柴油吸入引發吸入性肺炎合併急性呼吸窘迫症候群及肺纖維化-案例報告

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摘要

由於直接吸入氣溶膠或吸入液體柴油會導致嚴重的化學性肺炎甚至急性呼吸窘迫症候群。通過嘔吐物間接攝入柴油也可導致誤吸。本病例報告描述了一名 57 歲的男性患者他在從油箱中抽取柴油時不慎被柴油窒息，出現胸部不適，呼吸急促和發冷的症狀。到達急診室後，患者被診斷為吸入性肺炎和急性呼吸衰竭。不幸的是，這名患者的病情在兩天后惡化，他隨後發展為急性呼吸窘迫症候群。值得慶幸的是，患者倖免於難，並成功脫離了呼吸器。然而，患者後續出現了肺纖維化的後遺症。該病例強調了考慮肺炎患者詳細職業暴露史的重要性。確定任何潛在的有害物質暴露至關重要，尤其是在涉及化學品或燃料的行業工作的患者中。早期識別和管理吸入性肺炎，對於預防嚴重併發症，如急性呼吸窘迫症候群的發展至關重要。此外，需要長期監測可能出現的任何延遲後遺症。

關鍵詞：柴油吸入、急性呼吸窘迫症候群、肺纖維化

Aspiration Pneumonitis Complicated with Acute Respiratory Distress Syndrome and Pulmonary Fibrosis due to Diesel Fuel Inhalation : A Case Report

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Abstract

Diesel fuel inhalation can cause severe chemical pneumonitis or acute respiratory distress syndrome (ARDS) through direct inhalation of aerosol or aspiration of liquid diesel. Indirect ingestion of diesel oil through vomitus can also lead to aspiration. This case report describes a 57-year-old male patient with chest discomfort, shortness of breath, and chills after accidentally choking on diesel fuel while siphoning it from a tank. Upon arriving at the Emergency Room, the patient was diagnosed with aspiration pneumonitis and acute respiratory failure. Unfortunately, the patient's condition worsened two days later, and he developed ARDS. The patient survived the episode and was successfully weaned off the ventilator. However, the patient did develop pulmonary fibrosis as a sequelae. This case emphasizes the importance of considering a detailed occupational exposure history in patients with pneumonia. Identifying any potential exposure to harmful substances is crucial, especially in patients who work in industries involving chemicals or fuels. Early recognition and management of aspiration pneumonitis are essential to prevent the development of severe complications such as ARDS. In addition, long-term follow-up is necessary to monitor for any delayed sequelae that may arise.

Keywords: Diesel fuel inhalation, Acute respiratory distress syndrome, Pulmonary fibrosis

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I. Introduction

Aspiration of foreign substances can lead to severe pulmonary disorders and even death. Accidental aspiration of hydrocarbons like diesel fuel is uncommon. While aspiration of substances like micro-bacteria or gastric contents is more common, accidental aspiration of hydrocarbons like diesel fuel can also occur. Aspiration of diesel can lead to exogenous lipoid pneumonia, a form of chemical pneumonitis that can progress to fetal acute respiratory distress syndrome (ARDS)[1–2]. Diesel fuel has a low viscosity and is highly volatile, allowing it to rapidly reach the alveoli without triggering a significant cough response. Siphoning of diesel fuel from tanks is a common practice in developing countries and can pose a significant occupational hazard. ARDS is a heterogeneous clinical syndrome characterized by severe hypoxemia and decreased lung compliance caused by inflammatory pulmonary injury that leads to increased pulmonary vascular permeability in the pulmonary blood vessels. In this case report, we describe a 57-year-old male patient who presented with chest discomfort, shortness of breath, and chills after accidentally choking on diesel fuel while siphoning it from a tank. Despite surviving the episode, he developed pulmonary fibrosis as a sequelae.

Early recognition and management of aspiration pneumonitis are crucial in preventing the development of severe complications such as ARDS. Long-term follow-up is necessary to monitor for delayed sequelae that may arise, such as pulmonary fibrosis.

