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# Severe Pulmonary Edema due to Nitric Acid Fume Inhalation at Home

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**Abstract** A 65 year-old man presented to the Emergency Department (ED) with dyspnoea at rest, peripheral cyanosis, general paleness and a frothy fluid escaping from the nose and mouth. He also showed severe burns in his hands. He had been trying to clean a copper lamp with a 50–70% nitric acid solution at home. He was transferred to the intensive care unit (ICU). Non-invasive mechanical ventilation with PEEP was immediately applied. Treatment was started with 60 mg of furosemide intravenously (IV), and prednisolone 250 mg IV four times daily. As he responded, he was weaned off non-invasive ventilation and administered high-flow oxygen via a facemask (10 L/min). At day 5, he was transferred to the general ward. The patient was discharged from hospital at day 10 without serious pulmonary complications. He was followed-up as an outpatient for three months and respiratory function tests did not show any impairment.

***Keywords:*** *nitric acid, pulmonary edema, inhalation*

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## 1. Introduction

Nitric acid (HNO3) is one of the strongest acids and a strong oxidizing agent. It is commonly used in production of ammonium nitrate fertilizer and explosives and the chemical cleaning of metals. When it contacts metals, it generates nitric oxide (NO) and nitrogen dioxide (NO2) which can cause extensive inflammation of the lower respiratory tract [1]. The few reported cases of toxicity are characterised by a rapidly progressive and often fatal, noncardiogenic pulmonary edema, presumably as a consequence of increased micro vascular permeability associated with capillary injury [2,3].

The level of toxicity of nitric acid inhalation seems to depend on duration and intensity of exposure. Exposure to 100–150 ppm for 30 to 60 minutes produces mild clinical toxicity but levels of 200 to 700 ppm may produce fatalities after a few minutes [4]. In milder cases, patients may stay asymptomatic during several hours (up to 24 hours). After severe exposure, progressive pulmonary edema develops instantaneously [5]. A few cases have been described in the last years, mainly related to industrial inhalation [5,6,7,8,9].

## 2. Case Presentation

A 65 year-old man, smoker, presented to the Emergency Department (ED) with a complaint of severe shortness of breath. He had no relevant past medical history. He reported he was trying to clean a copper lamp with a 50–70% nitric acid solution at home, by putting both the chemical and the lamp into a bowl. Though he was wearing a mask, he experienced throat irritation and severe dyspnoea a few minutes after opening the can of nitric acid.

Approximately 40 minutes later, he arrived at our ED. Upon arrival, he was conscious but showed dyspnoea at rest, peripheral cyanosis, general paleness and a frothy fluid escaping from the nose and mouth. He had no evidence of caustic injury to his mucous membranes or face but showed quite extensive burns on his hands, presumably associated to direct skin contact with the acid, since he was not wearing gloves. Blood pressure was 150/90 mmHg and heart rate 110 beats per min. Auscultation of the chest revealed bilateral pulmonary rales. Baseline laboratory tests showed: hemoglobin (Hb): 17,7 g/dL, hematocrit (Ht): 47%, white blood cell count (WBC): 13000/mm3 with a normal WBC differential count. Test results for liver and renal function and serum electrolytes were normal. Oxygen pulse saturation (SpO2) on arrival was 83%. The chest computed tomography (CT) scan on admission showed diffuse bilateral pulmonary infiltrates with alveolar pattern (Figure 1). He was transferred to the intensive care unit (ICU).

An arterial blood gas (ABG) was obtained upon arrival at the ICU, with the patient on 100% oxygen via nonrebreather mask: pH of 7.34, pO2 = 40 mmHg, pCO2 = 45 mmHg, HCO3 = 22 meq/L, and non-invasive mechanical ventilation with PEEP was immediately instituted.

He required rapidly progressive increment in FlO2, to 70%. PaFIO2 was 250. Treatment was started with 60 mg of furosemide intravenously (IV), and prednisolone 250 mg IV four times daily. He developed metabolic acidosis

and marked increase (x 3) of liver enzymes during the first 48 hours but the renal function remained normal. The patient slowly improved, was weaned off non-invasive ventilation, and administered high-flow oxygen via a facemask (10 L/min). At day 5 after ICU admission, he was transferred to the general ward. A new chest CT scan at day 7 showed bilateral perihilar consolidation and ground-glass attenuation, suggesting the persistence of acute alveolar injury, but in a lesser degree than the CT at admission, while the patient was hospitalized the spirometry showed an obstructive pattern.

