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Case Report

Severe chemical pneumonitis by vanadium pentoxide responded well to aggressive steroid therapy

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ABSTRACT

The optimal treatment of acute chemical pneumonitis remains controversial. Here we report a healthy man with severe chemical pneumonitis caused by accidental inhalation of vanadium pentoxide. He presented with acute respiratory distress and received aggressive steroid therapy on arrival. Pulmonary symptoms and chest X-ray were improved dramatically the next day. The beneficial effect of steroid therapy for such a critical patient may outweigh the infection risk following inhalation of relative sterile material. We suggest early and aggressive steroid therapy may help shorten the disease course.

1. Introduction

Vanadium pentoxide (V_2O_5) is often used as catalysts to produce sulfuric acid by converting sulfur dioxide to sulfur trioxide [1]. Occupational exposure of vanadium takes place during processing and refining vanadium ores, manufacturing of vanadium-containing products, burning of fuels rich in vanadium content, and cleaning of oil boilers [2]. Inhalation is the main route of entry into the body [3]. Occupational exposure to vanadium pentoxide produces mucosal irritation of eyes, gut, and respiratory tract [4]. Respiratory tract, including larynx, bronchioles, and alveoli, is the most sensitive target following inhalation of vanadium pentoxide [5]. Acute inhalation of vanadium pentoxide may cause chemical pneumonitis, pulmonary edema, and acute tracheobronchitis [3]. However, the treatment of acute inhalation injury by vanadium pentoxide is not well established in the medical literature. Here we report such a critical patient who improved dramatically in a day after early and aggressive steroid therapy.

2. Case presentation

The 39-year-old man with no systemic disease had a ten pack-year smoking history but quitted ten years ago. He worked in a chemical factory that produced sulfuric acid by utilizing vanadium pentoxide as a catalyst, and he was assigned to clean the oil tank containing the orange color ash of vanadium pentoxide for 3 h. He developed a dry cough and progressive shortness of breath 10 h later, although he had worn an N95 and a face mask when cleansing. Two other coworkers on the scene also had mild pulmonary discomfort but recovered spontaneously. Subsequently, he reported a worsening cough with yellow-orange sputum and noisy breathing

Abbreviations: CT, Computer tomography; CXR, Chest X-ray; FEF25-75%, Forced mid-expiratory flow.

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sound. Fever up to 38 ˚C, itchy throat, general malaise, and dizziness were noted. He denied eye irritation, rhinorrhea, hemoptysis, chest pain, nausea, abdominal pain, and leg edema. He had no known history of allergy to foods or medications.

Initial vital signs included blood pressure of 172/95 mmHg, heart rate of 127 beats per minute, temperature of 35.2 ˚C, respiratory rate of 20 breaths per minute, and oxygen saturation of 61 % by pulse oximetry while breathing ambient air. Physical exam was remarkable for tachypnea, labored breathing with use of accessory muscles, and diffuse bilateral lung crackles. White blood cell count was 24,600/μL (4000–10000/μL) with 89.2 % neutrophils. C-reactive protein level was 2.8 mg/dL (< 0.3 mg/dL) and Creatine phosphokinase level was 552 U/L (45–163 U/L). An artery blood gas under non-rebreather oxygen mask disclosed a pH of 7.422 (7.35–7.45), PaCO2 of 44 mmHg (35–45 mmHg), and PO2 of 119.9 mmHg (75–100 mmHg). A chest X-ray (CXR) showed bilateral alveolar infiltrates predominantly in the peripheral and upper lung fields (Fig. 1A) 16 h after exposure. Chest computed tomography (CT) further revealed peribronchial ground-glass opacities in addition to alveolar infiltrates 21 h after exposure (Fig. 1B).

