

# A rare case of lipoid pneumonia due to diesel aspiration

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## ABSTRACT

Acute exogenous lipoid pneumonia is a rare chemical pneumonia following aspiration of volatile hydrocarbon compounds. In this case report, we present a 63-year-old male patient who developed acute exogenous lipoid pneumonia following accidental aspiration of diesel oil from the fuel tank of his vehicle with a hose. The patient presented with sore throat, cough, dyspnoea and tachypnoea. Posteroanterior chest radiography showed paracardiac infiltrations in the right lower and left lower lung fields. The patient presented again on the 21<sup>st</sup> day after aspiration with cough and sputum production. Chest X-ray showed a cavitory lesion with air-fluid level in the lower right probe. One month after the treatment, radiological regression of the consolidations was observed in the chest radiograph. In conclusion, this rare disease occurs after inhalation of hydrocarbon products. Symptoms and radiological findings develop rapidly and may lead to serious pulmonary complications and sequelae.

**Keywords:** Lipoid pneumonia, aspiration, diesel

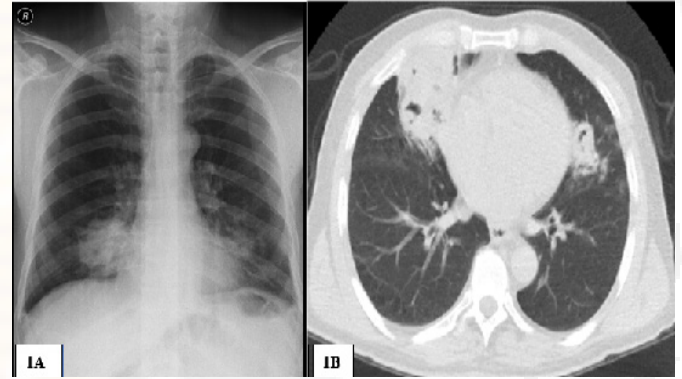
## INTRODUCTION

Lipoid pneumonia (LP) is a type of pneumonia where lipids (fats) are found in the lung tissue. It can result from the inhalation or aspiration of oily or fatty substances. Lipoid pneumonias are classified as endogenous and exogenous.<sup>1</sup> Exogenous lipoid pneumonia occurs when external substances, such as diesel, paraffin oil, mineral oil or vegetable oils are inhaled or aspirated into the lungs.<sup>2</sup> Inhalation or consumption of diesel may lead to hydrocarbon pneumonitis, which is a type of exogenous LP.<sup>3</sup> This report presents a case of a patient who accidentally consumed diesel while siphoning.

## CASE

A 63 year old man, with no previous medical illness reported experiencing a seven day sore throat, cough and dyspnea. Seven days before, he had aspirated accidentally a water glass of diesel fuel, while attempting to siphon it from the fuel tank of his vehicle. His respiratory rate was 30 breaths/min. His pulse rate was 110 beats/min, the temperature was 38°C, blood pressure was 110/60 mmHg, oxygen saturation 94% under room air and the patient did not need oxygen therapy. Patient was dyspnoeic but there was no cyanosis. Physical exam was notable for normal oral mucosa without lesions. In the auscultation, mild rales were heard bilaterally. His total leucocyte count was  $7 \times 10^3$  cells/ $\mu$ L with a differential of 81% polymorphonuclear leucocytes and 11% lymphocytes. Arterial blood gas analysis was within the reference range. C-reactive protein (CRP) level was 102 mg/L. The posteroanterior chest radiograph showed opacifications and infiltrations paracardiac

right lower and left lower lung fields (Figure 1A). Computed tomography of the thorax revealed bilateral consolidation in the lower lobes (Figure 1B).



**Figure 1.** A. Posteroanterior chest radiograph demonstrating opacifications and infiltrations paracardiac right lower and left lower zone B. Computed tomography scan of the thorax demonstrating right lower and left lower lobe consolidation

The patient's bacterial sputum culture was negative. Since Türkiye is an endemic region in terms of tuberculosis, sputum acid-fast organism test was performed and it was negative. He was diagnosed with hydrocarbon pneumonitis and treated with piperacillin/tazobactam (3x4500 mg/day, IV), clindamycin (3x600 mg/day, IV) and oxygen supplementation. He was discharged from hospital after nine days of treatment. After two weeks patient presented with recurrence of cough associated with expectoration. His chest radiograph showed

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features of worsening consolidation and a cavitary lesion with an air-fluid level over the lower zones of right lung (Figure 2).



