Case Series

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The management of exogenous lipoid pneumonia: A case series

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Abstract:

Exogenous lipoid pneumonia is a rare but serious respiratory condition caused by inhalation or aspiration of lipid-based substances. It often presents with nonspecific symptoms, such as cough, dyspnea, and fever, which can mimic infectious pneumonia and lead to diagnostic delays. Radiological imaging typically reveals ground-glass opacities, consolidations, or nodular lesions, particularly in the lower and middle lobes. Diagnosis is supported by a clinical history of exposure to specific oils or solvents, and confirmation often requires bronchoalveolar lavage demonstrating lipid-laden macrophages. Management includes cessation of exposure, supportive care, corticosteroids in selected cases, and treatment of secondary infections. This case series highlights three distinct clinical presentations of exogenous lipoid pneumonia—two involving fire-breathers and one related to occupational solvent exposure—emphasizing the importance of early recognition and tailored therapeutic strategies to prevent long-term complications.

Keywords:

Aspiration, fat-laden fluids, lipoid pneumonia

Introduction

Lipoid pneumonia is an uncommon condition resulting from the accumulation of lipids in the alveoli. Lipoid pneumonia is defined as a form of pneumonia resulting from the unintentional inhalation or aspiration of a lipid-containing fluid.[1] The lipids in the respiratory system are categorized into two main categories based on their origin: exogenous lipoid pneumonia, derived from an external source, and endogenous lipoid pneumonia, originating from an internal or idiopathic source.[2,3] Exogenous lipoid pneumonia results from aspiration of fat-laden fluids or particles, whether deliberate or accidental.

Exogenous lipoid pneumonia results from the aspiration or inhalation of lipid-containing substances, most commonly mineral oils, animal fats, or plant-based oils.[4,5] Traditional risk factors include the use of oil-based nasal drops, laxatives, occupational exposure to oily aerosols, and aspiration related to swallowing dysfunction

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or neurological disease.[2,5,6] Recently, e-cigarette or vaping product use–associated lung injury (EVALI) has emerged as a novel and clinically relevant cause of exogenous lipoid pneumonia.[7,8] Propylene glycol and vegetable-sourced glycerine, particularly vitamin E acetate used as a diluent in vaping products, have been implicated in triggering an intense inflammatory response and lipid accumulation within alveolar macrophages.[7,8] This emerging etiology has broadened the clinical spectrum of exogenous lipoid pneumonia and has highlighted the importance of a detailed exposure history in affected patients.

Exogenous lipoid pneumonia is an uncommon pulmonary condition resulting from the aspiration or inhalation of lipid-containing materials. Its nonspecific clinical manifestations and imaging findings often resemble those of infectious or other inflammatory lung diseases, making the diagnosis particularly challenging. Prompt identification and proper management are essential to avoid complications such as fibrosis or acute respiratory distress syndrome. This case series presents three patients with exogenous lipoid pneumonia and outlines their clinical course, diagnostic evaluation, and treatment, emphasizing the crucial role of occupational exposure history in establishing the diagnosis.

Case 1

A 19-year-old male patient, employed as a fire-breather, presented to the emergency department with dyspnea, cough, and fever, which had emerged following multiple episodes of kerosene aspiration 2 weeks and 4 days prior. He had no known comorbidities and no history of tobacco use. Alcohol intake was 1–2 glasses per week. On admission to the emergency department, the pulse was 115 bpm with an oxygen saturation of 87%. Following administration of nasal oxygen at 3 L/min, oxygen saturation increased to 97%. The temperature was 38.3°C. Pulmonary auscultation revealed diminished breath sounds and coarse crackles at the right lung base. The laboratory findings exhibited the following abnormal values: white blood cell count: 23.69 ×10³/μL with neutrophilic predominance; C-reactive protein: 21 mg/L. The liver function tests were within normal parameters. Thoracic computed tomography (CT) revealed extensive consolidation and fluid-filled regions with intermittent air bronchograms in the right middle lobe [Fig. 1a]. The patient was brought to the chest diseases ward, where nasal oxygen was administered and antibiotic treatment with piperacillin-tazobactam 4.5 g (three times a day) and ciprofloxacin