II. Case

A 57-year-old male patient presented to the Emergency Room with chest discomfort, shortness of breath, and chills after attempting to siphon the diesel fuel from a tank while working in this evening. The patient had no fever, cough, diarrhea, nausea, vomiting, headache, or neck stiffness. He was a non-smoker and had no other significant personal or systemic diseases. Upon examination, the patient had a body temperature of 37.2°C, a heart rate of 85 beats per minute, a respiratory rate of 24 breaths per minute, and blood pressure of 122/71 mmHg. The patient was alert, but had mild coarse breathing sounds in both lungs. Other systemic examination findings were normal. Laboratory tests showed normal hemoglobin, platelet count, renal and liver function, cardiac enzymes, and urine analysis. However, leukocytosis (WBC: 10.2 $x10^3$/ uL), lactic acidosis, elevated C-reactive protein (CRP: 62.4 mg/L) and procalcitonin (PCT: 12.6 ng/ml). A serum Pro-BNP test (460 pg/mL) did not suggest acute heart failure (as a value > 900 ng/mL is typically indicative of acute heart failure). A cardiac echocardiogram revealed adequate cardiac performance. A chest radiograph showed infiltrations in the bilateral middle and lower lung fields. The patient’s shortness of breath worsened, and he developed acute respiratory failure. An endotracheal tube was inserted, and mechanical ventilation was initiated. The patient was diagnosed with aspiration pneumonitis and acute respiratory failure and was transferred to the intensive care unit (ICU). He was treated with extended-spectrum antibiotic therapy with piperacillin-tazobactam, hemodynamics support, mechanical ventilator, and nutrient support. However, the patient's condition worsened as his blood pressure dropped, and he required massive fluid resuscitation and vasopressor levophed treatment. Two days later, the chest x-ray showed bilateral lungs infiltrations (Figure 1), and arterial blood gas showed PaO2/FiO2 ratio (P/F ratio:134) less than 200, indicating acute respiratory distress syndrome (ARDS) development. Ventilator setting with low tidal volume and high positive end expiratory pressure (PEEP) was initiated, and high concentration oxygen, sedative agent ativan, muscle relaxant tracrium, and corticosteroid solumedrol were used. Cultures of blood, urine and sputum did not reveal any pathogen at first, but subsequent sputum culture revealed S. maltophilia. Bacterial pneumonia complicated the patient's condition along with chemical pneumonitis. During the ICU admission, the patient’s ARDS improved after antibiotics therapy with piperacillin-tazobactam, aggressive hemodynamics support, and adjusted mechanical ventilation management. However, the patient

experienced a right pneumothorax episode as a ventilator complication, and a chest tube was inserted for treatment. After the patient's general condition improved, he was transferred to the Respiratory Care Center (RCC) due to difficulty weaning off the ventilator. More than one month after admission, the patient was weaned from ventilator, survived and dischargedHowever, he developed pulmonary fibrosis (Figure 2) sequelae, and he was followed up at the Department of Chest Medicine

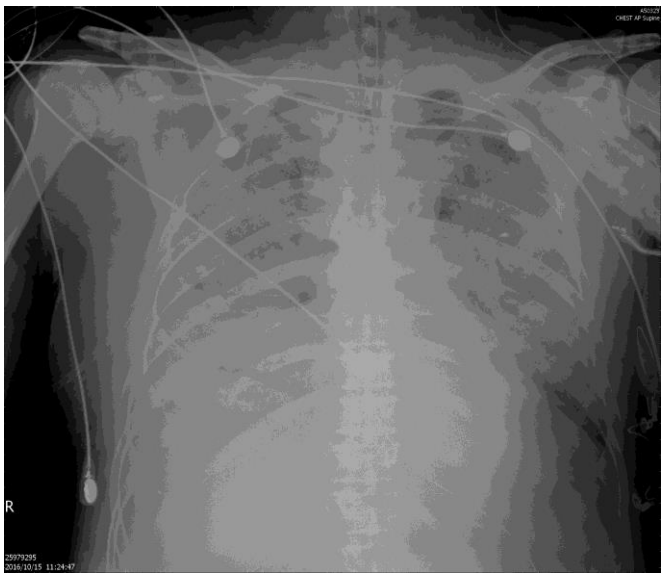

Fig.1 *An X-ray of the chest reveals diffuse alveolar and interstitial infiltration over both lung fields, which suggests the presence of Acute Respiratory Distress Syndrome (ARDS).*

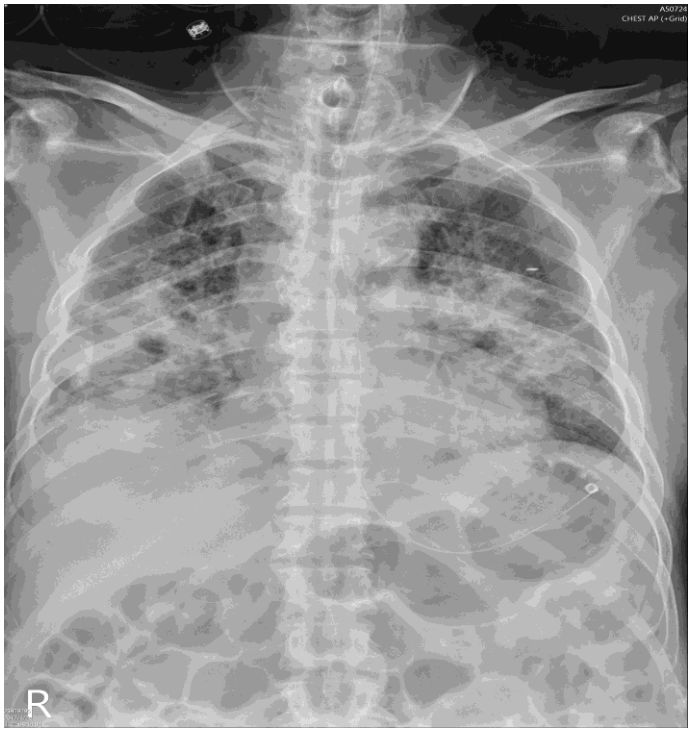

Fig.2 *The X-ray of the chest displays widespread infiltration and reticular shadowing in both the lower and upper lung fields, indicating the possibility of pulmonary fibrosis. Additionally, a tracheostomy is observed to be in place.*

