Severe lung injury following inhalation of nitric acid fumes

Banu Çevik, Ayşegül Çizen, Serhan Çolakoğlu, Ersan Şensoy

To the Editors,

Nitric acid (HNO_3) is a solution of nitrogen dioxide (NO_2) in water, commonly used as an industrial chemical and cleaner. Following the inhalation of nitric acid, seriousness of effects depends more on the highest concentration reached in the body and less on the duration of inhalation. We present the case of a patient who survived following inhalation of nitric acid fumes after developing diffuse pulmonary edema suggesting Acute Respiratory Distress Syndrome (ARDS). This case indicates to the physicians that HNO_3 inhalation may be initially asymptomatic with the delayed onset of severe symptoms. Intensive respiratory management and use of steroids can help in survival.

A 39-year-old male worker in a chemical industry was admitted to the Emergency Department with a complaint of shortness of breath which began four hours after accidental inhalation of nitric acid fumes from falling over the unlocked nitric acid container. He did not report any odor or oropharyngeal irritation during or immediately after exposure. He went to his home at end of the shift and was well during this period. On admission he was conscious, well oriented, severely dyspneic with respiratory rate of 35/min and

tachycardic with pulse rate of 120/min. He was transferred to intensive care unit (ICU) for further management and monitored non-invasively. His peripheral oxygen saturation on face mask was 89%. On auscultation, there were bilateral extensive rales. Rest of the systemic examination was normal. Chest X-ray confirmed pulmonary edema and a normal-size heart (Figure 1). Electrocardiogram revealed sinus tachycardia. The patient's clinical condition showed gradual deterioration and an arterial blood gas analysis revealed severe hypoxemia with pH: 7.31, PCO_2 : 50 mmHg, PO_2 : 41 mmHg, HCO_3 : 34.8 mEq/L, SpO_2 : 71.2%. He was intubated emergently for respiratory failure approximately six hours after exposure and copious serosanguinous secretion was aspirated from endotracheal tube. He was sedated and mechanically ventilated on pressure controlled mode with low tidal

Figure 1: Patient's chest X-ray four hours after exposure suggesting pulmonary edema.

Banu Çevik¹, Ayşegül Çizen¹, Serhan Çolakoğlu¹, Ersan Şensoy¹

Affiliations: ¹Dr. Lutfi Kırdar Kartal Education and Research Hospital, Anesthesiology and Intensive Care, Istanbul, Turkey.

Corresponding Author: Banu Çevik, Bagdat Cad., Nöte Sok, Yazıcıoğlu Apt, 10/12 Erenköy/Istanbul-Turkey, Postal code: 81400; Ph: +902163020523; Fax: +902164413900; Email: banueler@yahoo.com

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volume. A repeated ABG was performed one hour later and the patient's oxygenation had improved significantly. In addition to supportive ICU medications he received methylprednisolone, ipratropium bromide monohydrate+albuterol sulfate nebulization, budesonide nebulization and ampicillin/sulbactam sodium. Thorax computed tomography (CT) scan indicates bilateral increase in lung density suggesting pulmonary edema (Figure 2). He was ventilated with $\text{FiO}_2 > 0.5$ for more than 72 hours. The patient continued to require full ventilatory support during eight days with the short weaning processes and failed extubation attempts. Serial chest X-ray studies showed gradual recovery (Figure 3) and the patient could be weaned on the eight day and extubated. Bronchodilator therapy was continued up to 13th day of his admission and he was discharged to Pulmonary Clinic for further follow-up in fully conscious state. Pulmonary function test revealed moderate restrictive lung disease and the follow-up of the patient is still continuing because of the suspicion of pulmonary fibrosis.

Nitric acid is commonly used in various industries. Its accidental spillage generates oxides of nitrogen, including nitrogen dioxide, a potent pneumotoxin when inhaled. The clinical presentation manifests as rapidly progressive and often fatal non-cardiogenic pulmonary edema which is a consequence of increased microvascular permeability initiated by NO_2 -mediated capillary injury [1]. In report by Hajeal et al., electron microscopic findings of the lungs in a fatal NO_2 inhalation case revealed alveolar capillaries containing many degranulated neutrophils together with necrotic endothelial cells. Lungs showed bronchiolar epithelial necrosis, marked capillary engorgement and protein-rich edema fluid filling all alveolar spaces [2].

Existing case reports related with nitric acid related inhalation injury describe it as a rapidly progressive pulmonary edema of either acute or delayed onset.

Figure 2: Thorax CT scan of the patient showing the progressive impairment of the lungs.

Figure 3: Radiologic improvement of the patient.

Hajela et al. [2] reported the deaths of three US pulp-mill workers within 24 hours of exposure to the fumes. Another case involved a 25-year-old male worker who survived pulmonary edema that developed eight hours after exposure and was discharged on 11th day of his admission [3]. Murphy et al. [4] also reported a fatal pulmonary edema and circulatory collapse 53 hours after occupational exposure to nitric acid. Treatment is largely supportive and the use of nitric oxide (NO) has not been investigated in nitric acid induced pulmonary edema. Inhalation therapy with NO was previously tried in two cases but the patients were lost due to refractory respiratory failure [4, 5].

In our case, the patient developed symptoms approximately four hours after exposure and once symptoms occurred clinical condition deteriorated rapidly leading to ARDS. Invasive ventilatory support, symptomatic treatment and the use of corticosteroids contributed to the patient's survival. In respect of data about nitric acid inhalation induced pulmonary edema; there is no promising therapeutic intervention so the aim must be directed towards prevention of nitric acid exposure.

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The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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