LIPOID PNEUMONIA: AN UNCOMMON ENTITY

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ABSTRACT

Lipoid pneumonia is a rare form of pneumonia caused by inhalation or aspiration of fat-containing substances like petroleum jelly, mineral oils, certain laxatives, etc. It usually presents as an insidious onset, chronic respiratory illness simulating interstitial lung diseases. Rarely, it may present as an acute respiratory illness, especially when the exposure to fatty substance(s) is massive. Radiological findings are diverse and can mimic many other diseases including carcinoma, acute or chronic pneumonia, ARDS, or a localized granuloma. Pathologically it is a chronic foreign body reaction characterized by lipid-laden macrophages. Diagnosis of this disease is often missed as it is usually not considered in the differential diagnoses of community-acquired pneumonia; it requires a high degree of suspicion. In suspected cases, diagnosis may be confirmed by demonstrating the presence of lipid-laden macrophages in sputum, bronchoalveolar lavage fluid, or fine needle aspiration cytology/biopsy from the lung lesion. Treatment of this illness is poorly defined and constitutes supportive therapy, repeated bronchoalveolar lavage, and corticosteroids.

Key words: Lipid-laden macrophages, lipoid pneumonia, mineral oil aspiration

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INTRODUCTION

Lipoid pneumonia (LP) is a rare form of pneumonia caused by inhalation or aspiration of a fatty substance. It was first described in 1925 by Laughlin and later by others in the first half of the twentieth century.[1-4] Since then, there are many reports with different names like paraffinoma, cholesterol pneumonia, lipid granulomatosis, all denoting its association with the inhalation or ingestion of various substances like petroleum jelly, mineral oils, “nasal drops,” and even intravenous injection of olive oil.[5-13] Many of us are unfamiliar with this condition, a fact that may be responsible for the underdiagnosis of LP. This entity does not have characteristic clinical or radiological features, which makes its diagnosis difficult, unless it is already suspected. Therefore, awareness of this type of pneumonia is important. Making a diagnosis of LP requires a high degree of clinical suspicion. It should be considered in the differential diagnoses of many pulmonary
conditions, especially in cases with history of exposure to fatty substances. Importantly, if diagnosed in a timely manner, the progression may be halted or at least slowed by stopping the exposure to the offending agent. Once suspected, diagnosis can be established by demonstrating the presence of lipid-laden macrophages in various specimens such as sputum, bronchoalveolar lavage, and lung biopsy. The aim of this review is to increase awareness of this uncommon entity among physicians.

CLINICAL FEATURES

Lipoid pneumonia is an unusual cause of respiratory symptoms and has been reported in all age groups. Initial reports of LP predominantly included children, often with local anatomic defects like cleft palate, or debilitated adults, but several reports indicate that it can occur in healthy individuals as well.[8,14] There are reports of LP associated with aspirating or inhaling mineral oil, oil-based laxatives, lip balm, lip glow etc.[15,16] Siphoning of various mineral oils (like diesel) from containers is a common practice in India and may be an important risk factor for LP. Other risk factors described in infants and small children are the use of traditional folk remedies like the use of oily nasal drops, forceful feeding of animal fats such as “ghee” to establish regular bowel habits, or transnasal use to treat cough and cold.[17,18] Other common examples of risk factors for LP are the use of sesame oil to flush nasal secretions (in India) and to relieve small bowel obstruction due to *Ascaris lumbricoides* (in Brazil).[19,20] It has also been reported after bronchography due to the use of dye propylidone (now rarely performed) and nasogastric tube feeding.[21]

The usual presentation of LP is an insidious onset of dyspnea and/or cough, similar to those of other chronic lung diseases. Less commonly described clinical features include chest pain, hemoptysis, and intermittent fever which may be related to the inflammatory reaction to oil, or which may be secondary to infection.[22] Systemic symptoms are uncommon unless chronic hypoxia precipitates weight loss due to chronically increased work of breathing. Physical examination findings may be unremarkable or there may be dullness detected on percussion, crackles, wheezes, or ronchi. With progressive, longstanding disease, physical findings related to chronic hypoxia can develop. Pulmonary function tests results have shown a restrictive pattern but they may also be normal.[22,23] Acute presentation simulating infectious pneumonia with fever, with or without cough, is unusual.[24] However, acute presentation with respiratory failure has been reported with accidental/suicidal massive exposure to mineral oil but not with submassive exposure or inhalation and in children.[25-28] Laboratory test results are usually normal although leukocytosis and an increased erythrocyte sedimentation rate may occur, especially when complicated by infection.

RADIOLOGICAL FEATURES

Chest X-ray findings are diverse and can mimic many other diseases including carcinoma, acute or chronic pneumonia, ARDS, or a localized granuloma.[29-31] A homogenous dense consolidation, often with air bronchograms, and sometimes a fine, “spun glass” appearance may be appreciated in early disease.[29] Involvement may be diffuse or focal, unilateral or bilateral, although unilateral pneumonic consolidation with lower lobes predilection is more commonly reported.[29,30,32,33] Occasionally, one may also
A reticular pattern may be observed as the emulsified oil leaves the alveoli and enters the interstitium and lymphatics, creating edema, inflammation, and fibrosis in those sites. Fibrosis and coalescence of oil can result in nodules and masses with irregular margins, closely mimicking lung cancer. Atelectasis and pleural effusion have also been described. CT scan and magnetic resonance imaging can detect fat within pulmonary tissue. The most commonly described features are alveolar consolidations of low attenuation values (-30 to -150 HU), ground glass opacities with thickening of intralobular septa (crazy paving pattern), or alveolar nodules. Magnetic resonance imaging may reveal high signal intensity on T1-weighted imaging that is consistent with lipid content. Lower lobe predominance of the radiographic findings are often described but not uniformly seen.