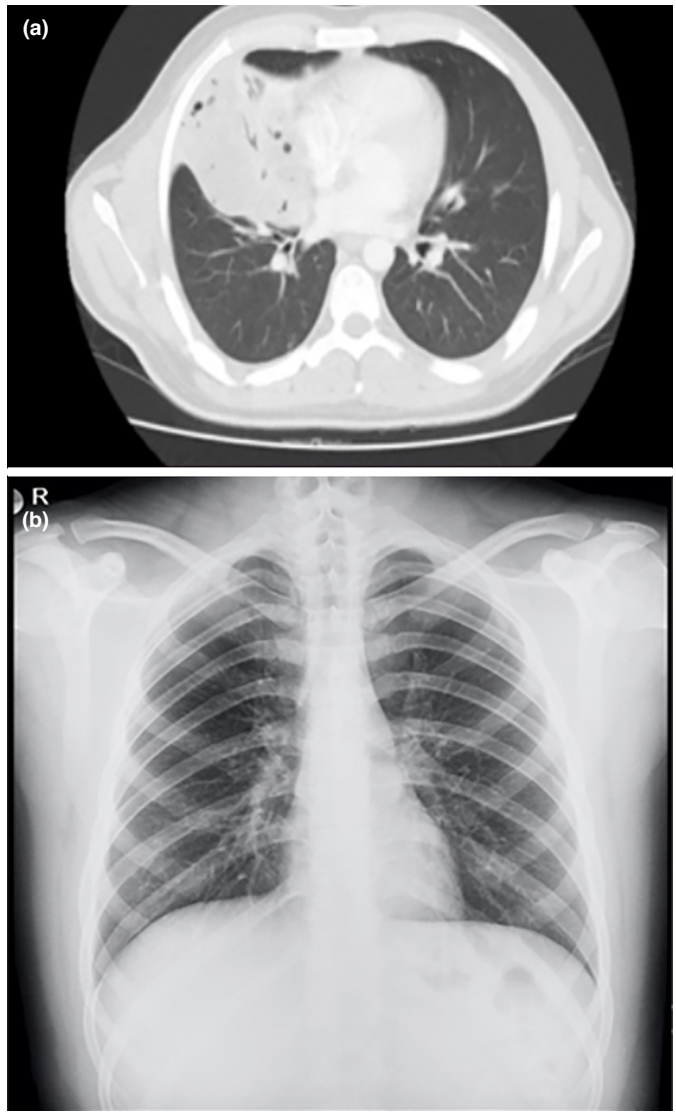

Figure 1: (a) CT shows consolidation in the right middle lobe. (b) Chest X-ray obtained after 1 year of follow-up revealed resolution of the consolidation

400 mg (two times a day) IV, together with supportive care, was commenced. A fiberoptic bronchoscopy (FOB) was scheduled. FOB revealed dense, dark-hued inflammatory mucoid secretions in the right main bronchus, which were aspirated, and bronchoalveolar lavage (BAL) was performed. No growth was observed in the bronchoalveolar lavage culture. Pathological examination demonstrated a pronounced inflammatory pattern and a significant presence of alveolar macrophages. Oil Red O staining revealed lipid-laden macrophages. On the tenth day of hospitalization, the patient, who exhibited improved symptoms and radiographic regression on chest X-ray (CXR), was discharged on outpatient antibiotic therapy. No sequelae were noted on the CXR performed during the initial year of follow-up [Fig. 1b].

