

Case Report

HYDROCARBON PNEUMONITIS DUE TO ACCIDENTAL CONSUMPTION OF DIESEL-A RARE CASE REPORT

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ABSTRACT

56 year old male automobile worker had accidental aspiration of diesel which was mistaken for drinking water. After a day he developed vague ill health followed by malaise, cough and fever.

Chest X ray showed bilateral mid to lower zone airspace opacities. Sputum gram stain and culture were inconclusive. ABG analysis showed hypoxemia. Patient received antibiotics, bronchodilators, steroids and symptomatic and supportive care without any mechanical ventilator support. After 9 days patient had symptom resolution with marked regression in chest x-ray opacities and discharged. On follow up after 2 weeks chest x ray also showed clearance of airspace opacities.

Key Words: Diesel Aspiration, Hydrocarbon Pneumonitis

INTRODUCTION

Hydrocarbon pneumonitis due to aspiration of diesel during siphoning of diesel though rare can happen in people who are working in the automobile field. The case report belongs to a person who accidentally poured diesel into the mouth contained in a mineral water bottle thinking it as drinking water. It was in 1897, that Hamilton first described pneumonitis due to hydrocarbon aspiration¹. The rarity of bilateral pneumonitis as a consequence of accidental diesel aspiration prompted this report of a 54-year-old automobile worker who developed hydrocarbon pneumonitis following consumption of diesel which was mistaken as water in a mineral water bottle. Case reports pertaining to accidental aspiration of diesel are rare. Diesel aspiration leading to bilateral pneumonitis is also documented very rare in our country but middle lobe aspiration is reported commonly.

CASES

56 years old never smoker nonalcoholic diabetic, hypertensive working in the automobile field had accidental ingestion of diesel while at work. Actually patient poured the diesel by mistake as the diesel in the mineral water bottle was mistaken for the drinking water; which the workers usually use to collect drinking water. Suddenly he had burning sensation of oral cavity with sneezing and coughs. After a day patient developed malaise and vague ill health followed by fever which was of low grade and associated breathlessness. 3rd day after incident he had generalised weakness with persistence of fever and gone to near medical centre. On 6th day he had hemoptysis. Same time he had nausea and loss of appetite. Patient was on treatment for diabetes mellitus, systemic hypertension and coronary artery disease. Physical examination revealed moderately built and nourished patient without cyanosis, clubbing, Anemia or jaundice. Body temperature was 39.2⁰c. Blood pressure was 130/90 mm Hg, heart rate was 68 per minute and respiratory rate was 28 per minute. Respiratory system examination revealed decreased breath sounds in the infra scapular area bilaterally. Bilateral crepitations were heard in both lung fields especially in the infra scapular and infra axillary area but without any rhonchi. No bronchial breathing or pleural rub were there. Routine blood examination showed Hb 10.4 gm per dL, TC 7600, with 95% polymorphs and 5% lymphocytes and ESR was 135 mm in the first hour. Sputum gram stain no organism identified. Blood culture was sterile. ECG showed sinus tachycardia. Renal and liver function test were within normal limits. FBS/PPBS were 100/206 mg% respectively.

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Chest X ray revealed bilateral air space opacities mid and lower zones (figure 1). HRCT thorax (figure 2) showed ill defined areas of opacities in central regions of both lungs predominantly bilateral lower lobes, middle lobe and lingula with multiple tiny lymph nodes in prevascular, pretracheal, pulmonary window and bilateral hilar region. Opacities were predominantly surrounding the broncho vascular regions. Sputum culture and blood culture were sterile. ABG PH =7.4, Pao_2 = 55mm Hg, $PaCo_2$ = 31 mmHg bicarbonate = 22 mmol, SpO_2 =85%.

Patient received antibiotics, steroids, insulin, bronchodilators, supplemental oxygen and other symptomatic and supportive treatments. Patient had marked improvement in chest X ray with symptom resolution after 9 days. He was discharged with advice for follow up after 2 weeks.

