



E-ISSN: 2706-9575
P-ISSN: 2706-9567
IJARM 2022; 4(1): 215-217
Received: 03-03-2022
Accepted: 05-04-2022

Samer Talib
MD, Department of Internal
Medicine, Raritan Bay Medical
Centre, Perth Amboy, New
Jersey, USA

Ali Nadhim
MD, Department of
Pulmonary and Critical Care,
Jersey shore medical Centre,
Neptune, New Jersey, USA

Corresponding Author:
Samer Talib
MD, Internal Medicine
department at Raritan Bay
Medical Centre, Perth Amboy,
New Jersey, USA

A clinical approach to diagnosis and treatment of lipid pneumonia

Samer Talib and Ali Nadhim

DOI: <https://doi.org/10.22271/27069567.2022.v4.i1c.379>

Abstract

Lipid pneumonia is a rare form of pneumonia caused by the accumulation of exogenous or endogenous lipid in the alveoli and host reaction to inhaled lipid particles; symptoms vary among patients from asymptomatic to acute respiratory distress. The diagnosis depends on the history of exposure to oil, characteristic imaging findings on CT scan of the chest (negative attenuation values within areas of consolidation), and lipid-laden macrophages in sputum or BAL. There is no standard treatment option for lipid pneumonia; the best course of action is to identify the offending agent and discontinue exposure. Treating asymptomatic patients remains controversial, while treatment options for patients with severe disease may include (systemic corticosteroids surgical interventions).

Keywords: Exogenous lipid pneumonia, HRCT, chest x-ray, BAL, lipid-laden macrophage, FNA

Introduction

Exogenous Lipid pneumonia results from the pulmonary accumulation of fat-like compounds of animal, vegetable, or mineral origin. Exogenous LP is divided into acute and chronic. Acute LP results from a massive quantity of oil aspiration quickly, and slow recurrent long-term oil inhalation leads to chronic LP. Exogenous LP is most commonly caused by inhalation of oil in food or oil-based medications used for constipation, such as (olive oil, cod liver oil, and paraffin oil) ^[1]. It's considered a disease in children and the elderly. Other factors may contribute, such as anatomical abnormality of the pharynx and esophagus (Zenker diverticulum, GERD, achalasia, cleft palate), critically ill patients receiving tracheostomy care, and now vaping is a significant cause ^[2]. However, there are no apparent predisposing factors in many cases, and the incautious use of oil-based products is highly associated with Lipid pneumonia. The aspiration of oily substances usually occurs unnoticed because they don't elicit a cough reflex ^[3]. Patients with Chagas disease have a higher chance of developing LP due to prolonged constipation caused by a functional disorder of the colon (megacolon), as well as a functional disorder of the esophagus (megaesophagus), both increase their risk of aspiration and eventually LP ^[4]. ELP pneumonia lesions are pathologically characterized by chronic inflammation, alveolar and interstitial fibrosis with the presence of giant cell granuloma. Recent lesion biopsy may show normal alveolar wall and septa, but lipid-laden macrophage filling alveolar cavity advanced lesion will show inflammatory infiltrate of alveolar walls and septa with bronchial walls and large vacuoles containing lipid. The older lesions will show parenchymal destruction with fibrosis surrounding lipid-containing vacuoles ^[5].

Case presentation

Seventy-eight-year-old female with a history of GERD, Irritable bowel syndrome, and a long history of intermittent dry cough, the patient was told that this is probably from allergy. The patient denied hemoptysis, shortness of breath, and chest pain, and the patient was referred to her primary care provider after an abnormal CT scan of the chest. The patient was told that she had fluid in her lungs. The patient had a remote history of pneumonia and was treated by her primary care provider as an outpatient; a CT scan of the chest showed bilateral infiltrate, symptoms improved, and the patient did not follow up after that. The patient had a history of mineral oil ingestion for many years due to constipation, vitals (temperature 96.8, RR 14, Spo2 97%, HR 80, BP 128/80), and physical exam was unremarkable. The patient blood work was (WBC 7000, Hb 14.7).

The patient's CT chest showed mass-like consolidation in the left upper lobe and right middle lobe, and both lower lobes. A bronchoscopy was done for non-resolving lower and upper lobes infiltrate, and the bronchoscopy results were as below:

- Vocal cords normal
- Subglottic space à normal.
- Trachea caliber is normal.
- Carina is sharp.
- Tracheobronchial tree is normal.
- Bronchial anatomy and mucosa look normal. No endobronchial lesions with no secretions.
- Bronchoalveolar lavage was performed in the left upper lobe and the left lower lobe of the lung and sent for cell count, bacterial culture, viral smear, and culture, fungal with AFB analysis, and cytology (oil red o stain). Twenty mL of fluid was instilled, and 40 mL were returned. The return was clear. There were no mucoid plugs in the return fluid. Multiple specimens were collected, pooled into one sample, and analyzed.

This patient's bronchoalveolar lavage was positive only for a few lipid-laden macrophages (2- 4/hpf) noted in Oil Red O-stained preparation raising the possibility of lipid pneumonia. This warranted transferring the patient to another center for total pulmonary lavage.