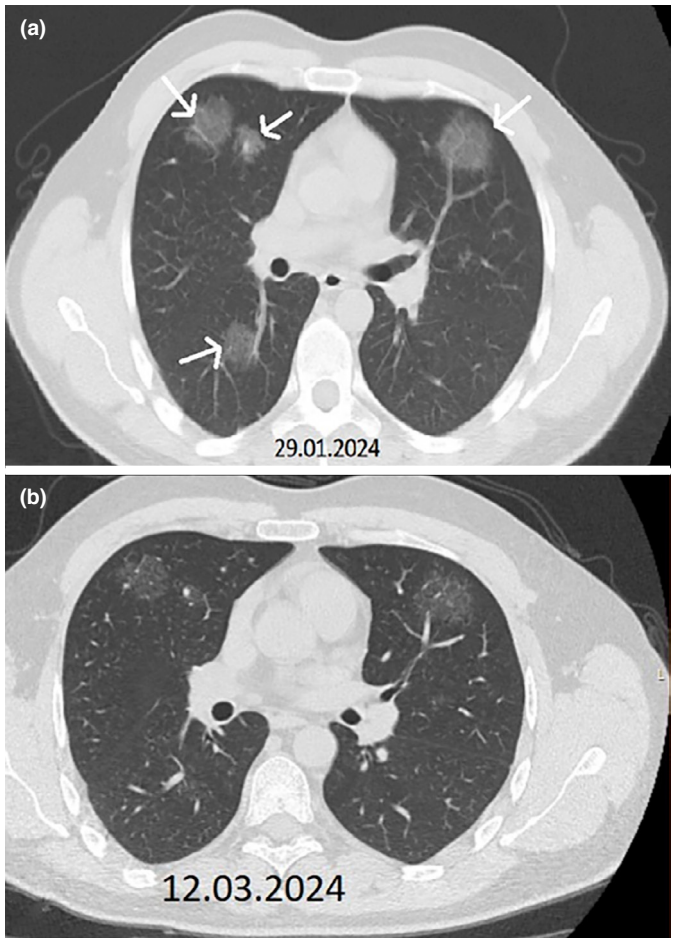

Figure 2: (a) CT scan shows asymmetric, scattered infiltrates with well-defined ground-glass opacities in both lungs. (b) Following cessation of exposure and medical treatment, resolution of the infiltrates was observed at 2 months

Case 2

A 31-year-old male patient was admitted to our outpatient clinic with complaints of cough and dizziness persisting for the past two months. No additional comorbidities were documented. The patient had a 10 pack-year smoking history. There was no record of alcohol consumption. No prior history or exposure to tuberculosis was noted. The patient's medical and family history revealed no significant findings. He was not taking any medication. It was discovered that, in his professional experience, he had engaged in laser cutting of metal and wood, as well as in painting with solvent-based epoxy and paint thinner, for six years. Pulmonary auscultation revealed bibasillar inspiratory crackles. Lung CT revealed nodular lesions and diffuse ground-glass opacities involving (suggestive of atypical pneumonia) both lungs [Fig. 2a]. Moxifloxacin 400 mg/day was prescribed for outpatient treatment for one week. One week later, the patient returned for a follow-up, although no clinical improvement was noted. The patient, presenting with dizziness, was admitted to the Department of Chest Diseases with a provisional diagnosis of lipoid pneumonia. FOB was offered, but the patient refused the procedure. Additionally, the presence of extensive ground-glass opacities prompted consideration of several alternative diagnoses, including e-cigarette or vaping product use–associated lung injury (EVALI), hypersensitivity pneumonitis, organizing pneumonia, and atypical or viral pneumonia. EVALI was considered unlikely due to the absence of any history of e-cigarette or vaping product use. Hypersensitivity pneumonitis was deemed less probable given the lack of relevant antigen exposure. Given the chronic occupational solvent exposure, systemic toxicity was also considered. The patient's fundoscopic examination and brain MRI were unremarkable. Consequently, ophthalmology and neurology consultations did not establish a diagnosis. The patient's radiological findings prompted an investigation into the presence of connective tissue disorders. Results for the ANA profile, complement levels, HLA B27, CCP, RF, anti-SS-A, anti-SS-B, anti-Jo-1, and anti-DS-DNA were all within normal limits. The rheumatology consultation concluded that the patient did not have an active rheumatological condition. Infectious etiologies were excluded based on negative microbiological studies and lack of clinical response to antimicrobial therapy. Diffusing capacity for carbon monoxide (DLCO) was measured. DLCO/VA was 71%. Pulmonary function tests (PFTs) showed an FEV_1 of 75%, an FVC of 76%, and an FEV_1/FVC ratio of 96%. Based on these findings and the clinical history, lipoid pneumonia associated with chronic solvent exposure was considered the most likely diagnosis. The patient received methylprednisolone at a dosage of 16 mg per day for one month. He was provided with a protective mask appropriate for his profession. A notable beneficial response in PFT and DLCO was recorded at the one-month follow-up. Pulmonary function testing showed a DLCO/VA of 82%, FEV_1 of 87%, FVC of 92%, and an FEV_1/FVC ratio of 94%, findings suggestive of a mild restrictive ventilatory defect. A notable improvement in pulmonary function test results was observed prior to therapy. The complaint of dizziness has been recorded as resolved. A control non-contrast thoracic CT performed during the first month of treatment revealed a notable resolution in ground-glass opacities, which were diffusely distributed across all lobes of both lungs [Fig. 2b]. The patient's symptoms of dizziness and cough have subsided, and he is currently under surveillance and therapy.