**PATHOPHYSIOLOGY**

Pathologically, LP is a result of a chronic foreign body reaction to inhaled/aspirated mineral oil. Mineral oils enter the tracheobronchial tree without stimulating cough reflex and impair the mucociliary transport system. Therefore, once inside the alveoli, it is difficult to expectorate the lipid out. This situation may be complicated by some associated neurological and gastrointestinal disorders that affect swallowing and the palatal or cough reflex. Once in the alveoli, oil is taken up by macrophages after emulsification and because alveolar macrophages cannot metabolize the fatty substance, the oil is repeatedly released into the alveoli after the death of the macrophages. The oil thus released illicits a giant cell granulomatous reaction (hence, LP is also called lipid granulomatosis), chronic inflammation, and alveolar and interstitial fibrosis. Evolution of the lesions with time has been described. Fresh lesions show alveolar infiltration by lipid-laden macrophages and almost normal alveolar walls and septa. Advanced lesions show larger vacuoles and inflammatory infiltrates of the alveolar walls, bronchial walls, and septa. Fibrosis and parenchymal destruction around large lipid-containing vacuoles are features of the

![Figure 1](Noncontrast computerized tomographic images of young patients with lipoid pneumonia. Mediastinal, (a) and lung window, (b) showing consolidation in right middle lobe, lower lobe, and also left lung. Few low density areas (arrow) are seen within the middle lobe consolidation. (Reproduced with permission from Biomed Central and Cases Journal)](image-url)
oldest lesions. Special staining techniques (oil red O, Sudan black etc) can more effectively demonstrate that the vacuoles are lipid-filled. The specific type of oil aspirated can be determined by chemical analysis, spectroscopy, and chromatography.\textsuperscript{[44,45]}

**DIAGNOSIS**

Diagnosis of LP is often difficult as symptoms, signs, and radiographic findings are all quite nonspecific. LP is difficult to diagnose clinically, and physicians may not consider this in the differential diagnoses unless a detailed history of the use of fatty substances is taken. In suspected cases, diagnosis of lipoid pneumonia depends on detecting fat-laden macrophages in the specimen. Frozen samples should be examined by various stains to discriminate various types of oils. Chromatography and infrared spectroscopy may also be used. Examination of the sputum, bronchoscopic alveolar lavage (BAL), fine needle aspiration cytology (FNAC), or biopsy from the lesion may be used for demonstrating the presence of lipid-laden macrophages [Figure 2]. Sputum examination may be used but with questionable reliability. BAL may reveal a turbid or whitish fluid with fat droplets visible at the surface.\textsuperscript{[46]} FNAC may be diagnostic but false negative results have also been reported to occur.\textsuperscript{[47]} In some cases, lung biopsy or even surgical biopsy may be required.\textsuperscript{[48,49]}

**TREATMENT**

Treatment of LP is not well studied and experience with treatment are only based on case reports. Avoiding ongoing exposure and providing supportive care is the mainstay of treatment. There are anecdotal reports of the use of systemic corticosteroids to slow the inflammatory response.\textsuperscript{[50]} However, as corticosteroids are not to be used routinely, their use is only if the lung injury is severe and ongoing. Other treatment modalities described in literature are immunoglobins,\textsuperscript{[51]} and whole lung lavage.\textsuperscript{[52,53]} Repeated bronchoalveolar lavage (BAL) with saline has been shown to be an effective therapeutic modality for the treatment of LP.\textsuperscript{[28]} Some researchers successfully used a solution of 0.05% polysorbate 80 in Ringer’s lactate as a fat emulsifier for repeated therapeutic BAL.\textsuperscript{[43]} Some authors have described resection of nodules and masses in these cases.\textsuperscript{[17,54]} Surgical resection is usually unwarranted as lipoid pneumonia is typically indolent and may regress spontaneously. Resection may, however, be done in cases of high suspicion of cancer.\textsuperscript{[23]}

**PROGNOSIS AND COMPLICATIONS**

Lipoid pneumonia is usually indolent although it may be progressive also. Risk factors for progressive disease are concurrent debilitating illness and continued exposure to mineral oil. Patients may show persistent or progressive radiological abnormality despite symptomatic relief.\textsuperscript{[8]} Additionally,
this may be complicated by superinfection by microorganisms like *Mycobacterium chelonei*, *M. fortuitum*, and other bacteria.[55,56] Colonization by Cryptococcus has also been reported.[22] Protracted exposure may cause respiratory insufficiency and may lead to cor-pulmonale.[57] Hypercalcemia is one of the uncommon complications of granulomatous reaction as seen in tuberculosis and sarcoidosis.[58] Association of lipoid pneumonia with lung cancer has also been reported in a few cases.[59,60]

REFERENCES


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