**Figure 2.** Posterioranterior chest radiograph demonstrating features of worsening consolidation and a cavitary lesion with an air-fluid level over the lower zones of right lung

His leukocyte counts was  $14 \times 10^3$  cells/ $\mu$ L, CRP level was 123 mg/L. Bacterial sputum culture and acid-fast organism test were negative. During follow-up the patient's sputum culture was negative for *Mycobacterium tuberculosis*. There was no history of drug use that would cause immunosuppression and serology tests were regular.

Meropenem  $3 \times 1000$  mg/day IV was implemented for 14 days. A follow-up computed tomography of the thorax (Figure 3) a month after discharge showed radiological regression of consolidations.



**Figure 3.** Computed tomography scan of the thorax demonstrating resolution of the pulmonary infiltrates

## DISCUSSION

LP is a chronic inflammation of the lung parenchyma caused by the accumulation of fat-containing substances within the distal airways. This accumulation leads to an inflammatory response that inhibits exchange of gases.<sup>4</sup> LP are classified as endogenous and exogenous. Endogenous LP, also known as cholesterol pneumonitis occurs due to the distal obstruction of the airways by tumors and suppurative processes.<sup>5</sup> Exogenous LP is occurred by the aspiration or inhalation of

animal, mineral or vegetable oils. Petrol, diesel and gasoline are the most used hydrocarbons in daily life. Inhaling these hydrocarbons, either intentionally or accidentally, can result in hydrocarbon pneumonitis.<sup>6</sup> In our case, the patient accidentally aspirated diesel when siphoning it from the fuel tank of his vehicle. The airways do not readily absorb hydrocarbons after being inhaled can easily reach the alveoli and this causes structural damage in the alveoli. These pathologic changes are caused by the inflammatory response that develops due to increased cytokine release from macrophages and leads to chemical pneumonia.<sup>7</sup> Acute LP is typically characterized by common clinical symptoms such as cough, dyspnea, fever, and chest pain.<sup>8</sup> Radiological abnormalities are bronchovascular markings, consolidations with an air bronchogram which may become cavitated and/or develop pneumatoceles, ground-glass opacifications, pneumothorax, and pleural effusion or pneumo mediastinum. It usually takes between two weeks and eight months for radiological opacities to resolve after clinical recovery.<sup>9</sup> The blood or lungs have no specific tests to detect the level of diesel exposure. The diagnosis is typically based on the patient's anamnes.<sup>10</sup> The identification of lipid-laden macrophages in bronchoalveolar lavage fluid and the presence of alveoli in bronchoscopic lung biopsy results in a definitive diagnosis.<sup>11</sup> In our cases, we could not do bronchoscopy because there is no bronchoscopy unit in our hospital and applied non-invasive diagnostic technique like sputum. Treatment options for LP are not well-defined, and reports of cases only provide an overview of their progress. The primary focus of treatment is on avoiding continuous exposure and providing support. Hydrocarbon pneumonitis cannot be treated with antibiotics, but the majority of patients receive antibiotic treatment due to the inability to differentiate between hydrocarbon and superimposed pulmonary infections. Systemic corticosteroids limits the inflammation and ongoing fibrosis but are supported only by case reports. The other treatment options include bronchoalveolar lavage, immunoglobulins and surgery.<sup>12</sup> Our patient was treated with antibiotic and oxygen supplementation. He had complete clinical and nearly complete radiological improvement two weeks after discharge from hospital.

## CONCLUSION

Hydrocarbon pneumonia, resulting from the accidental inhalation of diesel fuel, can lead to extensive and severe lung damage. It's important for healthcare professionals to be aware of the potential respiratory hazards associated with hydrocarbon exposure and to consider this diagnosis in individuals with relevant symptoms and exposure history.

## ETHICAL DECLARATIONS

### Informed Consent

The patient signed and free and informed consent form.

### Referee Evaluation Process

Externally peer-reviewed.

### Conflict of Interest Statement

The authors have no conflicts of interest to declare.

### Financial Disclosure

The authors declared that this study has received no financial support.

### Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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