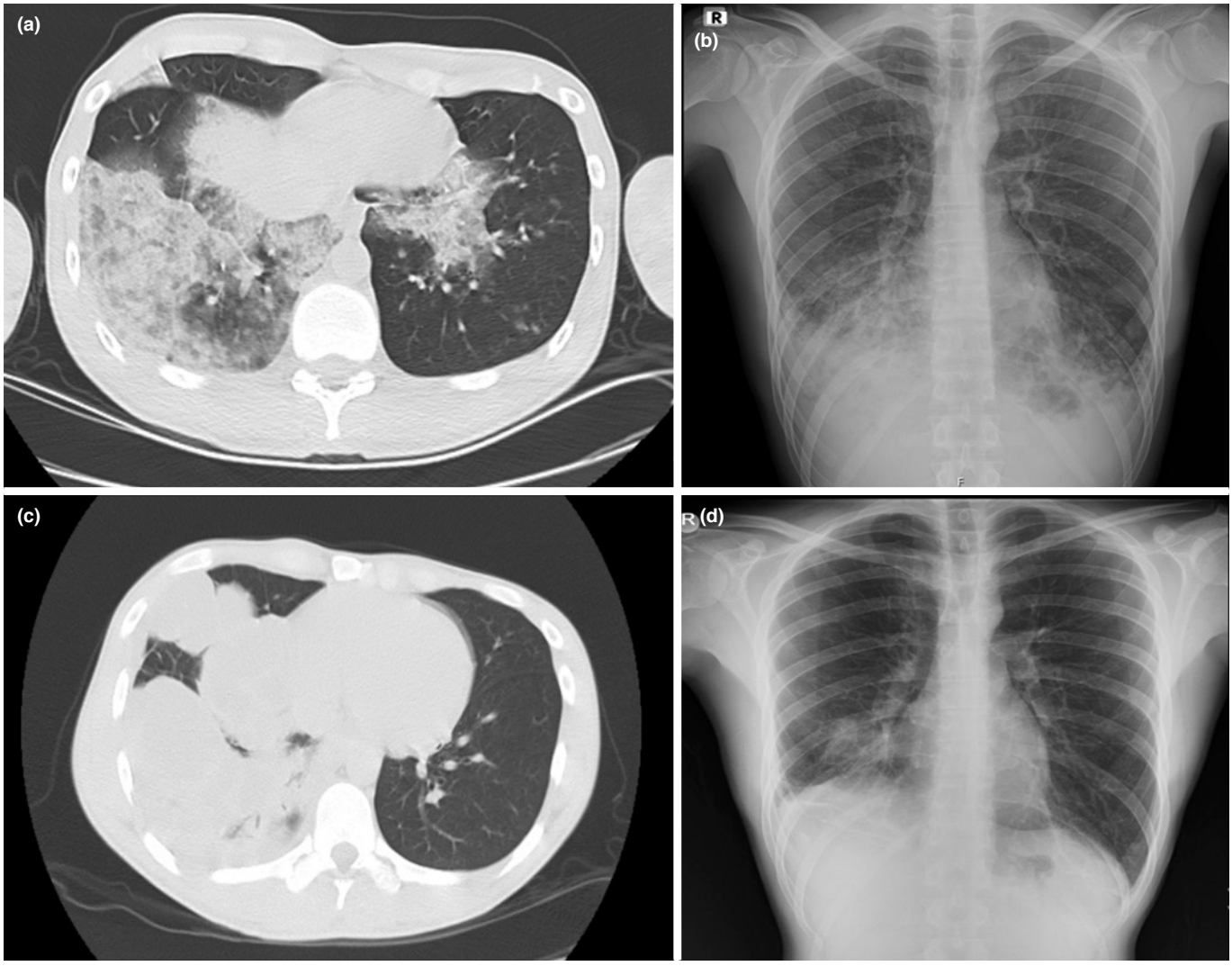

Figure 3: (a) CT scan shows extensive ground-glass opacities and consolidations in the lower lobes of both lungs. (b) Chest X-ray reveals bilateral lower lobe opacities, with denser consolidation more prominent on the right side. (c) On CT scan, pleural-based, well-defined, loculated collections are observed, consistent with homogeneous density increases compatible with pleural effusion; surrounding areas of consolidation are also noted. (d) Chest X-ray demonstrated significant resolution of the consolidated areas after treatment

