



Acute exogenous lipid pneumonia: Unusual presentation as cavitating lung disease with pneumothorax

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ABSTRACT

Acute lipid pneumonia is quite uncommon and is associated with oily or lipid contents within the alveoli. Exogenous lipid pneumonia due to kerosene poisoning, manifests with a wide clinical spectrum ranging from subtle chemical pneumonitis to marked severe pulmonary and systemic inflammation. We present an interesting case of an adult male with kerosene poisoning. He manifested with severe cavitating lung disease. In addition, he developed spontaneous pneumothorax. Both cavitating lung disease and pneumothorax are unusual manifestations of acute exogenous lipid pneumonia and perhaps follow severe lung injury following high volume kerosene exposure.

1. Introduction

Acute exogenous lipid pneumonia is uncommon and is caused by ingestion and aspiration of a large quantity of a petroleum or lipid-based product. Inflammatory reaction in the lungs and resultant toxicity depends on the route and degree of exposure, and volatility, and viscosity of the product [1]. While ingestion or aspiration of kerosene, a petroleum distillate, is a common cause of accidental poisoning in children, is rarely seen in adults. This leads to lung damage ranging from chemical pneumonitis to marked severe pulmonary and systemic inflammation. There is no consensus regarding the treatment of lipid pneumonia at present; Management strategies are primarily supportive and conservative. The role of systemic corticosteroids remains controversial [2]. Lung injury when severe, can lead to grave complications like hypoxia, pneumothorax, pneumomediastinum, and emphysema [3]. Imaging findings in the absence of an appropriate clinical setting, can mimic many diseases. Acute exogenous lipid pneumonia typically manifests clinically as cough, dyspnoea, and low-grade fever that usually resolves with supportive therapy. Radiological findings may develop within 30 minutes to 24 hours of exposure, and consists of ground-glass opacities, crazy paving pattern, areas of consolidation, and irregular nodules [4]. We present a case of acute lipid pneumonia which had unusual radiological features in the form of cavitating lung disease, with abscess cavities and pneumatoceles and pneumothorax due to spontaneous rupture of a pneumatocele.

1.1. Case presentation

A 62 years gentleman patient presented to emergency services of our tertiary care hospital, with a 2-week history of fever and

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cough with expectoration without hemoptysis. He also had generalized weakness and shortness of breath for the last 2 weeks, worsening over the last 48 hours. Additional history of hypothyroidism and uncontrolled type 2 diabetes mellitus was noted. His blood sugar was 585mg/dL and his HbA1c was 10.4%. Urine dipstick test showed small amounts of ketone and protein with a large amount of glucose. The patient was started on intravenous insulin and fluid. The patient was also a chronic smoker. Oxygen saturation was 93% on room air. On examination, his breath sounds were decreased with inspiratory rales and crackles bilaterally. Working diagnosis at admission was diabetic ketoacidosis with pneumosepsis. Routine blood investigations revealed leucocytosis with a white blood count