Based on the history and investigations, a diagnosis of acute chemical pneumonitis was made. He was initially treated in the emergency room with intravenous methylprednisolone 80 mg along with inhalation of dexamethasone 5 mg, ipratropium bromide and salbutamol (Combivent) 2.5 ml, and norepinephrine 1mg, followed by intravenous dexamethasone 5 mg and inhalation of dexamethasone 10 mg 3 h later. Besides, Augmentin 1.2 g was given for only one dose. After transferring to the ordinary ward, he received intravenous methylprednisolone 40 mg every 8 h and oxygen therapy (10 L/min) with a non-rebreather mask. On the next day, he felt better and could tolerate nasal cannula oxygen therapy (3L/min) with oxygen saturation of 95–97% by pulse oximetry, and follow-up CXR showed marked regression of pulmonary infiltrates (Fig. 1C). Subsequent CXR showed further improvement on day 5 (Fig. 1D). No bacterial growth was observed in the sputum culture. Intravenous methylprednisolone was shifted to oral prednisolone 30 mg

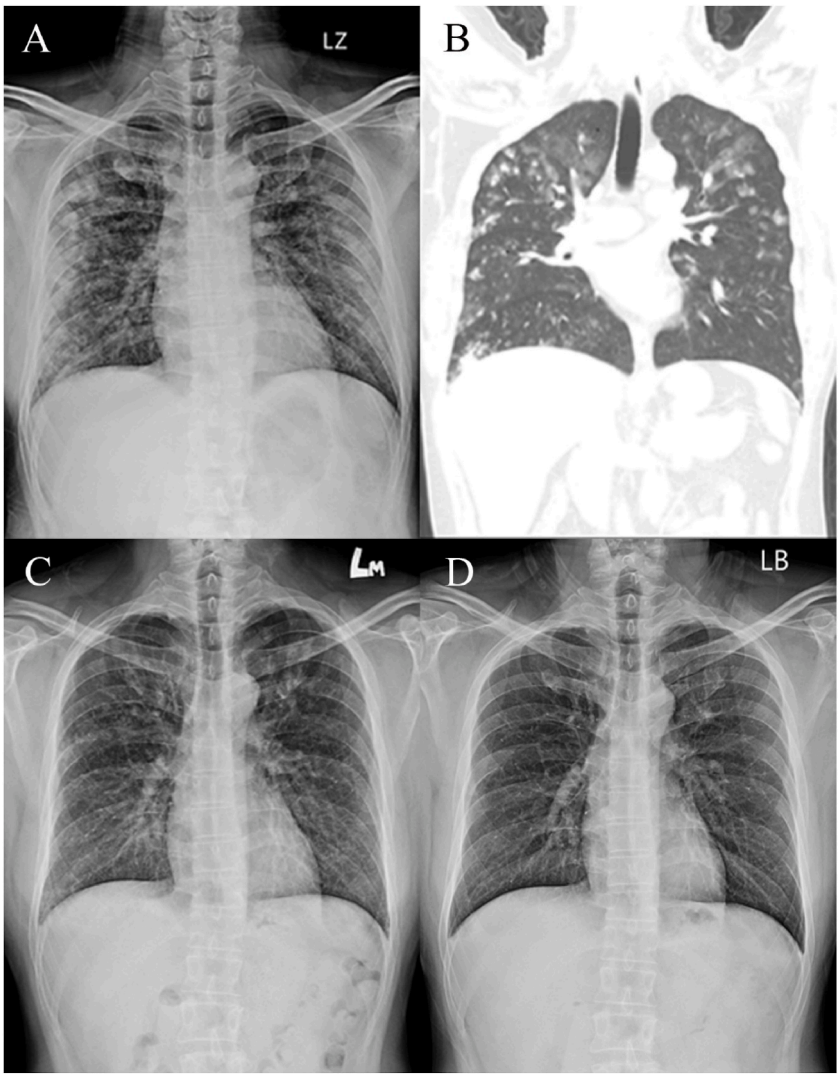

Fig. 1. (A) CXR showed bilateral alveolar infiltrates predominantly in the peripheral and upper lung fields (B) CT further revealed peribronchial ground-glass opacities in addition to alveolar infiltrates (C) Subsequent CXRs showed marked regression of pulmonary infiltrates on day 2 and (D) on day 5.

daily on day 4, and pulmonary function test on that day was generally normal except for a reduction in forced mid-expiratory flow (FEF25-75%) (Table 1).

On day 5, oxygen therapy was not required and subjective symptoms were remarkably improved. He was discharged and continued steroid therapy with oral prednisolone 30 mg daily for five days. Bronchoalveolar lavage was not performed due to rapid symptom improvement. Six weeks and seven months after discharge, there were no pulmonary symptoms; CXRs were normal; pulmonary function continued improving, but with mild small airway dysfunction.