Case 3

A 25-year-old male patient, employed as a fire breather, was admitted to the emergency department with complaints of dyspnea, cough, and fever that manifested 24 hours following the aspiration of approximately 75 ml of kerosene during an animation show for tourists. He had no recorded medical history; nonetheless, he was an active smoker with a smoking history of six pack-years. Upon admission to the emergency department, the Glasgow Coma Scale (GCS) score was 14; the pulse rate was 142 bpm; oxygen saturation on room air was approximately 85%, and with nasal oxygen at 6 L/min, the saturation was 92%. Physical examination revealed oropharyngeal hyperemia, reduced respiratory effort, and decreased breath sounds accompanied by coarse crackles at the bilateral lung bases. Laboratory findings were remarkable for leukocytosis (WBC $17.57\times10^3/\mu L$) with neutrophil predominance, an elevated CRP level of 180 mg/L, and a procalcitonin level of 2.52 μg/L. Thoracic CT revealed extensive ground-glass opacities and consolidations in the lower lobes of both lungs, as well as in the right middle lobe [Fig. 3a, b]. The patient was admitted to the pulmonology department and intravenous antibiotic therapy with piperacillin-tazobactam 4,5 g (three times a day) and ciprofloxacin 400 mg (two times a day) was commenced. During the patient's follow-up, the fever persisted. Following the detection of a dense right-sided pleural fluid collection on thoracic CT, the thoracic surgery department was consulted for drainage [Fig. 3c]. The antibiotic regimen was modified to meropenem (3 times 1 g) and ami-

kacin (2 times 1 g). The patient's overall condition shows partial improvement, and follow-up assessments are ongoing [Fig. 3d]. Both the clinical findings and radiological assessment, together with a history of acute kerosene aspiration, supported the diagnosis of lipoid pneumonia. Histopathological confirmation was not pursued, as the clinical evidence was considered sufficient.

Discussion

The diagnosis of lipoid pneumonia relies on radiological characteristics due to the inhalation or aspiration of fats and oily substances, the identification of lipid-laden macrophages in bronchoalveolar lavage fluid or lung tissue via Oil Red O or Sudan III, IV, or Sudan Black B staining, and the exclusion of other diseases that may account for the radiological anomaly.[5] Lipoid pneumonia was initially documented by Laughlen in 1925.[9] Lipoid pneumonia has been documented globally across all age demographics.[10–12] Lipids are characterized as endogenous, exogenous, or idiopathic based on their source. The exogenous type is more prevalent and is linked to the administration of mineral oil for constipation relief with laxatives, particularly observed in the elderly.[10]

Fire-breather's pneumonia represents a well-described but rare form of exogenous lipoid pneumonia resulting from accidental aspiration of hydrocarbon-based fuels during fire-eating performances.[5,13] Published case series typically involve young, otherwise healthy individuals presenting with acute respiratory symptoms shortly after exposure, accompanied by bilateral ground-glass opacities or consolidations on chest imaging, often with lower lobe predominance.[5] Consistent with these reports, both of our fire-breather cases developed symptoms rapidly after exposure and demonstrated imaging findings consistent with acute inflammatory lung injury. However, compared with previously published series, the clinical course in our patients was relatively mild, possibly reflecting limited exposure volume, early recognition of symptoms, and prompt cessation of further exposure. Prior studies have suggested that the amount of aspirated hydrocarbon and the interval between exposure and diagnosis are key determinants of clinical severity, supporting this interpretation.[5,14]

In contrast, the second case, related to chronic occupational exposure to solvents, represents a less frequently reported but clinically important cause of exogenous lipoid pneumonia. Unlike the acute presentation observed in fire-breather's pneumonia, this patient exhibited a more insidious onset, progressive respiratory symptoms, and more extensive radiological involvement. The novelty and relevance of this case lie in the prolonged low-dose inhalational exposure, which may lead to cumulative lipid deposition and sustained alveolar inflammation over time.[4,5] Occupational exposure–related lipoid pneumonia has been sporadically described in the literature, and such cases emphasize the importance of a detailed occupational history in patients with unexplained or slowly progressive pulmonary infiltrates.[2,5]

In addition, the prevalence of electronic cigarette usage has increased rapidly in recent years, particularly among younger populations.[15] A substantial proportion of patients with e-cigarette or vaping product use–associated lung injury(EVALI)reported by the Centers for Disease Control and Prevention (CDC)in 2019 demonstrated clinical, radiological, or pathological features consistent with lipoid pneumonia.[8,16] Accordingly, EVALI has been recognized as an emerging cause of exogenous lipoid pneumonia, as lipid-containing substances—most notably vitamin E acetate used as a diluent in vaping products—are introduced into the lungs via inhalation, leading to lipid accumulation and inflammatory lung injury.[7,8,16]