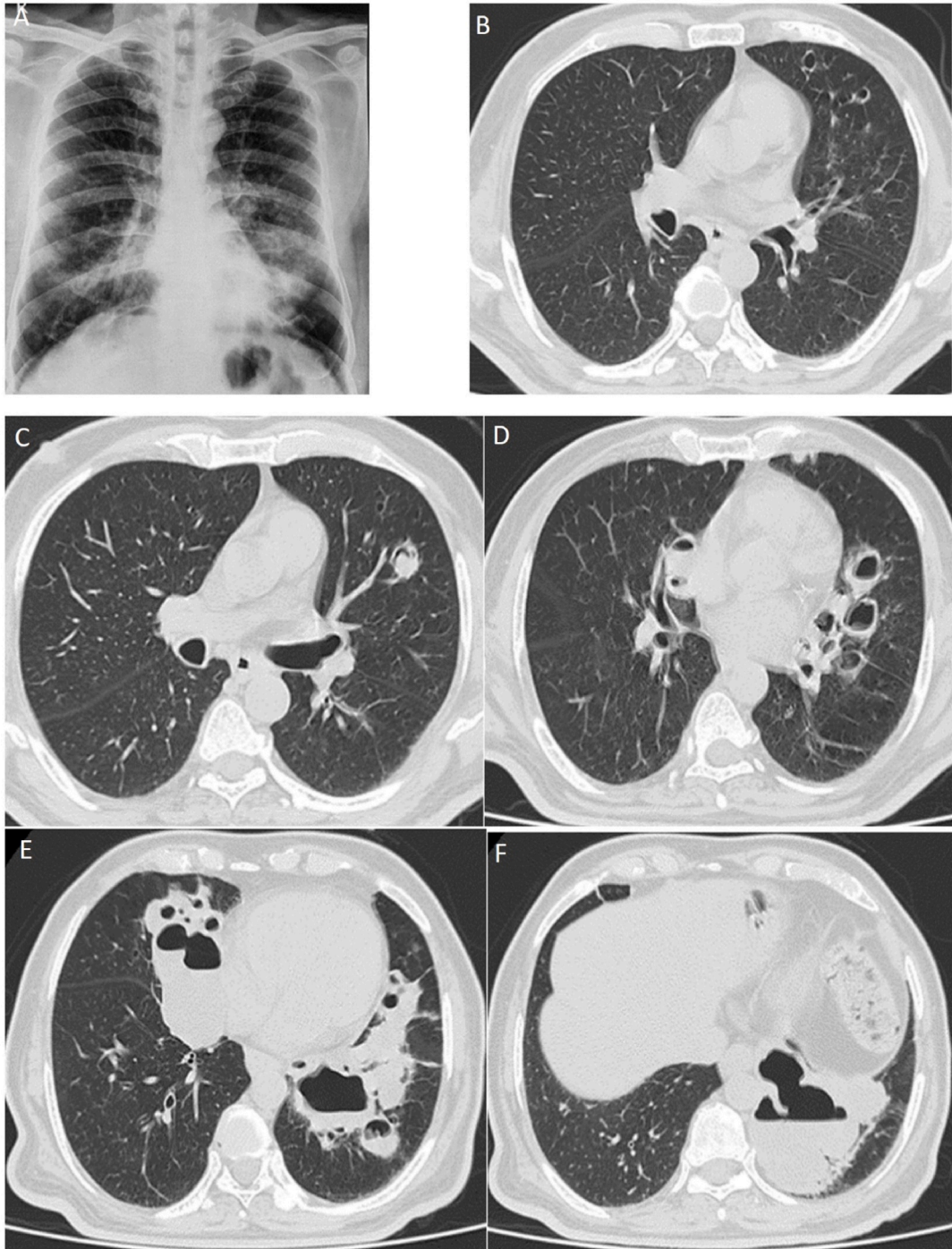


Fig. 1. Chest Xray (A): showing patchy infiltrates in bilateral lower zones. HRCT lung revealing (B): air-filled pneumatoceles and (C): cavitating nodule in left upper lobe (D&E): bilateral consolidation and pneumatoceles with air-fluid levels and (F): abscess cavity in the left lung.

of 12500/cumm and predominant neutrophils (90%). Chest X-ray done at admission showed patchy infiltrates in bilateral lower zones obscuring the cardiac outline partially (Fig. 1A). HRCT Chest was also done which showed bilateral dependent dense consolidation and multiple pneumatoceles, some of them showing air-fluid levels. In addition, multiple abscess cavities were present in the left lower lobe, and a cavitating nodule was seen in the left upper lobe (Fig. 1B–E). A guarded prognosis was given.

On being pointedly probed at this juncture, the patient reluctantly gave a history of kerosene ingestion 20 days back, after which he developed shortness of breath and was admitted to a local hospital. CT scan of the thorax at that time had shown ill-defined areas of ground-glass opacities with superimposed interlobular septal thickening (crazy-paving pattern) in both lungs, with a predominant lower lobar distribution (Fig. 2A and B). In addition, the mediastinal window showed the presence of low attenuating (lipid-containing) opacities (Fig. 2C). Following a course of antibiotics, the patient had improved and was discharged. He was fine for the next 2 weeks but, subsequently became febrile and developed cough and shortness of breath for which he was brought to our hospital. CT scan of the Chest was done at our hospital showed significant worsening of the imaging findings as compared to the earlier CT.

While undergoing treatment, the patient developed a left-sided pneumothorax two days after admission (Fig. 3A), likely due to spontaneous rupture of one of the cavitating lesions/pneumatocele. He underwent drainage of pneumothorax using a large-bore intercostal thoracic drain (Fig. 3B). He also underwent a pigtail catheter drainage of the largest cavitating lesion in the lower lobe of the left lung (Fig. 3C), which revealed thick, foul-smelling pus with plenty of air bubbles. The aspirate was sent for microbiological analysis. Sample from the wall of the cavity was also sent for lipid-laden macrophages. Bacterial, fungal, and mycobacterium cultures on the aspirated sample were negative. Analysis of aspirate from cavity wall revealed lipid-laden macrophages on wet mount, and a diagnosis of lipoid pneumonia was established. Intravenous antibiotics and systemic corticosteroids were administered during the 2-week course of treatment at our hospital. The large bore drain was removed once pneumothorax was resolved. Subsequent chest X-rays

