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Case Report

Nocardia and *Mycobacterium fortuitum* infection in a case of lipoid pneumonia

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ABSTRACT

Exogenous lipoid pneumonia is a potential problem with any form of oil aspiration. The association of *Mycobacterium fortuitum* with lipoid pneumonia is a rare occurrence and ascribed to as yet-unknown interplay between the oil in the pulmonary parenchyma and the waxy Mycobacterial cell wall. We describe a case of lipoid pneumonia due to "oil aspiration", complicated by infection with *Mycobacterium fortuitum* as well as *Nocardia*, in an individual with no obvious immune deficiencies.

1. Introduction

Lipoid pneumonia is a chronic inflammatory reaction of the lungs to the presence of lipid substances. It is of two types, exogenous and endogenous. The endogenous variety is due to release of lipids from lysis of lung tissue on account of tumor, drugs, radiation, etc. Exogenous lipoid pneumonia results from the aspiration of vegetable, animal, or most commonly mineral oils. The presence of a lipoid environment seems to favour *Mycobacterium fortuitum* infection. We present such a case with exogenous lipoid pneumonia associated with *Mycobacterium fortuitum* infection superadded with *Nocardia* infection.

2. Case history

A 60-year-old male presented to the outpatient department with fever of four days duration, and cough with scanty expectoration. The patient, a non-smoker, had been involved in running a paper-manufacturing unit until eight years ago. Three years ago he had been treated for pulmonary tuberculosis of the right middle lobe (Brock's syndrome), and he had completed the complete course of anti-tubercular therapy with good compliance.

One month ago, the patient accidentally drank coconut oil stored in a drinking water bottle and had a choking episode followed by cough that lasted a few days. General examination was mostly unremarkable, and there was no obvious clubbing or superficial lymphadenopathy. Physical examination of the chest revealed sparse crackles in the right inframammary area and also bilaterally in the infrascapular areas.

The total leucocyte count was 16.9×10^3 (Neutrophils 80%), and the Erythrocyte Sedimentation Rate (ESR) 60 mm. Human Immunodeficiency Virus (HIV) test was negative. The chest radiograph showed bilateral basal patchy consolidation, much more prominent on the right side (see Fig. 1). A Computerized Tomographic (CT) scan of the chest showed dense consolidation with an airbronchogram in the right middle lobe and alveolar infiltration in left lingula and both the lower lobes (see Fig. 2). These showed a moderate degree of heterogenous post-contrast enhancement. Multiple nodular opacities were also observed in both lungs. Sputum showed acid-fast bacteria (grade 3+) on direct staining.

Since it was thought that tuberculosis could not by itself explain the radiological abnormality—particularly the nodularity—the consolidated area was targeted with a CT guided transthoracic needle aspiration. Cytological features of granulomatous inflammation were seen. The stains were positive for fat globules (see Fig. 3). Acid-fast bacilli were noted, along with the characteristic delicate gram-positive weakly acid-fast filaments of *Nocardia* (see Fig. 4). Culture of both the sputum and the fine-needle aspirate grew *Mycobacterium fortuitum* which eventually showed sensitivity to ethambutol, sparfloxacin, ofloxacin, capreomycin and amikacin—and resistance to streptomycin, isoniazide, rifamycin, pyrazinamide and clofazimine.

The patient, who had already been started on the standard four-drug anti-tubercular therapy plus sulphonamides, was commenced on ofloxacin, amikacin and clarithromycin. Pyrazinamide was withdrawn. At the end of eighteen months, the patient had shown complete clinical and radiological recovery (see Fig. 5), and treatment was discontinued.

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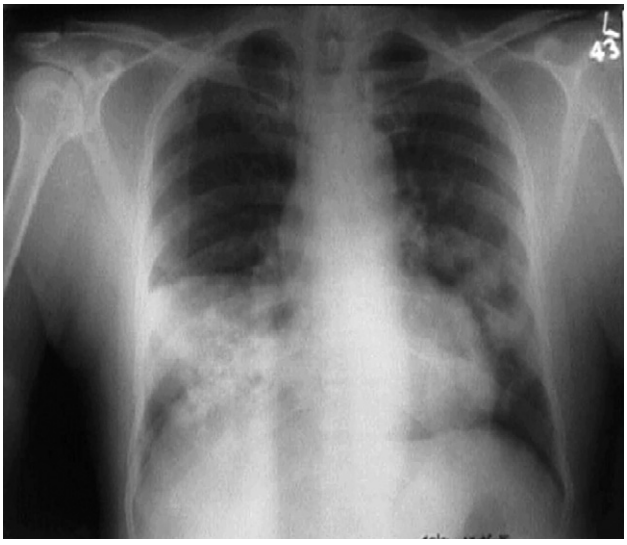

Fig. 1. Chest radiograph showing right middle lobe consolidation with left mid zonal infiltration.

