Chronic reactive airway disease following Chlorine inhalation lung injury

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Citation

M Nepal, R Heintzelman. *Chronic reactive airway disease following Chlorine inhalation lung injury*. The Internet Journal of Pulmonary Medicine. 2008 Volume 10 Number 2.

Abstract

Inhalations of smoke and toxic fumes from chemicals like chlorine are known to cause mild mucosal irritation with lacrimation, nasal congestion, nasopharyngeal edema, transient reversible reactive airways and sometimes acute respiratory failure in the setting of acute respiratory distress syndrome due to bronchospasm, pulmonary consolidation presenting with rapid onset symptoms of cough, wheeze and shortness of breath. Inhalations of these fumes have also been known to cause on rare occasions diffuse bronchiolitis and chronic respiratory sequelae including decreased lung function and persistence of asthma. We are reporting one such rare case report of acute inhalation lung injury that developed progressive shortness of breath and bilateral lung consolidation as a result of inhalation of chlorine fumes from bleaching agents and later recovered on high dose steroids slowly over months only to have chronic reactive airway dysfunction syndrome requiring bronchodilator therapy.

INTRODUCTION

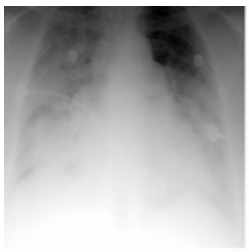
Chlorine gas is one of the most common substances involved in irritant inhalation exposures both occupationally and environmentally largely due to its use in household products such as bleach powder , in water purification and others as gassings in industrial bleaching operations, cleaning products, swimming pool chlorination mishap and storage tank failure. The largest sources of exposure to humans were in warfare but have ceased to be relevant now and exposure to chlorine in swimming pool and bleach agents are the most important causes of day to day occurrences.

CASE SUMMARY

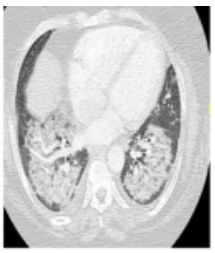
A 50 year-old African American female presented with shortness of breath and sharp pleuritic chest pain with nonproductive cough after doing laundry of clothes and cleaning bathroom where she had inhalation fumes from bleaching agents. She denied fever, nausea, vomiting and flue like symptoms. She had past medical history of hypertension asthma, and depression. She denied leg swelling, or recent travel and contact with pets and any other exposure recently. Physical examination revealed an obese lady with use of accessory muscles and in mild respiratory distress. Chest examination revealed tachypnea, bilateral coarse crackles mainly in bibasilar regions. Cardiac examination was normal including neurological and musculoskeletal examination.

An arterial blood gas evaluation on room air revealed pH of 7.44, PCo_2 55, and Po_2 of 68. Laboratory examinations including blood, sputum cultures were sterile and all immunological workup were negative and work up for infective and connective tissue disorder did not reveal any abnormality .Chest x-ray revealed bilateral consolidation and CT-chest revealed bilateral patchy consolidative changes with some ground-glass opacities. She was initially started on antibiotics .She finally underwent open lung biopsy which showed non-specific inflammatory changes. She was later changed to high dose steroids in view of inhalation pneumonitis to chlorine and she recovered gradually. On subsequent follow up examination, she remained symptomatic with severe reversible airway obstruction.

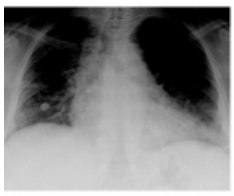
Figure 1



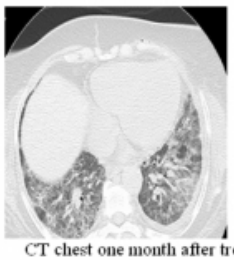
Chest Xray on presentation



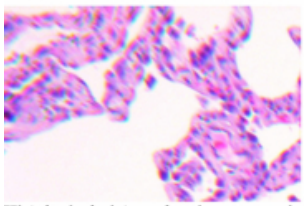
CT Chest on presentation



CXR one month after treatment



CT chest one month after treatment



Histological picture showing nonspeci-Fic inflammation

DISCUSSION

People usually present after inhalation of noxious smoke or chemicals and may present with cough, shortness of breath and wheezing [1,2] and sometimes lower epithelial damage with inhalation pneumonitis. Chlorine is a greenish-yellow, noncombustible gas at room temperature and atmospheric pressure and is a pulmonary irritant with intermediate water solubility that causes acute damage in the upper and lower respiratory tract . The earliest description of chlorine associated lung injury was reported by Lehmann in 1887 AD, who described hemorrhagic pulmonary edema in animals following chlorine gas exposure at concentration of 1ppm [3] . The pulmonary effects of chlorine in human was described by Berghoff in about 2000 victims of gas attack , 50% of soldiers had either bronchitis or emphysema where the subjective symptoms far exceeded than clinical findings [4] .