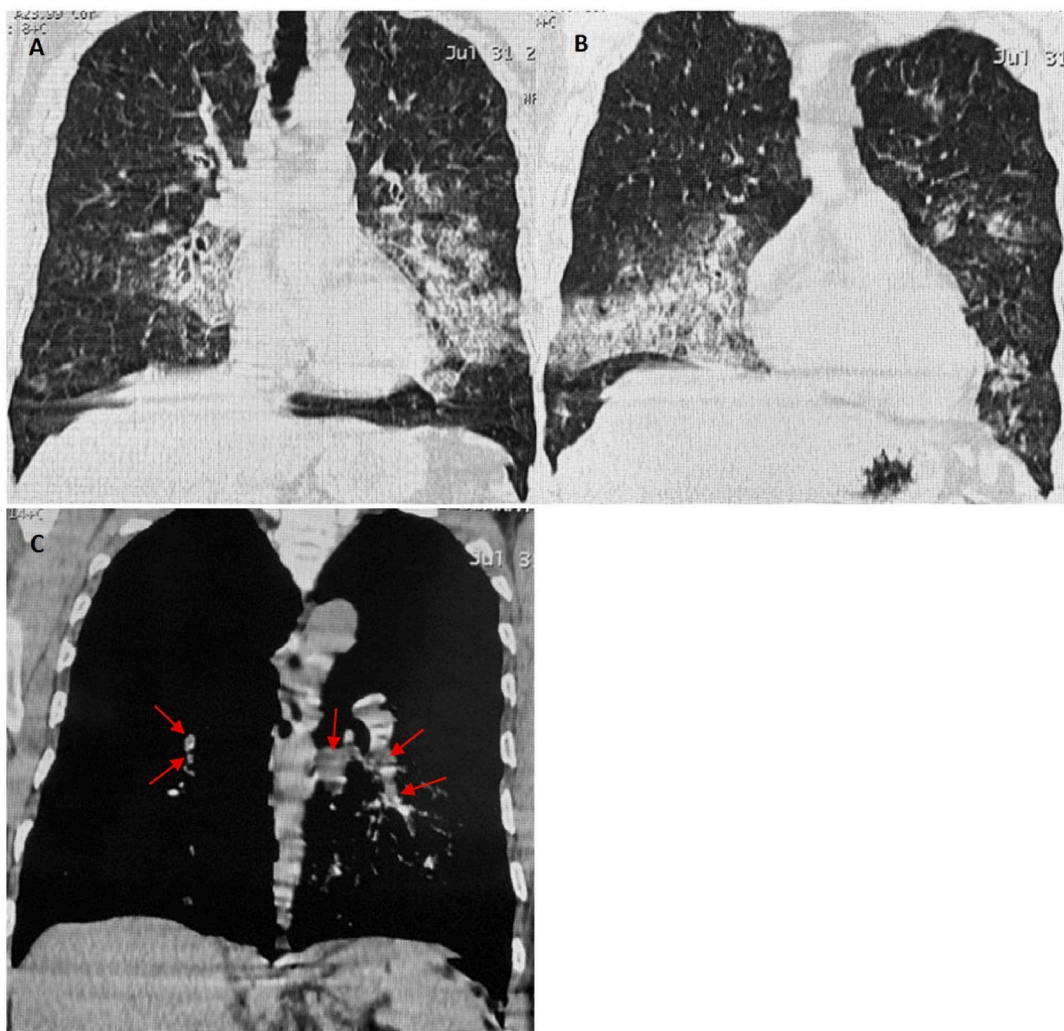


Fig. 2. CT Chest. Lung windows (A&B) reveal bilateral, large confluent areas of ground-glass opacities with superimposed interlobular septal thickening, giving a crazy-paving pattern with posterior predominance in the right middle and left lower lobes. (C): low attenuation foci on the mediastinal window, in involved areas in the lung (arrows).