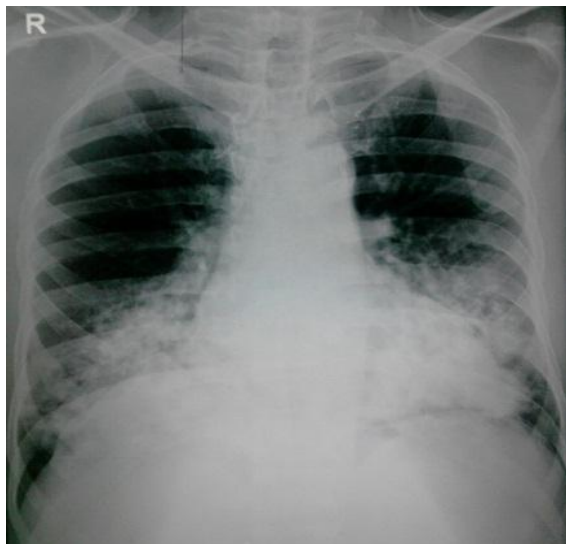


Figure 1: CXR PA Hydrocarbon Pneumonia

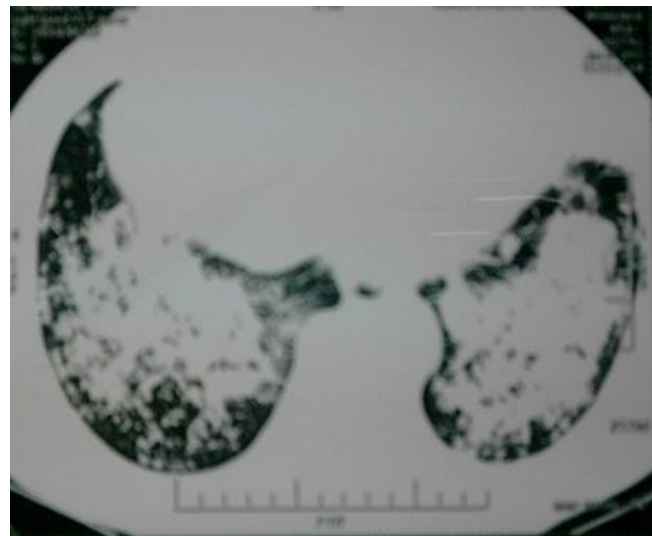


Figure 2: HRCT Thorax Showing Bilateral Lower Lobe Pneumonitis

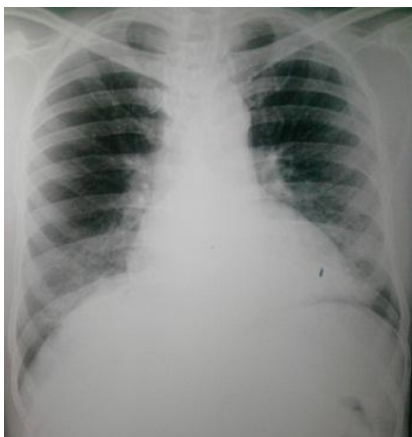


Figure 3: Chest X Ray after A) 2 Weeks From Discharge



B) After 4 Weeks from Discharge

DISCUSSION

Aspiration of hydrocarbons causes respiratory pathologies from simple symptoms to critical result. Hydrocarbon pneumonitis, known also as fire-eater pneumonia, always develops after aspiration of low viscosity, volatile hydro carbides. Accidental aspiration of diesel can result as a result of direct inhalation

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or may follow ingestion (Kirsanov, 1970). It is occasionally seen in adults, especially drivers, farmers and auto mechanics (Carlson, 1981 and Sertogullarindan *et al.*, 2011). It may produce pulmonary oedema, atelectasis and consolidation often the most common presentation of hydrocarbon toxicity (Olsen and Poisoning, 1967). The toxic potential of hydrocarbons is directly related to their physical properties. Highly volatile with low viscosity and lower surface tension hydrocarbons are more likely to be inhaled or aspirated into the respiratory system (Dyer, 1990). The hydrophobic nature of them let them to penetrate deep in to the tracheobronchial tree. Direct contact with alveolar membranes can lead to hemorrhage, hyperemia and edema. They provoke the activation of macrophages, leading to an increased release of cytokines and a prolonged inflammatory reaction (Burkhardt *et al.*, 2003). The type II pneumocytes are most affected, resulting in a decreased surfactant production.

The surfactant layer, which is composed of lipids, is made soluble by hydrocarbons, causing further damage (Giammona, 1967). The end results of hydrocarbon aspiration are necrotizing chemical pneumonitis, atelectasis, pleural effusion and hypoxemia (Olsen and Poisoning, 1967). The clinical presentation of hydrocarbon pneumonitis is often nonspecific and includes breathlessness, cough, chest pain and hemoptysis (Selikoff, 1962). The central nervous system, the gastrointestinal tract and the lungs are most commonly involved (Rouse *et al.*, 1974). The radiological lesions are generally out of proportion to the clinical findings. Radiographic findings include unilateral or bilateral lung consolidation, pneumatoceles, pleural effusion, atelectasis and spontaneous pneumothorax (Franquet *et al.*, 2000). The treatment is usually empirical, as there is insufficient data advocating the utility of corticosteroids and antibiotics (Steele *et al.*, 1972). The inflammatory nature of the disease suggests that steroid treatment can be useful in these patients. However there have been conflicting reports about the effectiveness of steroids (Steele *et al.*, 1972). Studies reported that some complications may develop, which included abscess, broncho pulmonary fistula, bacterial super infection and acute respiratory failure and death. Present case highlights the fact that hydrocarbon pneumonitis can occur due to accidental diesel aspiration in workers in the automobile field not only with siphoning but also with accidental ingestion. Physicians should be aware of hydrocarbon pneumonitis following aspiration of diesel.

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