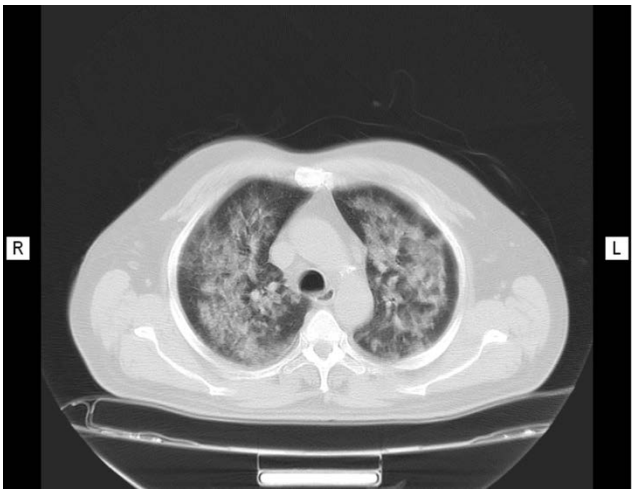


**Figure 1**.

The patient was discharged from hospital at day 10 without serious pulmonary or systemic complications. A thoracic CT scan at 30 days of the initial inhalation showed significant improvement (Figure 2). The patient was followed-up as an outpatient and 3 months later he did not show any impairment on pulmonary function tests, measured by normal spirometry.

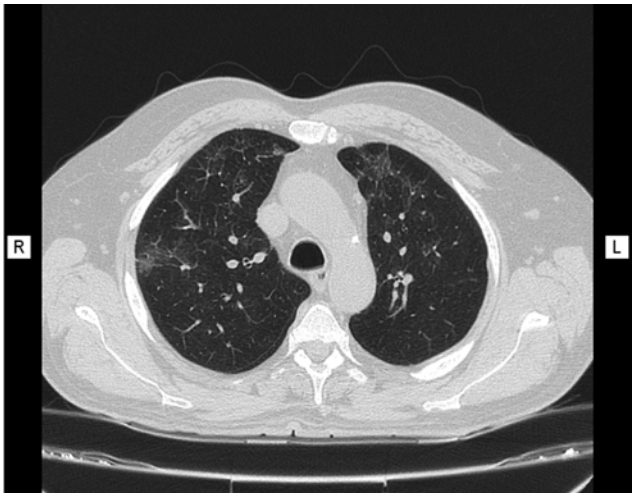


**Figure 2**.

## 3. Discussion

Nitrogen dioxide and other nitrous oxides are rapidly generated when nitric acid comes in contact with the air [2]. Inhalation effects are due to a mixture of nitric acid vapor and nitrogen oxides (NOX), mainly nitrogen dioxide (NO2) and nitric oxide (NO). The toxic effects of nitric acid cannot be isolated from those of its side products, since the liberation of NOX is immediate after the contact with air, organic matter and some metals [10].

Toxicity may cause delayed-onset lung injury which can take several hours to develop. This is one of the reasons why inhalation of gases and vapors originating from nitric acid is extremely dangerous as there is no early warning of the potentially serious damage (as it can be seen with chlorine and ammonia). Early symptoms have been reported associated to exposure to very high concentrations which often cause immediate fatalities [5,6,7,8,9].

Our patient had respiratory symptoms as soon as he started handling the nitric acid. This is uncommon and could be related to some previously undiagnosed bronchial hyperactivity since he was a heavy smoker. The resulting short time of exposure, the fact that he was wearing a mask -although it was verified later that it was not working properly- and the rapid access to the ED, were probably were probably the reasons why the episode resulted in a severe but non-fatal self-limited pulmonary edema. He suffered quite severe burns in the hands but did not experience any injury to the eyes, skin, or nose mucous membranes, which coincides with the toxicity mechanisms described for nitric acid and its side products. HNO3 can be corrosive to the eyes, skin, nose, mucous membranes, respiratory tract, gastrointestinal tract, or any other tissue with which it comes in direct contact. But as nitrogen oxides are insoluble in water, the vapours and mists that emanate from HNO3 may cause less irritation on conjunctiva and oropharynx.