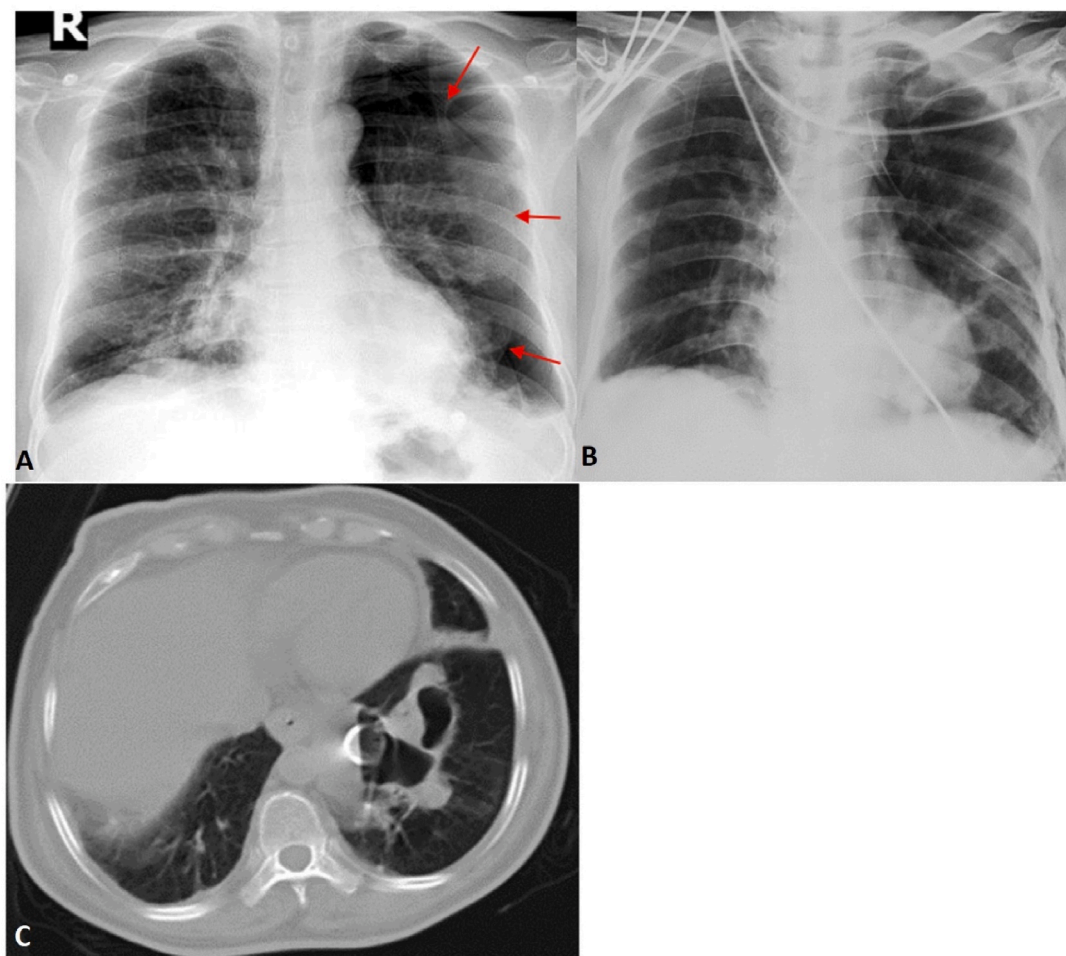


Fig. 3. (A) Chest X-ray done 2 days later, shows pneumothorax (arrows). (B): Large bore thoracic drain seen in situ with resolved pneumothorax, and decrease in lung opacities. (C): Chest CT showing pigtail catheter in situ in left lower lobe abscess cavity.

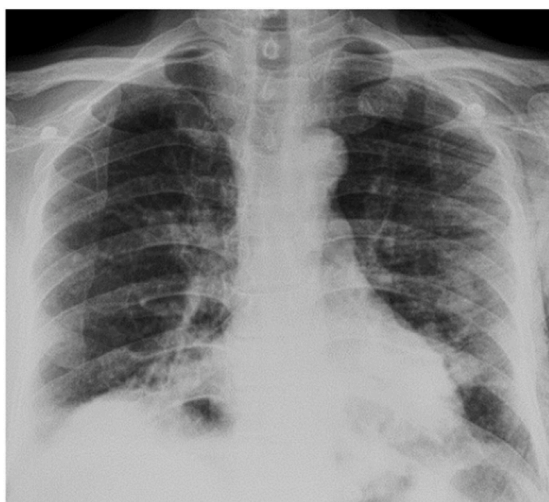


Fig. 4. Chest Xray, done after removal of chest drain.

showed resolution of the pneumothorax and a significant decrease in lung infiltrate shadows (Fig. 4). The patient became afebrile and asymptomatic, and he was discharged on oral antibiotics. Follow-up chest X-ray done at an outside hospital was normal with no abnormal infiltrates.

