and 200 microgram inhaler budesonide were administered. Since he had no chest pain and no evidence of ischemia on ECG, cardiac enzyme and ECG were monitored.

Cardiac markers and arterial blood gas sample results at the second hour of admission were: creatine kinase (CK)-MB 4,15, Troponin I 0,645ng/mL, pH 7.46, PO₂ 99 mm Hg, PCO₂ 32,9 mm Hg, SO₂: 98,5%, HCO₃ 24,6 mmol/L.

The patient was admitted to the coronary care unit because of suspicion of acute coronary syndrome. Acetylsalicylic acid (300 mg), clopidogrel (300 mg), ramipril (2,5 mg), atorvastatin (40 mg), and enoxaparin subcutaneous (6000 IU) was initiated. Coronary angiography was performed due to increased troponin. Coronary angiography revealed plaques. The cause of troponin increase was associated with the inhalation of chlorine gas.

After the troponin level was regressed the patient was discharged from the hospital on the third day.

Discussion

By mixing chemicals such as sodium hypochlorite and hydrochloric acid or applying them to the same surface consecutively, a chemical reaction develops, and chlorine gas is released which is poisonous (1-2).

Chlorine gas dissolves in mucous membranes in the respiratory tract and forms HCL and O⁻ radicals responsible for toxicity (8). It may cause the burning of the throat, eyes, and conjunctiva. The most common symptoms are cough, dyspnea, nausea, vomiting, headache, dizziness and palpitation. Myocardial infarction, non-cardiogenic pulmonary edema, acute respiratory distress syndrome

(ARDS), pneumomediastinum and even death can be seen (2-7). Our patient's complaints were lacrimation and nasal discharge. In a study of 1566 cases of acute chlorine gas exposure, respiratory complaints and eye-burning were the most common complaints. Nine of these cases exposed to chlorine gas poisoning resulted in death (9).

Inhalation of chlorine gas may progress to hypoxia and may cause free oxygen radicals and increased sympathetic activity (3). Chlorine gas affects nitric oxide signaling pathways that are anti-inflammatory, antithrombotic, antioxidant and causes endothelial disruption and damages the cardiovascular system. (10) In addition to the effects of nitric oxide pathways, excessive HOCl⁻ production after exposure to chlorine gas is thought to be the cause of damage to vascular tissues (11).

In the animal studies, it was determined that cardiovascular cell damage occurred due to the decrease in sarcoendoplasmic reticulum calcium ATPase (SERCA) activity after chlorine gas exposure and so cardiac damage markers were found to be high (12). Animal studies have shown that troponin I levels are high in animals with cardiovascular system involvement (13). Our patient had no chest pain but troponin levels were increased during the coronary care unit.

Inhalation of chlorine gas was considered as a stressor. So far, cardiovascular adverse effects have rarely been reported, although respiratory system damage due to chlorine gas is well documented and well known.

Conclusion

Exposure to chlorine gas is commonly manifested by damage to the respiratory system. However, cardiovascular effects such as arrhythmia, acute coronary syndrome, heart failure, or even death may also occur. Those side effects should be kept in mind, cardiac enzyme and ECG monitoring should be performed especially in elderly patients. Emergency medicine physicians should pay attention to these cardiovascular effects and consider that patients exposed to chlorine gas may experience heart damage even if they do not have a history of heart disease.

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