Although none of our patients reported e-cigarette use, the recent emergence of EVALI has further expanded the etiological spectrum of exogenous lipoid pneumonia. EVALI represents a contemporary form of inhalational lipid-mediated lung injury, most commonly linked to vitamin E acetate, and reinforces the need to consider both traditional and modern sources of exogenous lipids in the differential diagnosis of compatible clinical and radiological presentations.[7,8]

Conversely, the endogenous type is secondary to bronchial constriction or to pulmonary fat embolism, alveolar proteinosis, and lipid accumulation disorders. The idiopathic variant is an uncommon disease observed in smokers.[17] Additional risk factors encompass neuromuscular problems, structural anomalies of the nasopharynx that increase the risk of aspiration, and increased occupational hazards among "fire breathers" who perform in circus or other entertainment acts. Two of our cases involved performers who practiced fire-breathing, while one patient presented with exogenous lipoid pneumonia resulting from exposure to paint thinner during painting work. Mineral oil is the material most frequently associated with exogenous lipoid

pneumonia; it is not degraded by pulmonary enzymes but is phagocytosed by alveolar macrophages. Lipid-laden alveolar macrophages induce a granulomatous response. Chronic inflammation may result in progressive lung fibrosis.[4,6] Common signs of exogenous lipoid pneumonia include fever, weight loss, cough, dyspnea, chest discomfort, and hemoptysis. Approximately 40% of lipoid pneumonia patients have mild symptoms or are asymptomatic, complicating the diagnosis and resulting in frequent incidental findings.[18] Conversely, substantial exposure to the detrimental substance may result in severe hypoxemic respiratory failure, potentially advancing to acute respiratory distress syndrome (ARDS).[2] The predominant symptoms seen in our cases were dyspnea, cough, and fever. In our patients, symptoms and clinical status have been observed to vary from mild to severe.

In lipoid pneumonia, the initial radiological abnormalities have been documented to manifest within minutes to hours. The intensity and duration of exposure to the harmful agent are associated with a diverse array of findings. These findings typically encompass pulmonary ground-glass opacities, a crazy paving pattern linked to alveolitis and interlobular septal thickening, consolidative foci, and/or lung nodules that may disappear within 2 weeks to many months.[11,19] The lesions are often distributed bilaterally, may be segmental or lobar, and predominantly affect the middle and lower lobes.[19] In our cases, as in the literature, the most commonly observed findings were ground-glass opacities and consolidation. Moreover, uncommon observations have also emerged, including severe toxic exposure potentially resulting from alveolar, bronchial, and/or vascular necrosis; multiple discrete and confluent nodules; abscesses; and air-fluid levels indicative of pneumatoceles associated with cavitating lung lesions, all indicative of fulminant disease.

The primary treatment for lipoid pneumonia involves eliminating the causative agent, ensuring sufficient oxygen support with antimicrobial therapy for secondary bacterial infections, administering corticosteroids to suppress the inflammatory response in the lungs, and implementing additional supportive measures such as chest physiotherapy to aid in the drainage of tracheobronchial secretions.[20] Moreover, it has been documented that the mechanical extraction of lipid constituents using bronchoscopy via bronchoalveolar lavage is advantageous in severe instances of lipoid pneumonia.[21] In one of our cases, tube drainage and abscess evacuation were performed to mitigate exposure and inflammation resulting from pneumonia and abscess that were unresponsive to antibiotic therapy and from pneumothorax.

Conclusion

Exogenous lipoid pneumonia is a serious clinical condition that can arise from various causes and present with different and variable radiological appearances depending on the cause. In patients presenting with atypical infiltrates on chest X-rays or CT scans, situations such as occupational exposures, inhalation of foreign or different substances either intentionally or accidentally, and the use of electronic cigarettes should definitely be inquired about in the patients' medical history. Radiological appearances can present in various forms, ranging from ground-glass infiltrates to consolidation and even ARDS. In patients, acute respiratory failure, cough, and fever may often be prominent. Bronchoalveolar lavage is helpful in diagnosis. Removal of the causative agent and treatments with steroids, antibiotics, bronchodilators, and an approach to acute respiratory failure are the first treatment interventions to consider.

Ethics Committee Approval
This is a case series, and therefore ethics committee approval was not required in accordance with institutional policies.

Informed Consent
All patients gave their written, signed informed consent, which is retained in their medical records, on admission to the hospital for the use of their medical information in scientific studies as long as their names remain anonymous.

Conflict of Interest
The authors have no conflicts of interest to declare.

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