2. Discussion

Diagnosis of lipid pneumonia requires a high index of suspicion and can be confirmed by demonstration of lipid-laden macrophages in respiratory samples such as sputum, bronchoalveolar lavage fluid, or fine-needle aspiration cytology/biopsy from lung lesions, as in our patient [5,6]. Accidental poisoning by kerosene, a petroleum-based product, is usually seen in children below 5 years [7]. Suicidal poisoning is also seen, due to the easy availability of fuel. Due to the low viscosity and high volatility of liquid kerosene, after aspiration, it diffuses rapidly throughout the bronchial mucosa, causing pulmonary interstitial and alveolar inflammation. This aspirated lipid material then either induces a granulomatous reaction around it and gets surrounded by connective tissue, fibroblasts, and lymphocytes, forming nodules or mass termed paraffinomas or are phagocytosed by macrophages which migrate to the interlobular septae being seen as foamy lipid-laden macrophages [2,3].

While some patients aspirate with no sequelae, most cases of aspiration cause minor symptoms or pneumonitis. In a few patients, however, pulmonary toxicity may be severe and cause major morbidity and mortality. In these cases, aspiration is followed by inflammation and hemorrhagic exudative alveolitis with resultant surfactant loss and alveolar collapse producing exogenous lipid pneumonia.

Radiologically, a wide spectrum of findings may be seen, with the earliest signs appearing within minutes to hours, and correlating with the intensity and duration of exposure to the toxic substance. These findings more commonly include pulmonary ground-glass opacities, crazy paving pattern due to alveolitis with interlobular septal thickening, consolidative foci, and/or pulmonary nodules which may resolve in 2 weeks to a few months [8,9]. The distribution of lesions is usually bilateral, maybe segmental or lobar, most commonly involving the middle and lower lobes [10]. Initial CT in our patient done within 48 hours of exposure to kerosene, showed the typical findings of bilateral crazy paving pattern, involving the middle and lower lobes. The presence of low/fat attenuating areas within the consolidative opacities and nodules, as seen in our patient, is highly suggestive of the possibility of lipid pneumonia.

CT done in our patient 2 weeks, showed dense areas of consolidation replacing the low attenuation areas. The presence of superimposed inflammation/infection can increase the attenuation of the infiltrates and can obscure the fat component [10–12]. In addition, multiple discrete to coalescing nodules, cavitating lung lesions with air-fluid levels indicative of abscesses and pneumatoceles had also formed, which are uncommon findings, and are a feature of severe toxic exposure and fulminant disease probably resulting from alveolar, bronchial, and/or vascular necrosis [13–16]. Spontaneous pneumothorax, as seen in our patient, is only rarely seen as part of the disease spectrum. This could occur due to persistently elevated transpulmonary pressure, facilitating interstitial emphysema and dissection of air towards visceral pleura, or due to the formation of bronchopleural fistula. In a large study on 274 cases with hydrocarbon poisonings, reported by Lifshitz et al., 118 (43%) patients had bilateral interstitial pneumonia, presenting with hypoxia and fever. Cavitating lung lesions, pneumatoceles, and pneumothorax as seen in our case were not seen in any of them [13]. To the best of our knowledge, kerosene toxicity presenting as cavitating lung disease with the presence of lung abscesses with air-fluid levels, further progressing on to spontaneous pneumothorax, as seen in our case, has not yet been reported.

3. Conclusion

Lipid pneumonia is a rare, often underdiagnosed disease, as it mimics many other diseases, due to its protean clinical and radiological manifestations. The disease should be suspected based on a careful history, thorough assessment of the clinical and radiological features, and should be confirmed through a multidisciplinary approach. The presence of fat attenuating (lipid-containing) opacities on CT scan, if present, may be helpful. Demonstration of lipid-laden macrophages in the involved area helps to establish the diagnosis. Our patient, who progressed to a severe cavitating lung disease with spontaneous pneumothorax, represents an unusual, more dramatic form of the disease spectrum of acute exogenous lipid pneumonia.

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None.

Author Contributions

Usha Goenka: Conceptualization, Methodology, Resources, Writing- Original draft and review and Supervision; **Surabhi Jajodia:** Conceptualization, Formal analysis, Visualization, Writing- Original draft, review and editing; **Debraj Jash:** Resources and patient management; **Somali Ghosh:** Writing- review and editing, Visualization; **Syamasis Bandyopadhyay:** Resources and patient management

Patient consent statement

We have obtained informed consent from the patient to publish case details and radiological images pertaining to the case while ensuring anonymity of all identifying information.

Declaration of competing interest

None.

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None.

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