III. Discussion

Inhalation can have adverse pulmonary consequences, such as the entry of gastric or oropharyngeal fluids containing bacteria and/or exogenous substances into the lower airways, which can cause ”aspiration pneumonitis" due to gastric acid or exogenous substances, or "aspiration pneumonia" due to bacterial infection[3,4]. Hydrocarbon aspiration is a rare cause of acute respiratory failure and even death, typically affecting children under the age of six who accidentally ingest hydrocarbon-containing products. However, occupational exposure to hydrocarbons, such as those encountered by fire eaters, airport ground staff, miners, and construction workers, can also cause hydrocarbon aspiration. Direct aspiration of hydrocarbon containing products, such as petroleum distillates, kerosene, gasoline, furniture polish, and cosmetics like baby oil and massage oil, can cause severe lipoid pneumonitis with poor outcomes[5–6]. Hydrocarbon aspiration can most commonly affect the central nervous system, gastrointestinal tract, and lungs. Mineral oil pneumonitis is an inflammatory, granulomatous, and fibrotic reaction of the pulmonary system to the aspiration of mineral oil. Hydrocarbons with lower viscosity and surface tension are more prone to aspiration. Diesel, one of several distillate oils prepared by fractionation of crude oil, is commonly used as a vehicle fuel. Ingestion of diesel fuel, which can occur following siphonage, has also been reported to cause acute hydrocarbon toxicity[7]. The clinical presentation of hydrocarbon-induced pneumonitis is often nonspecific and includes dyspnea, cough, chest pain, and hemoptysis. Diesel fuel has a low viscosity and is highly volatile, making it a potential hydrocarbon to cause severe progressive lung damage and fatal ARDS[8–9]. The histopathological feature of oil aspiration is the presence of lipoid cells or foamy cells in fine state of subdivision. The lung biopsy was not performed in this patient. In this case, the patient developed chemical pneumonitis after diesel aspiration, complicated with bacterial pneumonia and ARDS, which progressed rapidly after admission.

ARDS is an acute, diffuse, and inflammatory form of lung injury associated with a large number of etiologies[10] and a high mortality rate[11]. In this case, a diagnosis of moderate ARDS was made based on chest x-ray findings of bilateral opacities, a PaO2/FiO2 ratio between 100 to 200 on ventilator settings that include a Positive end expiratory pressure (PEEP) > 5 cmH2O, and the respiratory distress of patient not fully explained by cardiac failure or fluid overload, to exclude hydrostatic pulmonary edema, in accordance with the clinical diagnosis by the Berlin definition of ARDS[12]. Treatment of ARDS patients requires superfine supportive care, including intelligent hemodynamic management, nutritional support, use of sedatives and neuromuscular blockade, and meticulous mechanical ventilator support. Early application of low tidal volume ventilation, high PEEP, and permissive hypercapnia strategies improves the mortality rate in patients with ARDS[13]. In this case, the patient was prescribed sedative agents, neuromuscular blockade, corticosteroids and administered protective lung strategies with low tidal (4-6 ml/kg) and high PEEP soon after the ARDS developed. There is limited and controversial data on the role of corticosteroids and antibiotics in the outcomes of ARDS induced by diesel aspiration pneumonitis. Cardiac abnormalities, such as arrhythmia and cardiomyopathy, have been reported in cases of hydrocarbon intoxication[14]. However, the case presented here did not result in lethal arrhythmia despite hydrocarbon intoxication. In ARDS, various mechanisms can trigger an early activation of the fibro-proliferative response, such as persistent neutrophilic infiltration into the lung parenchyma, which predominates throughout the course of the disease.

Pro-inflammatory cytokines, including TNF-α, IL-1β, IL-2, and IL-6 are released by inflammatory cells and are increased within 24 hours of the onset of acute lung injury, persisting in fetal cases. TNF-α and IL-1β are chemotactic and mitogenic for lung fibroblasts, stimulating collagen synthesis by these cells. In mice exposed to diesel exhaust, increased collagen deposition and hydroxyproline residue have been observed, and repetitive exposure has led to tissue remodeling in the lung, resulting in fibrotic foci and smooth muscles.[15–16]. The patient presented in this case developed pulmonary fibrosis as a consequence and was followed up at the

Department of Chest Medicine. Although there are sparse clinical reports of pulmonary fibrosis due to diesel aspiration, it is a rare and lethal consequence of severe ARDS induced by diesel fuel.

IV. Conclusion

This case highlights that mineral oil pneumonitis can occur due to accidental diesel fuel inhalation while siphoning which can result in a critical and potentially lethal clinical condition. Pulmonary damage in diesel aspiration is rapidly progressive and extensive, leading to ARDS, and in severe cases, survivors may develop pulmonary fibrosis, a serious lifelong lung disease. We suggest that intensive care givers should be aware of this rare and fatal condition and start aggressive management early to improve the outcome.

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