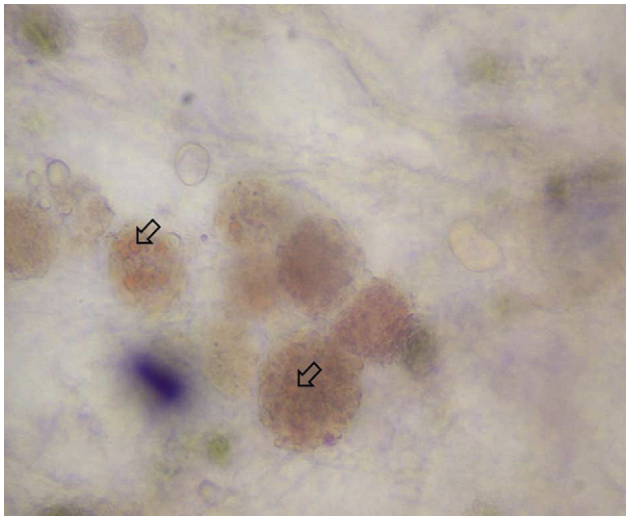

Fig. 3. Fine needle aspiration cytology of lung showing fat globules (arrows) within the macrophages.

3. Discussion

Lipoid pneumonia is of two types: endogenous and exogenous. In the *endogenous* variety there is lysis of lung tissue distal to an obstruction by tumor or broncholitis obliterans. Release of lung lipids can also occur in association with chemotherapy, amiodarone therapy or radiotherapy. Lipid like material is also found in alveolar proteinosis. *Exogenous* lipoid pneumonia can occur in a variety of unusual settings such as in blackfat tobacco smoking seen in Guyana[1] or by exposure to oil mists,[2] but it most often occurs in the setting of medicinal use of liquid paraffin for chronic constipation by accidental aspiration. Oil based nasal drops can also result in lipoid pneumonia, but these are less commonly used nowadays for obvious reasons. Mineral oils being relatively inert are less prone to produce alveolar inflammation (this explains the paucity of symptoms in some individuals) than animal fats (such as in milk: these are hydrolysed into fatty acids by lung lipases). Lipases digest the lung parenchyma and evoke a vigorous inflammatory response. Vegetable oils can be aspirated during feeding or during emesis and have a highly variable effect. Several individuals have associated esophageal problems predisposing to aspiration. Lipoid pneumonia has no characteristic radiologic appearances but a "Paraffinoma" can cast a nodular infiltrate chest x-rays or CT films, and may present as a "hot spot" on Positron Emission Tomography (PET) scans mimicking malignancy.[3] The diagnosis must therefore be suspected in the setting of the appropriate history with radiological infiltration, usually in the basal segments of the lower lobes, the superior segment of the lower lobe or the posterior segment of an upper lobe. Bronchoalveolar lavage or lung histopathology will then usually demonstrate lipid laden foamy macrophages with occasional giant cells. True granulomas are unusual.

The association of Nontuberculous Mycobacteria (NTM) with lipoid pneumonia has been noticed. It is intriguing that Mycobacteria, possessed of the most complex cell wall in nature (that enabled their survival at the interface of water and soil over the millenia), appear to derive sustenance from the lipids as in lipoid pneumonia. This in some way actually appears to increase

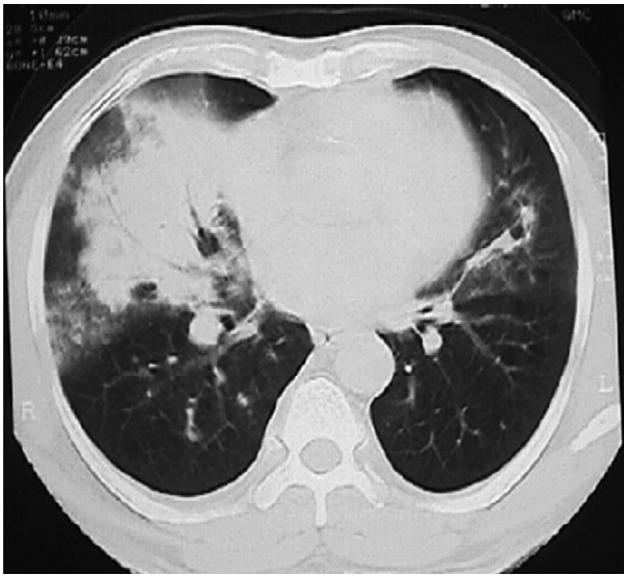

Fig. 2. Computerized tomography of chest in the lung window, showing right middle lobe consolidation with airbronchogram and left lingular alveolar infiltration.

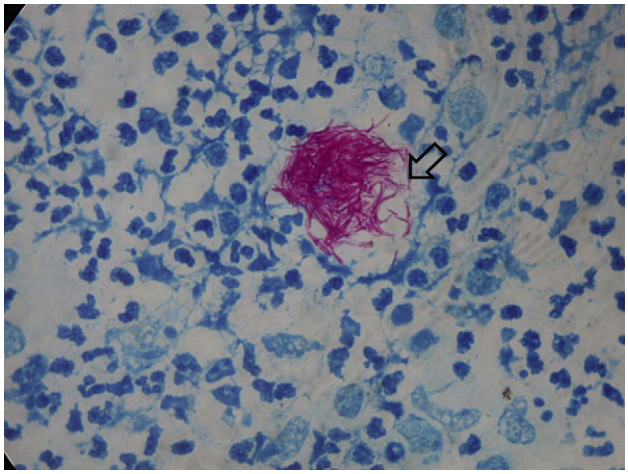

Fig. 4. Fine needle aspiration cytology of the lung showing weakly acid-fast filamentous *Nocardia* (arrow).