The immediate effects of chlorine gas toxicity include acute inflammation of the conjunctivae, nose, pharynx, larynx, trachea, and bronchi. Irritation of the airway mucosa leads to local edema secondary to active arterial and capillary hyperemia and congestion without mast cell degranulation [5] .

The long-term pulmonary sequelae of accidental exposure to chlorine in humans have been described in few reports. Kowitz et al described decrease lung volumes secondary to patchy atelectasis, nodular scarring and fibrosis [6] . Weil and associates followed 12 people over seven years and concluded that significant permanent lung term damage does not result from acute chlorine exposure [7] while Chester and colleagues found among 139 workers followed over many years only three had significant airway obstruction [8] .

Winternitz, et al found after massive chlorine exposure to dogs, those dying within 24 hours had tracheitis, bronchitis with peribronchial inflammation and focal necrosis of lungs while those dying 2 to 5 days after had pulmonary infection mainly pneumonia and bronchitis and the animals dying 15 to 193 days after had bronchitis and bronchiolitis [9] .

Inhalation injury occurs in three way- by cell injury and pulmonary parenchymal damage by irritation, hypoxemia by interruption of oxygen delivery, and end organ damage by systemic absorption through the respiratory tract. Ciliary function is impaired with secondary neutrophil infiltration, destruction of macrophages and bacterial invasion leads to pneumonitis. Hypoxemia results from a decrease in inspired oxygen concentration at the scene of injury, a mechanical inability to exchange gas because of airway obstruction or parenchymal pulmonary disease, and inhibition of oxygen delivery and tissue utilization by toxins. Our patient showed features of pneumonitis and reversible airway obstruction but the histopathology revealed areas of inflammation with some fibrosis but no evidence of BOOP. Any bacterial secondary infection was ruled out and there was also no evidence of co-incidental connective tissue diseases.

Bacterial pneumonia may complicate inhalation injury within 4-5 days of presentation. This additional cellular damage can cause significant mortality days to weeks after the initial injury. Most of the pulmonary damage is usually self-limited and resolves within 2-3 days. The degree of recovery depends on the extent of the pulmonary parenchymal injury and subsequent hypoxic damage to the organs. However, some authors documented long-term respiratory symptoms such as cough, wheeze, and shortness of breath even after mild inhalation injury, indicating a more prolonged nature of the lung injury [10,11] .

Roentgengraphic abnormalities are frequently delayed and may not manifest on the initial chest radiograph.

Radiographic evidence of pulmonary injury typically appears 24-36 hours after the inhalation. Decrease in pulmonary compliance, vital capacity, and functional residual capacity occurs on spirometry. Airway obstruction causes a decrease in forced expiratory volume in one second (FEV1) and peak flow [12,13,14]. Our patient showed some radiological improvement over several months.

Corticosteroids are attractive for suppressing inflammation and reducing edema but owing to reduced healing long-term use is discouraged. Oxygen is used in cases with significant inhalation injury along with bronchodilators in patients with bronchoconstriction.

Reports exist of residual reactive airway disease, bronchiectasis, bronchiolitis obliterans, and interstitial fibrosis [12,13,14,15,16,17]. However, the cause-and-effect relationships between toxic exposure and pulmonary sequelae remain controversial and are not validated throughout the literatures. Airway hyper-reactivity generally improves over several months following inhalation injury and resolution of pulmonary abnormalities in most individuals over the course of one week to one month following exposure. However, there is no uniform agreement throughout the literature.

CONCLUSION

Our patient initially presented with cough, progressive shortness of breath and chest pain. In view of infiltrate on chest x-ray and CT scan, she was started on antibiotics. However, blood and sputum cultures along with broncho-scopic examination did not reveal infective etiology and she did not improve, open lung biopsy was performed. Thus the biopsy revealed nonspecific inflammation and was negative for BOOP or other connective tissue diseases. Inhalation pneumonitis was diagnosed and managed with steroid and the patient improved slowly only to have reactive airways and is currently on bronchodilators.

ACKNOWLEDGEMENTS

We would like to appreciate our greatest thanks to Dr William McNamee and Dr James McMaster for help in preparing the manuscript.

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