

# A case of lipid pneumonia associated with occupational exposure to solvents in a dry-cleaning worker

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## Keywords

Dry cleaning, lipid pneumonia, mineral oils, occupational exposure, solvents.

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## Abstract

Lipid pneumonia can develop from exposure to different types of oil, but occupational exposure is rare. A 58-year-old woman was referred to our hospital for patchy airspace opacities in the lungs with lower lobe predominance on chest computed tomography. She was diagnosed with non-Hodgkin's lymphoma seven years ago, but was in complete remission. She had mild cough and sputum, but no history of taking any lipid-containing agents. The bronchoalveolar lavage fluid revealed lipid-laden macrophages with Oil Red O staining, which led to the suspicion of lipid pneumonia. Re-evaluation of her personal history revealed that she was a dry-cleaning worker who worked with organic solvent sprayers. Her condition was successfully managed with corticosteroids and avoidance of further occupational exposure to the substance. This rare case of occupational exogenous lipid pneumonia in a dry-cleaning worker suggests the importance of considering a patient's occupational history during diagnosis.

## Introduction

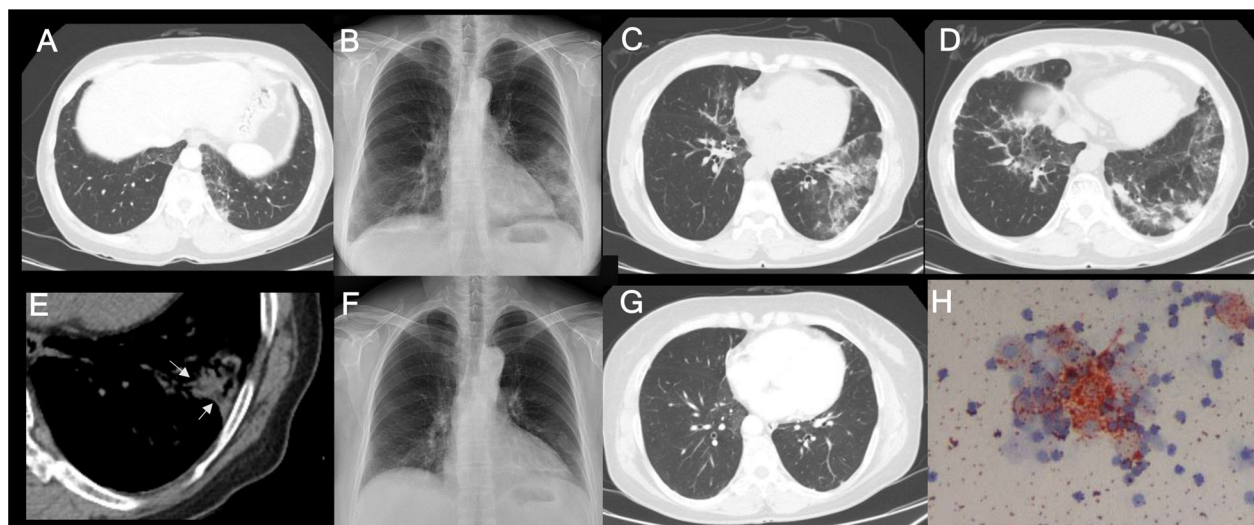
Exogenous lipid pneumonia resulting from the aspiration or inhalation of various types of lipids is an uncommon inflammatory lung disease [1]. It is majorly associated with the use of mineral oils, which are a mixture of inert, long-chain, saturated hydrocarbons obtained from petroleum [2]. Occupational exposure to these mineral oils is an uncommon cause of lipid pneumonia [1–3]. Moreover, limited data are available on cases of occupational lipid pneumonia [2,3]. Here, we report a rare case of lipid pneumonia that occurred in a dry-cleaning worker because of repeated exposure to a hydrocarbon-based solvent spray.

## Case Report

A 58-year-old woman, without a history of smoking, presented with lung abnormalities detected incidentally on chest computed tomography (CT). She had non-Hodgkin's lymphoma (NHL) seven years prior and was in complete remission after chemotherapy. In November 2018, a new focal subpleural ground-glass opacity with fibrotic density in the left lower lung was observed on chest CT performed during a regular check-up for NHL (Fig. 1A). The patient showed no

evidence of NHL recurrence and did not report any respiratory symptoms initially. Follow-up chest CT performed six months later showed no significant changes in the pulmonary lesions. In November 2019, she was admitted to our hospital for cough, mucoid sputum, and left lower pleuritic chest pain that persisted for two months. She did not have any underlying health conditions, except NHL, and was not taking any other medications, including lipid-containing agents. Her body temperature was normal and oxygen saturation in room air was 98%. Chest auscultation revealed inspiratory crackles in both the lower lungs. A physical examination revealed otherwise normal findings, with no signs of connective tissue disease. The results of complete blood cell count, C-reactive protein levels, liver function test, and serum biochemical and immunological analyses, including autoantibodies for collagen vascular diseases, were also unremarkable.