We have found only seven previous reports of acute lung injury due to the inhalation of nitric acid (Table 1) [5,6,7,8,9,11,12,13]. Including the present case, there are 13 patients available for analysis. Most of the previously reported cases (except one) are associated with an occupational exposure.

Nitric acid is used in the manufacture of dyes, fertilizers, celluloid and lacquers. Silos can also contain nitrogen dioxide because of plant decomposition in the presence of high nitrate levels [14]. Fire fighters may also be exposed to nitrogen oxide during fires taking place in chemical plants [4]. In spite of its widespread use, fatal toxicities are very rare. In the US, the Toxic Exposure Surveillance System data collected by US poison centres reports only 4 fatal cases in almost 25 years and only one a fatality due to pure HNO3 inhalation [8]. Of all the published cases, 7 survived (including our patient), which results in a mortality rate of 46%.

Necropsy studies in five cases revealed firm, heavier-than-normal lungs which released foamy fluid from cut section. Microscopic evaluation of the lungs demonstrated congestion of alveolar capillaries and larger vessels. A protein-rich edema fluid and hyaline membranes were found in alveolar spaces [5,7,8]. The pathogenesis of the development of pulmonary edema is not fully characterized, although it is clear that the nitric acid-mediated injury involves the damage of the alveolar-capillary membrane with the consequent increase of micro vascular permeability. The role of neutrophils and serum derived mediators in the development of this injury is not clear. The electron microscope examination in the case reported by Hajela et al [8] showed altered neutrophils and necrotic cells into alveolar capillaries with

small and large serum proteins revealed by immunohistochemistry in the edema fluid and hyaline membranes. This could suggest a direct micro vascular injury, leading to increased permeability and could justify the potential therapeutic effect of the administration of corticosteroids [5].

**Table 1.**

| Author | year | Country | n | Source of exposure | Time of onset of symptoms | Mechanical Ventilation | Outcome |
|---|---|---|---|---|---|---|---|
| Hajela | 1990 | Canada | 3 | Occupational (explosion of a tank containing nitric acid) | 4-6hs | yes | Deceased |
| Jayalakshmi | 2009 | India | 3 | Occupational (cleaning a nitric acid container in a fertilizer company) | 1 immediate<br>2 N/A | 1 yes, 2 no | Survived (full recovery) |
| Pillai | 2005 | India | 1 | Occupational (cleaning aluminum components in metal industry) | 8 hs | yes | Survived (full recovery) |
| Çevik | 2011 | Turkey | 1 | Occupational (contact with a nitric acid container) | N/A (hours) | yes | Survived (moderate restriction in PFT) |
| Murphy | 2010 | USA | 1 | Occupational (cleaning a treatment tank in a waste-treatment facility) | 3-4 hs | yes | Deceased |
| Shin | 2007 | Korea | 2 | Occupational (mixture of nitric acid and hydrofluoric acid fumes in an industrial electroplating plant) | 1-2hs | Yes (1 + ECMO) | 1 Survived<br>1 dead |
| Bur | 1997 | Austria | 1 | Domestic (cleaning a chandelier by putting nitric acid in a bowl) | 30 min | Yes (+ ECMO) | Deceased |
| Current case | 2013 | Argentina | 1 | Domestic (cleaning a metallic lamp with nitric acid in a bowl) | 10 m | Non invasive ventilation | Survived |

Treatment of nitric acid induced lung injury is largely supportive once a patient develops respiratory failure. There is no specific literature that investigates the effectiveness of the different treatment modalities used in the reported cases (mainly prednisone and nitric oxide).

The symptoms described in the published cases were dyspnoea, tachypnea, cyanosis, bronchospasm, haemoptysis, tachycardia, and chest pain associated with the development of pulmonary edema. Most of the patients did not show long term sequelae, such as bronchiolitis obliterans [15]. The results of spirometry and diffusion capacity in our patient were within normal limits one month later as it was described in the few cases that had a mid-term follow-up [9,13].

This case highlights the risk of handling products containing nitric acid at home. This is the first report of non-invasive mechanical ventilation in such cases. We believe it was effective because of the relatively limited extension of the pulmonary injury, probably associated with the short term exposure and the rapid access to the emergency department. The early use of steroids may have contributed to the successful outcome and prevented the development of bronchiolitis obliterans or pulmonary fibrosis.

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