3. Discussion

Chemical pneumonitis caused by inhalation of industrial dust has been widely addressed since the era of industrial revolution. However, acute vanadium pentoxide inhalation injury is rarely reported. Besides, its pathogenesis has not been well understood yet. Vanadium pentoxide is slightly soluble in water, with the solubility of 1 g in 125 ml water, thereby resulting in a delayed onset of the pulmonary symptoms following inhalation [5,6]. Acute exposure during cleansing of boiler ash may cause injury to upper and lower respiratory systems and even systemic toxicity [3]. Moreover, the disease severity depends on host factors, aerodynamic features, pH, water solubility, concentration of inhaled toxin, and duration of exposure [6,7]. In our case, the patient developed severe dyspnea 10 h after exposure to vanadium pentoxide. Chemical pneumonitis was confirmed on CXR and chest CT, with small airway injury in pulmonary function tests subsequently. This was consistent with a literature report that acute inhalation lung injury was observed in 17 men following vanadium exposure for 2 h. A reduction in FVC, FEV1, and FEF25-75% within 24 h was found in all, and their lung function did not return to pre-exposure levels by the eighth day [8]. By contrast, FVC and FEV1 were normal after steroid therapy for three days in our patient. He did not undergo bronchoscopy. However, neutrophils predominated in the bronchoalveolar lavage fluid after inhaling vanadium compounds (V^{5+}) in other human and monkey studies [9,10]. Besides, aspiration of vanadium compounds (V^{5+}) has been shown to cause lung apoptosis and inflammation through generation of reactive oxygen species (ROS) in a mouse model [11]. This indicates that acute pulmonary inflammation by oxidative stress is most likely implicated in the pathogenesis of chemical pneumonitis by vanadium compounds. A similar inflammatory response can also be induced by aspiration of gastric acid [12,13].

Symptoms and signs improved rapidly following early and aggressive steroid therapy in our patient. However, the treatment of acute vanadium pentoxide inhalation injury has rarely been reported. There are even no treatment guidelines for acute chemical pneumonitis. In a mouse model, high-dose steroid therapy with dexamethasone reduced acute inflammation if administered within 6 h after chlorine inhalation [14]. In a human study, corticosteroids were similarly effective against severe pulmonary injury following acute inhalation of nitrogen oxide [15]. However, the effect of steroid therapy for inhalation of other chemical agents was inconclusive due to small case number and unclear indication in a review study [16]. In addition, in 71 children with mild to moderate chemical pneumonitis following aspiration of hydrocarbon, steroid therapy for three days was not effective when compared to placebo [17]. We speculate that mild diseases may heal spontaneously and secondary bacterial infection may be a concern following aspiration. By contrast, our patient was in a critical condition following inhalation of relative sterile boil ash and had a dramatic improvement of hypoxia and pulmonary infiltrates on CXR one day after steroid administration. Therefore, the beneficial effect of steroid therapy may outweigh the infection risk in such a condition.

4. Conclusions

The pathophysiology and treatment of acute lung injury following inhalation of vanadium pentoxide remain controversial. We report such a severe case of chemical pneumonitis which developed soon after exposure and improved dramatically in a day after steroid therapy. It highlights the benefit of early and aggressive steroid treatment for severe inhalation injury by vanadium pentoxide.

Availability of data and materials

All data and material are available for sharing if needed.

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Table 1
A reduction in forced mid-expiratory flow (FEF25-75%) on day 4, but impaired FEF25-75% in 6 weeks and 7 months.

	Day 4	6 weeks	7 months
FVC percent of predicted	81 %	84 %	89 %
FEV1 percent of predicted	80 %	80 %	84 %
FEV1/FVC ratio	82 %	79 %	79 %
FEF25-75% percent of predicted	75 %	67 %	70 %
FEF50% percent of predicted	88 %	78 %	84 %
PEF percent of predicted	90 %	92 %	106 %
DLCO percent of predicted	94 %	92 %	
DLCO/VA percent of predicted	94 %	127 %	

Ethics approval and consent to participate

Not applicable.

Consent for publication

Informed written consent was received from the patient for publication of the manuscript.

CRediT authorship contribution statement

Ying Ju Chao: Writing – original draft, Formal analysis, Data curation, Conceptualization. **Ping Tsun Lai:** Writing – review & editing, Supervision, Resources. **Yen Ting Lai:** Writing – review & editing, Visualization. **Che Jen Chao:** Writing – review & editing.

Declaration of competing interest

The authors declare that they have no conflict of interest.

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