ARDS following Inhalation of Hydrochloric Acid

DP Bansal*, Rahul Ambegaonkar**, P Radhika***, Manish Sharma***

Abstract

The clinical spectrum of Inhalation injury can range from mild cough to a devastating ARDS. We herewith present a patient who is a goldsmith by occupation and his work consists of dissolving gold in Hydrochloric acid. He had accidentally inhaled fumes of Hydrochloric acid and presented with cough and breathlessness, later on required mechanical ventilation for ARDS and improved. This highlights the importance of not to neglect mild symptoms like cough and dyspnea in such a scenario which may have some hidden catastrophe.

Introduction

Acute respiratory distress syndrome is a clinical syndrome of severe dyspnea of rapid onset, hypoxemia and diffuse pulmonary infiltrates leading to respiratory failure. ARDS is caused by diffuse lung injury from many medical and surgical disorders. The lung injury may be direct as occurs in toxic inhalation or indirect as occurs in sepsis. We report a case of acid fumes inhalation injury leading to ARDS.

Case Report

A 56 year old nondoabetic and nonhypertensive male from Chennai, goldsmith by profession, came with complaints of dry cough, breathlessness and chest discomfort of 4 hours duration. There was no history of fever, sputum production or hemoptysis.

The patient is a goldsmith by profession and teaches how to make gold ornaments, for that he has to dissolve gold in Hydrochloric acid (HCl) in an instrument with an exhaust fan. After putting gold in HCl, he switched on the motor and the fan threw the acid fumes on him, which he inhaled for a few minutes. This resulted in a bout of cough and breathlessness which worsened over next 4 hours and he came to hospital for out patient consultation, but was admitted after assessing his clinical condition. At the time of admission, the patient was tachypneic (respiratory rate 36/min), pulse rate was 110/min, all peripheral pulses were palpable, blood pressure was 130/70mm Hg in supine position in left arm. There was no pallor, icterus, clubbing, cyanosis, lymphadenopathy, pedal edema or engorged neck veins. Accessory muscles were active. SPO2 at room air was 84% which improved to 99% on O2 inhalation. Examination of respiratory system revealed bilateral basal crepitations and ronchi. Rest of the systemic examination was normal.

Laboratory examination was as follows-Hb-9.5gm/dl, WBC-15,800/cumm, RBC-4.6million/cumm, blood sugar-116mg/dl, blood urea-23mg/dl, creatinine-1.55mg/dl, SGPT-9U/L, Alkaline phosphatase-95IU. Bilirubin-0.7mg/dl, Na-135mosm/l, K-4.1mosm/l, Cl-92mosm/l. Chest X ray showed bilateral mld and lower zone fluffy shadows suggestive of pulmonary edema/ARDS (fig. 1). ABG showed pH of 7.380, PCO2-31.6, PO2-88.3, and HCO3-18.3. He was managed with antibiotics, steroids, bronchodilators and non-invasive ventilation and Oxygen. After 24 hrs, his respiratory symptoms worsened with respiratory rate of 48/min, Pulse-124/min and had cough with mucopurulent expectoration. ABG- pH-7.4, PCO2-35.1, PO2-58.8, HCO3-21.1. Repeat Chest X Ray showed bilateral diffuse fluffy shadows. He was mechanically ventilated on volume control mode, with FiO2-100%, Tidal volume-450ml, Rate-14/min and PEEP-7cm. In view of mucopurulent secretions he was started on Broad spectrum antibiotics (Amoxycillin-Clavulanuc acid and Levofoxacin). He was given Hydrocortisone, Doxophylline, Pantoprazole and Low molecular weight Heparin. He improved clinically and radiologically and was weaned off the ventilator (Total duration of ventilation was 5 days). Patient was kept under observation in wards for the next 4 days and discharged on tenth day of admission.

Discussion

Hydrochloric acid is a highly corrosive and strong oxidant reacting explosively with many compounds. These reactions result in release of chlorine gas which is responsible for free radical injury to the tissue.

In our case, the exposure occurred accidentally in a professional goldsmith when he mixed gold in HCl and the exhaust fan threw fumes of acid which he inhaled for few minutes.

The acid vapours are colourless and irritant hence can cause upper airways and eye irritation. In the moist lower respiratory tract, the fumes penetrate the bronchial and alveolar membranes generating free radicals and chlorine and causes acute lung injury and ARDS.

This patient was initially managed with high flow O2, bronchodilator, steroid and antibiotics. Patient was in Type-I respiratory failure. He was put on non-invasive ventilation for 24 hours. However, on the 2nd day of admission, patient started producing copious mucopurulent expectoration and worsening of hypoxia and radiological deterioration. Patient had to be ventilated. 2D Echo revealed normal LV function and CVP was 10-12 cm of NS.

Patient improved clinically and radiologically in 5 days (fig. 2) and weaned off from the ventilator on day 7 of hospitalization. At discharge, he had normal respiratory rate, normal oxygen saturation, normal chest X-ray and ABG.

Literature search failed to reveal reported case of HCL fumes inhalation resulting in ARDS. Hence we report uncommon presentation of our case. Our case is the first of its kind where patient inhaled HCl fumes, developed ARDS, mechanically ventilated and recovered.

So, this case highlights the importance of prevention of fumes/gas inhalation at workplace. Early recognition and respiratory support is must for these patients who develop ARDS due to toxic gas and fume inhalation. We believe that early use of steroids also help in the survival apart from mechanical ventilation and other supportive therapy.
References