A chest radiograph showed bilateral patchy alveolar opacities, mainly in the lower lobes of the lungs (Fig. 1B). Chest CT revealed multiple areas of subpleural and peribronchial patchy consolidations with ground-glass opacity and interlobular septal thickening (Fig. 1C, D), and areas of low attenuation, indicative of intrapulmonary lipid (Fig. 1E). The patient underwent bronchoscopy with



**Figure 1.** The chest radiograph, computed tomography (CT) images, and microscopic findings of bronchoalveolar lavage (BAL) fluid. (A) Chest CT acquired one year before admission reveals ground-glass opacities with interlobular septal thickening in the left lower lung. (B) A chest radiograph acquired at admission reveals infiltrative opacities in the bilateral lower lung fields. (C–E) The chest CT images acquired at admission revealed ground-glass opacities with interlobular septal thickening and consolidation in the right middle and both lower lobes, and small areas of fat attenuation (–50 to –90 Hounsfield units) within the consolidation in the left lower lobe (white arrows). (F, G) A chest radiograph and CT image acquired nine months later show an improvement in the previous pulmonary lesions. (H) Lipid-laden alveolar macrophages are observed on Oil Red O staining of a BAL fluid sample.

bronchoalveolar lavage (BAL) fluid sampling. The BAL fluid contained  $1.3 \times 10^3$  cells/ $\mu\text{L}$ , comprising 88% lymphocytes, 1% eosinophils, 3% neutrophils, and 8% alveolar macrophages. The BAL fluid was negative for all pathogens. Nevertheless, lipid-laden macrophages were detected in the BAL fluid on Oil Red O staining (Fig. 1H). To determine a clinical history congruent with an exogenous origin of the lipid, we reassessed her medical and occupational histories. This revealed that the patient was a dry-cleaning worker, who worked with hydrocarbon-based solvent sprayers to remove dirt stains from clothes before putting them in a dry-cleaning machine daily for several years. Thus, she was diagnosed with exogenous lipoid pneumonia caused by chronic organic solvent inhalation. The patient was administered prednisolone 20 mg per day for two weeks because her symptoms were mild. In addition, the patient was instructed to avoid further exposure to the substance. Her respiratory symptoms resolved within one month. In addition, chest radiography and CT performed nine months later showed nearly complete resolution of the disease (Fig. 1F, G).

## Discussion

The diagnosis of exogenous lipoid pneumonia is based on a history of exposure to oil, corresponding radiological features, and the detection of lipid-laden macrophages in the BAL fluid or lung tissue on fat staining, as well as the exclusion of other relevant diseases potentially

explaining the radiological abnormality [1]. The source of lipid exposure is variable, and might include food, oil-based medications, and occupational settings [2]. Overlooking a history of oil ingestion often makes the diagnosis of exogenous lipoid pneumonia difficult.

In our case, lipid exposure was revealed by reassessment of detailed personal information after a diagnosis of lipoid pneumonia was suspected. The patient and her husband owned a small-scale dry-cleaning shop. She was in charge of the pre-spotting process, which included removal of dirt stains from clothes using hydrocarbon solvent sprayers. While spraying the solvents, a build-up of oil mist can fill the confined workplace. Prolonged inhalation of the substance might go unnoticed because of its characteristics that make it less reactive in the airways [3]. Therefore, the patient might have been exposed to the substance causing lipoid pneumonia for a long duration. Given that early identification and withdrawal of occupational exposure is the basis of the treatment of occupational diseases, progressive lung inflammation and fibrosis could be avoided in this case.

Dry-cleaning workers were found to have a high risk of work-related respiratory diseases, such as asthma, and inhalational accidents attributed to cleaning agents, including organic solvents [4]. The use of various hydrocarbon and chlorinated hydrocarbon solvents as dry-cleaning agents has been known to cause many adverse health problems [5]. Although exogenous lipoid pneumonia is a specific form of pulmonary toxicity associated with hydrocarbon solvents [1],

to the best of our knowledge, no previous report has documented organic solvent-induced lipoid pneumonia in a dry-cleaning worker. Aerosolization of oil due to spraying may contribute to the development of lipoid pneumonia through the inhalation of the aerosolized substances, as seen in this case.

This report of a rare case of lipoid pneumonia associated with occupational exposure to solvent aerosols in a dry-cleaning worker highlights the importance of careful consideration of a patient's occupational history during diagnosis. We also recommend that dry-cleaning workers be considered a population at risk of lipoid pneumonia.

### Disclosure Statement

Appropriate written informed consent was obtained for the publication of this case report and accompanying images.

### Author Contribution Statement

Sunji Park contributed to the concept and drafting. Ji Eun Park contributed to reviewing and revision. Jaehee Lee

contributed to the concept, drafting, reviewing, and revision. All authors critically reviewed the manuscript and approved the final version.

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