An 85-Year-Old Man With a Lung Mass*

David Adkins, MD; and Eric S. Bensadoun, MD, FCCP

A 85 year-old man was seen in consultation for an abnormal chest radiographic finding. His medical history was remarkable for hypertension, glaucoma, and lifelong dysarthria due to cerebral palsy. In recent years, signs of early dementia had developed, and he had to use a walker because of an unsteady gait. A chest radiograph after a recent fall showed an abnormality, which prompted the consultation.

At the time of evaluation, he complained of dyspnea on exertion and intermittent nonexertional retrosternal chest pain of 6-months duration. He denied cough, fever, hemoptysis, or weight loss. On review of systems, he complained of dyspepsia and long-standing constipation. His medications included an antihypertensive, timolol eye drops, calcium carbonate for dyspepsia, and psyllium for constipation. He had quit smoking cigarettes 56 years ago.

On examination, his vital signs were normal. His neck was fixed in flexion, and marked kyphosis was evident. Crackles were present at the lung bases bilaterally. The remainder of the examination was unremarkable. Laboratory tests including a CBC and chemistry panel were unremarkable. The chest radiograph (Fig 1) showed a noncalcified mass in the right upper lobe. A CT scan of the chest was performed (Fig 2).

What is the diagnosis?

![Figure 1. Chest radiograph showing a right upper lobe mass.](image1)

![Figure 2. CT scan of the chest with (top) lung and (bottom) mediastinal windows showing an irregular, spiculated mass in the right upper lobe; the central portion of mass measures between –40 Hounsfield units and –60 Hounsfield units.](image2)

*From the Division of Pulmonary and Critical Care Medicine, University of Kentucky, Lexington, KY. Manuscript received July 21, 2003; revision accepted September 19, 2003. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (e-mail: permissions@chestnet.org). Correspondence to: Eric S. Bensadoun, MD, FCCP, University of Kentucky, Division of Pulmonary and Critical Care Medicine, 800 Rose St, MN614, Lexington, KY, 40536-0298, e-mail: ebens0@uky.edu
Diagnosis: Exogenous lipid pneumonia (paraffinoma)

The CT scan of the chest (Fig 2) showed a right upper lobe mass with a hypodense center that measured between -40 Hounsfield units and -60 Hounsfield units, which is consistent with the density of fat. This raised the possibility of lipid pneumonia; although the patient could not recall ingesting mineral oil, a search of his household by a family member revealed several partially consumed bottles of mineral oil.

Lipoid pneumonia can be characterized as exogenous or endogenous based on the source of the lipid. Endogenous lipid pneumonia results from the lipid-containing material released from degenerating alveolar cell walls distal to an airway obstruction. The obstruction is usually due to a lung carcinoma, although endogenous lipoid pneumonia has also been described in association with inflammatory conditions such as bronchiectasis and lung abscess.

Exogenous lipid pneumonia usually occurs when an oily substance reaches the alveoli either by aspiration or inhalation. The pulmonary tissue reaction to lipid material depends largely on the source of the lipid. Animal oils (eg, cod liver oil) cause intense inflammation, while vegetable oils (eg, olive oil) result in little inflammation. Mineral oil, which is relatively inert, is initially emulsified and ingested by macrophages. With repeated aspiration, mineral oil can elicit a foreign body reaction that can result in fibrosis. As the fibrosis progresses, the oil may coalesce to form large droplets walled off by a rim of fibrous tissue and giant cells, creating a tumor-like mass called a paraffinoma.

The most common cause of exogenous lipid pneumonia is chronic aspiration of mineral oil used as a laxative; less frequent is the inhalation of oil-based nose drops. Studies in animals and humans have shown that mineral oil does not elicit the cough reflex and impairs mucociliary clearance, thus favoring entry into the distal airspaces. Predisposing factors for aspiration such as advanced age, debilitated state, gastroesophageal reflux, and neurologic disorders that interfere with cough or swallowing have all been associated with cases of exogenous lipid pneumonia; however, many cases occur in patients without any predisposing conditions.

Most patients with exogenous lipid pneumonia are elderly, in the sixth or seventh decade of life. Almost half of the patients with exogenous lipid pneumonia are without symptoms on presentation, and are only identified because of an abnormality seen on a chest radiograph. Patients with symptoms commonly present with chronic cough or dyspnea; fever, weight loss, chest pain, and hemoptysis are less common. The physical examination may be normal, or may reveal crackles or wheezes.

Chest radiographs in exogenous lipid pneumonia most commonly show airspace consolidation; however, reticular patterns, mixed alveolar/interstitial patterns, and nodular lesions have also been described. The abnormalities are often found in the dependent portions of the lower lobes or in the right middle lobe, and may be multifocal and bilateral. CT and high-resolution CT in exogenous lipid pneumonia have shown alveolar consolidation, ground-glass opacities, interstitial abnormalities, and nodular lesions. On CT scan, a consolidation or a mass with low attenuation is a characteristic finding. Although this finding is not present in all cases, a negative density (between -150 Hounsfield units and -30 Hounsfield units) is highly suggestive of intrapulmonary fat and a diagnosis of lipid pneumonia. The CT angiogram sign (the visualization of vessels within an area of consolidation on postcontrast images), and the crazy paving pattern (the superimposition of ground-glass opacity and a reticular pattern due interlobular septal thickening), which is more commonly seen with pulmonary alveolar proteinosis, have also been described in exogenous lipid pneumonia.

At this time, MRI does not appear to be as helpful as CT in establishing the diagnosis of lipid pneumonia. The diagnosis of exogenous lipid pneumonia is based on a history of exposure to oil, a compatible radiograph or CT scan, and the presence of lipid-laden macrophages on sputum or BAL analysis. If the diagnosis remains uncertain, transbronchial biopsy or open-lung biopsy may be necessary.

Once the diagnosis is made, treatment essentially consists of preventing further exposure. Once exposure has stopped, the radiographic findings improve or remain stable in the majority of patients. Systemic steroids and therapeutic BAL have been advocated in some cases, but a benefit has not been definitively shown.

In conclusion, although the diagnosis of lipid pneumonia is uncommon, it still needs to be considered in elderly patients. Lesions may mimic lung cancer and CT/high-resolution CT appears to be the imaging modality of choice for establishing the diagnosis of lipid pneumonia.

In our patient, the diagnosis of exogenous lipid pneumonia was made on the basis of characteristic findings on CT scan and evidence of exposure to mineral oil. No further testing was done, and the patient was advised to discontinue the use of mineral oil. Follow-up chest radiograph at 6 months showed that the lesion was unchanged.
REFERENCES


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