Clinical presentation

Exogenous lipid pneumonia symptoms are subtle. Patients may present with chronic cough with or without shortness of breath, hemoptysis, fever, and chest pain [6]. A physical exam is usually normal, or there will be some dullness to percussion with wheezing or crackles on auscultation. Diagnosis is challenging because many patients have only mild symptoms or no symptoms. Also, a history of oil ingestion is often missed. Various symptoms such as (stomachache, vomiting, dysphagia, and vertigo) were reported in some case reports. LP is a problematic diagnosis because many patients have mild symptoms or are asymptomatic, and the diagnosis is often missed, especially in the absence of a clear history of fatty substance exposure. It should be on the differential diagnosis list for patients with chronic dry cough and dyspnea. Chronic hypoxia Laboratory studies could be normal or may show leukocytosis and increased erythrocyte sedimentation rate. Pulmonary function testing could be normal or show a restrictive pattern, while radiological findings could be unspecific and involve middle and lower lobes. MRI shows a high-intensity signal on T1-weighted images and decreased signal on T2-weighted images [7]. Chest X-rays are normal or may assimilate findings seen in other differential diagnoses such as (localized granuloma, acute and chronic pneumonia, ARDS, and carcinoma). The most described CT scan of the chest findings includes alveolar consolidations, ground-glass opacities with thickening of intralobular septa (crazy-paving pattern), or alveolar nodules. Diagnosis of LP depends on detecting fat-laden macrophages in the sputum, bronchoalveolar lavage (BAL), fine needle aspiration cytology (FNAC), or biopsy from the lung lesion [8]. BAL fluid may give clues to the diagnosis of LP; macroscopically, it shows turbid fluid with fat globules; the biochemical analysis of the collected fluid can confirm the diagnosis by using fat stain [9]. The presence of lipid-laden macrophages microscopically is consistent with the

diagnosis of LP with fine-needle aspiration cytology/biopsy from lung lesions confirms the diagnosis [10]. LP is associated with superinfection by *Nocardia* or non-tuberculous mycobacteria because lipid in the pulmonary tissue enhances the growth of these organisms and prevents phagocytosis by macrophages. Repetitive aspiration may lead to lung fibrosis with hypoxic respiratory failure leading eventually to corpeulmonale1 lung volume loss, and retraction of the lesion may result in emphysematous appearance. Hypercalcemia can result from increased calcitriol production by inflammatory cells. There is no definitive therapy but supportive management options (oxygen therapy, control risk factors, whole lung lavage). Corticosteroid use remains controversial and should be reserved for severe cases LP bronchiectasis should be treated aggressively with medical management. For refractory cases, surgical removal of the affected lung tissue should be considered. The prognosis depends on the volume and distribution of aspirated lipid. Still, patients with acute LP may experience clinical and radiographic improvement with treatment, and some lesions may heal with fibrosis, resulting in lung volume loss. If the patient discontinues exposure to lipidic substances, symptoms and radiographic abnormalities may resolve within months. Fatal cases have been reported.

Conclusion

LP is a rare under-diagnosed disease resulting from Lipid substance aspiration. Clinical presentation differs from asymptomatic to mild respiratory symptoms such as cough and dyspnea to severe shortness of breath with oxygen-dependent from chronic lung fibrosis. Diagnosis depends on exposure, imaging, and BAL cytologic finding, with FNA results of lung lesions. Treatment is mainly supportive with steroids, and the cornerstone in the management is to limit exposure to fatty substances.

References

1. Guo Maoqing, *et al.* Exogenous lipid pneumonia in old people caused by aspiration: Two case reports and literature review. *Respiratory medicine case reports* vol. 27 100850. 2019 April, 26. doi:10.1016/j.rmcr.2019.100850
2. Beck LR, Landsberg D. Lipoid Pneumonia. [Updated 2021 Jul 26]. In: Stat Pearls [Internet]. Treasure Island (FL): Stat Pearls Publishing; 2022 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK554577/>
3. Bandla HP, Davis SH, Hopkins NE. Lipoid pneumonia: a silent complication of mineral oil aspiration. *Pediatrics*. 1999;103(2):E19. doi:10.1542/peds.103.2.e19
4. Marchiori E, Zanetti G, Nobre LF, Takayassu TC, Irion KL. Lipoid pneumonia complicating megaesophagus secondary to Chagas disease: high-resolution computed tomography findings. *J Thorac Imaging*. 2010;25(2):179-182. doi:10.1097/RTI.0b013e3181ad883b
5. Simmons A, Rouf E, Whittle J. Not your typical pneumonia: a case of exogenous lipid pneumonia. *J Gen Intern Med*. 2007;22(11):1613-1616. doi:10.1007/s11606-007-0280-7
6. Kim HJ, Lee WH, Jeong N, *et al.* Lipoid pneumonitis in a patient with an accidental ingestion of kerosene successfully treated with bronchoscopic segmental

- lavage and systemic steroid: A case report. *Medicine (Baltimore)*. 2018;97(41):e12786.
doi:10.1097/MD.00000000000012786
7. Case Report of Lipoid Pneumonia: An Unusual Etiology of a Lung Cavity. 10.23937/2378-3516/1410076
 8. Hadda V, Khilnani GC, Bhalla AS, Mathur S. Lipoid pneumonia presenting as non-resolving community acquired pneumonia: a case report. *Cases J*. 2009;2:9332. Published 2009 Dec, 16. doi:10.1186/1757-1626-2-9332
 9. Lauque D, Dongay G, Levade T, Caratero C, Carles P. Bronchoalveolar lavage in liquid paraffin pneumonitis. *Chest*.
 10. Hadda V, Khilnani GC. Lipoid pneumonia: an overview. *Expert Rev Respir Med*. 2010;4(6):799-807. doi:10.1586/ers.10.74