Shark Oil Pneumonia*
An Overlooked Entity

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Nonprescription drugs are often believed to be innocuous. We present a case of lipid pneumonia from ingestion of squalene, a derivative of shark liver oil, a popular over-the-counter Asian folk remedy. (Chest 1993; 103:976-77)

Lipoid pneumonia has been recognized since the early 20th century as a disease process in the lung related to the abnormal presence of organic or inorganic lipids. The first case was described in 1925 by Laughlin. An incidence of 2 to 5 percent was found in one autopsy study. To our knowledge, the case presented is the first description of a severe exogenous lipid pneumonia by means of aspiration of squalene, derived from shark liver oil. The capsules are readily available in Asian health food stores. Squalene is also used widely in cosmetics.

CASE REPORT

A 46-year-old hypertensive Korean man was admitted to Flushing Hospital complaining of three days of progressive left facial and extremity weakness. He denied taking any medications.

On physical examination, the only abnormal finding was a dense left hemiplegia with a left central facial nerve palsy.

Laboratory studies at the time of hospital admission included a normal complete blood cell count, electrolytes, and urinalysis. The aspartate aminotransferase was 67 U/L, alanine aminotransferase was 120 U/L, y-glutamyl transpeptidase was 129 U/L and lactate dehydrogenase was 263 U/L. Hepatitis profile was negative. Electrocardiogram suggested old inferior wall infarction and chest roentgenogram was normal. A computed tomographic (CT) scan of the head revealed bilateral basal ganglia infarcts, and sinus views were normal. Liver sonography showed fatty infiltration.

On day 3, he developed singultus requiring chlorpromazine (Thorazine). On day 6, the temperature was 38.4°C. Intravenous (IV) ceftazolin therapy was started after a repeat chest roentgenogram showed extensive left perihilar infiltrate and minimal right perihilar consolidation (Fig 1). Arterial blood gas determination on 35 percent oxygen was a pH of 7.44, Pco2 of 35 mm Hg, HCO3 of 23 mmol/L, and oxygen saturation of 97 percent. All blood and urine cultures were negative. Sputum Gram stain showed leukocytes with mixed flora and cultured only yeast. The patient continued to spike fever and maximal leukocyte count was 16,500/cu mm. Vancomycin, ceftriaxone, and erythromycin were substituted for ceftazolin. A CT scan of the chest revealed left upper and lower lobe and right lower lobe consolidation. Cold agglutinins, Legionella, and Mycoplasma titers, and serum precipitins were negative.

Fiberoptic bronchoscopy was performed on day 24 and all smears and cultures, including routine, mycobacterial, Legionella, Mycoplasma, and viral studies were negative on bronchoalveolar lavage fluid (BAL). Cytologic examination of BAL fluid revealed abundant lipid-filled macrophages (Fig 2). Transbronchial biopsy specimens from the left lower lobe showed marked fibrous connective tissue proliferation with aggregation of histiocytes simulating granulomas and lipid-laden macrophages. Special stains were negative. Antibiotic therapy was stopped and steroid therapy was begun. He defervesced with slight regression in the pulmonary infiltrates. After repeated questioning, it was discovered that he had taken 10 squalene capsules per day for the past year and that during hospitalization, his wife had been feeding him multiple capsules daily as a cure for hiccoughs and without the knowledge of the medical staff.

DISCUSSION

Lipoid pneumonia is divided into endogenous and exogenous forms. In the case of endogenous (cholesterol) pneumonia, the fatty materials are derived from the lung itself as in malignant obstructing bronchial tumors or bronchiectasis. In the more commonplace exogenous lipid pneumonia, lipids enter the lung parenchyma as a result of aspiration. The effects on the lung are primarily related to the content of fatty acid: vegetable oils such as poppyseed and sesame produce little if any pulmonary parenchymal damage vs chaulmoogra oil, which is rich in fatty acids. Mineral oil is fairly inert and causes an influx of phagocytizing alveolar macrophages with an accompanying infiltrate within several

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976

Shark Oil Pneumonia (Asnis, Saltzman, Melchert)
days and granulomas with fibrosis in two to three months. Animal fats produce the most injury to the lung when aspirated because of their high fatty acid content. Those described previously include milk fat, lard oil, rabbit fat, and cod liver oil.

Conditions associated with lipoid pneumonia include debilitated patients with achalasia, esophageal diverticulum, neurologic diseases, or altered mental status. Extremes of age are favored. The typical presentation is a patient with a chronic cough or an asymptomatic patient with incidental chest roentgenographic findings. On chest roentgenogram, either a diffuse multilobar infiltrate or a circumscribed lower lobe infiltrate develops. The diagnosis is made by sputum examination for fat with sudan IV stain. Macrophages with orange-brown vacuoles will be found in 90 to 95 percent. Bronchial washings, transbronchial biopsies, or open lung biopsies are occasionally needed. The treatment is to terminate the offending agent. Antibiotics are given if there is a superimposed infection. In severely ill patients, prednisone 40 to 60 mg is helpful and dosage is tapered over a month. Surgery is performed if concurrent malignancy is suspected.

In our case, the patient ingested squalene for one year and had abnormal liver function. Squalene fed to dogs in doses of 1,200 mg/kg orally accumulated 6 percent of the daily dose in their livers without toxic signs or enzyme abnormalities. Our experience suggests that hepatic squalene accumulation may be more toxic in humans than animal studies would have predicted.

It is proposed that the medical community be aware of the widespread use of squalene in Oriental communities and of its capacity to produce a marked exogenous lipoid pneumonia and possibly liver toxicity as well.

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REFERENCES