A Case Report: What is the Real Cause of Death from Acute Chlorine Exposure in an Asthmatic Patient?

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Abstract
This case report presents an acute and chronic inflammation process at the same time and resulted in death following exposure to chlorine gas. A 65-years-old woman died shortly after cleaning her bathroom with a mixture of various chemicals including bleach and an acid containing product. She was declared dead when she arrives to hospital. She is a non-smoker and has no significant medical history other than asthma. Toxicological analysis showed that paracetamol, methylprednisolone, venlafaxine and its metabolite O-desmethylvenlafaxine were detected in blood. Histological examinations showed edema, fresh intra alveolar bleeding and emphysematous changes in both lungs. Asthma and acute inhalation injury have a relatively lower mortality rates. However, the coincidence of both clinical presentations may be the cause of death.

Introduction

Chronic or acute airways diseases may cause similar clinical picture. This can cause problems in determining the cause of death. Numerous gases, smoke, and dust can cause damage in the lungs and other organs. These irritants can cause acute toxic inhalation in the respiratory tract. Moreover they can cause irritation in the upper respiratory tract, the eyes and other mucous membranes. Clinical symptoms are cough, stridor, and shortness of breath. Most of the patients recover without any complication. Chronic lung pathology can be seen in a small proportion of the cases in the long term, but mortality is usually low [1]. However, children, the elderly and people with underlying respiratory or cardiovascular disease compared to others in terms of progression to serious reactions and respiratory failure are at higher risk [2].

Asthma is a chronic inflammatory disease of the airways causing respiratory symptoms such as cough and dyspnea [3]. These symptoms may increase from time to time and the influence of different factors can cause acute symptoms. Anti-inflammatory treatment has been used effectively in asthma management recently and it lowers the mortality. In this report, we present a case due to acute chlorine exposure that shows an acute and chronic inflammation process at the same time and resulted in death.

Case
A 65-year-old woman died shortly after cleaning her bathroom with a mixture of various chemicals including bleach and an acid containing product. According to witnesses, her symptoms include cough, shortness of breath along with red tearing eyes. She is a non-smoker and has no significant medical history other than asthma. She was declared dead when she arrives to hospital.

The decedent was 155 cm tall and weighed 67 kg. The external findings were unremarkable. Internally the left and right lungs weighed 303 g and 421 g, respectively, and showed marked pulmonary edema, congestion and hyperemia. The heart weighed 270 g and the brain weighed 1056 g both organs were congested. The coronary artery showed no abnormal findings.

Histological examinations showed edema, fresh intra alveolar bleeding and emphysematous changes in both lungs, perivascular interstitial fibrosis in heart, and that brain, kidney and liver were congested.

Toxicological analysis showed that paracetamol (2300 ng/mL), methylprednisolone (274 ng/mL), venlafaxine and its metabolite O-desmethylvenlafaxine (133 ng/mL) were detected in blood. These drugs and their levels can be explained by the drugs she received for her asthma and depression.

Inhalated gas or particulates may cause respiratory tract diseases. Exposure to a toxic agent in this way generally appears during industrial or domestic accidents. One of the most common used toxic irritant gases is chlorine gas. Mixing sodium hypochlorite with any acid containing product generates chlorine gas. The respiratory damage of toxic gases is related to several factors. The most important factors are the concentration of gas and the degree of water solubility. Highly water soluble gas accumulates in the upper airways and can lead to more acute conjunctival, nasal and laryngeal symptoms. The less water soluble gas can reach the distal airways and alveoli. As chlorine gas water solubility at the medium level, it can cause acute injury to both upper and lower airways.

Discussion

Reactive airways dysfunction syndrome (RADS) is defined as asthma –like symptoms due to irritant gas inhalation and airway reactivity [1,2,4]. In a study which analyses emergency service admissions of 55 RADS cases, the presence of upper and lower
and the mechanism of death, existing pulmonary diseases should be investigated. Patients with a history of exposure to a toxic agent should be kept under observation for a sufficient time for possible severe clinical picture and respiratory failure. Treatment should be planned based on the severity of the findings.

More than 5000 people were exposed to chlorine gas in Graniteville train crash in 2005. However 9 of them were died at the scene and this is considered as “relatively lower death rate” [8]. On the other hand, since bathroom is a confined space and ventilation is poor, chlorine gas may reach lower respiratory tract. As in our case, the cause of death was acute inflammation due to acute chlorine gas inhalation on the ground of chronic airway disease.

In order to show histo-pathological changes in an acute lung injury case due to chlorine gas, bronchoscopic biopsies were taken post exposure 60 hours, 15 days, 2 months and 5 months in the study. Biopsies demonstrated that the most consistent findings were epithelial desquamation and sub epithelial hemorrhagic exudation [6]. The biopsies obtained in this study were taken from bronchial mucosa, so there was not shown alveolar damage. The most obvious signs of lung damage in chlorine toxicity is pulmonary edema [2,7]. Damage of pulmonary capillaries, resulting in hemorrhagic fluid deposited in the alveoli [2].

In our case, pulmonary histopathology demonstrated pulmonary edema, bleeding and fresh emphysematous changes in the alveoli. The latter is consistent with the presence of chronic airway disease. However, the first two symptoms are related to acute inflammatory airway disease and not to be found in chronic airway disease. As a result, chlorine gas inhalation causes damage in respiratory tract and pulmonary parenchyma. Although the clinical prognosis is good in most cases, the more severe clinical picture can be seen in elderly and patients suffering from chronic respiratory diseases. In the cases who have an history of chlorine exposure, in order to establish the cause and the mechanism of death, existing pulmonary diseases should be investigated. Patients with a history of exposure to a toxic agent should be kept under observation for a sufficient time for possible severe clinical picture and respiratory failure. Treatment should be planned based on the severity of the findings.

References