Lipoid pneumonia in a 40-day-old infant

Pneumonia lipoide em lactente de 40 dias de vida

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To the Editor:

Lipoid pneumonia is triggered by aspiration of endogenous or exogenous lipids.[1] Exogenous lipoid pneumonia is the most common form of lipoid pneumonia in children and is usually caused by the use of mineral oil to treat constipation.[1,2] Lipoid pneumonia is a difficult-to-diagnose disease, whose clinical and radiological presentation is nonspecific and similar to that of various other lung diseases, being asymptomatic in some cases.[1,3] The chronic progression can trigger pulmonary sequelae, such as bronchiectasis and pulmonary fibrosis.[2] Although the scientific community has issued warnings about the dangers of using mineral oil in children, the prescription of mineral oil for the treatment of constipation is still common practice in Brazil because this medication is inexpensive and widely available.[1,2]

In the case described here, a 40-day-old female patient was admitted to the emergency room of a secondary care hospital with a four-day history of moaning. On the second day of moaning while under outpatient care, the patient was prescribed mineral oil (because she had not evacuated for 2 days). Upon receiving the medication, the infant choked and, from that moment on, she had cough and breathlessness. She was exclusively breastfed and had no history of disease or regurgitation. Physical examination revealed moaning, tachypnea (RR = 80 breaths/min), wheezing, and hypoxemia (SpO_2 = 88%). Blood workup showed mild leukopenia with normal differential cell count, and chest X-ray revealed bilateral alveolar-interstitial opacities and lung hyperinflation (Figure 1). Blood cultures were negative. The patient received intravenous antibiotic therapy (ampicillin), oxygen therapy (via an oxygen tent), inhaled fenoterol, and respiratory therapy. On postadmission day 7, she still had clinical changes. Radiological findings remained unchanged. A CT scan of the chest was taken and showed areas of parenchymal consolidation affecting segments of upper and lower lobes with negative attenuation coefficients (Figure 2), findings that were consistent with a presumptive diagnosis of lipoid pneumonia with multisegmental involvement.

The infant was transferred to a tertiary care hospital for bronchoscopy and BAL. She underwent three sequential procedures, performed with a rigid bronchoscope, under intravenous general anesthesia and jet ventilation. In the first procedure, anatomical changes were ruled out. Material similar to an opalescent film was found to coat the trachea and bronchi. A BAL was performed in the right lower lobe. Cytological examination of the BAL fluid showed cells containing inclusions suggestive of lipids.

In the second bronchoscopy, there was a small quantity of diffuse opalescent secretion. A BAL was performed in the left lower lobe, and analysis of the BAL fluid revealed numerous lipid-laden cells and few structures suggestive of fat droplets. In the third procedure, a BAL was performed in the right upper lobe, structures suggestive of fat droplets and numerous lipid-laden cells having been found. The patient recovered well; she gained appreciable weight and became asymptomatic, radiological normalization being complete.

Mineral oil (liquid paraffin or liquid petroleum jelly) is a by-product of the petroleum distillation process that produces gasoline. It can be purified to be used for medicinal purposes as a moisturizer (cream) or as a laxative in the treatment of constipation and partial bowel obstruction caused by severe ascariasis. It is characterized as a transparent, unpleasant-tasting liquid that is not absorbed from the digestive tract. In the intestinal lumen, mineral oil reduces water absorption and acts as a lubricant.[2] It has low volatility and high viscosity, both of which produce undesirable effects, such as an inhibition of the cough reflex[1,2] when the oil is aspirated and a reduction in mucociliary transport in the lung because the oil changes the viscoelastic properties of secretions, thus impairing lung clearance.[2] Although chronic aspiration of mineral oil is

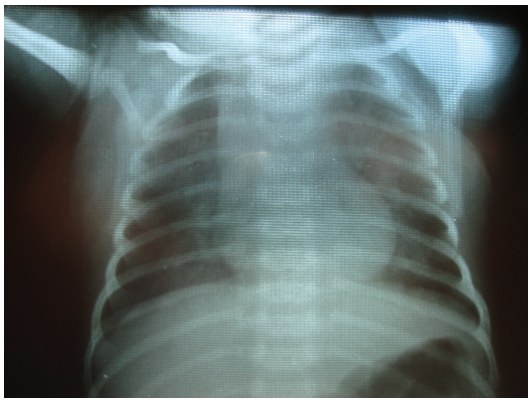

Figure 1 - Chest X-ray revealing a homogeneous opacity consistent with bronchopneumonia. Image taken before treatment with BAL.

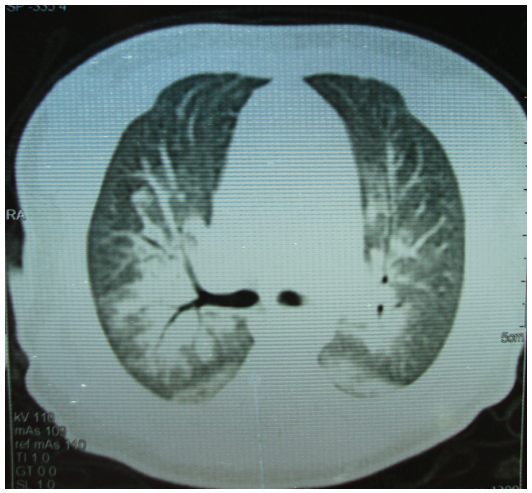

Figure 2 - HRCT scan of the chest revealing areas of parenchymal consolidation affecting segments of upper and lower lobes with negative attenuation coefficients. Image taken before treatment with BAL.

facilitated by dysphagia, esophageal diseases, and neuropathies, it can occur in patients who have no anatomic predisposition or functional abnormality,[4] as well as in those who resist taking the medication (e.g., infants).[1] When aspirated, mineral oil rapidly disseminates throughout the bronchial tree. It is not metabolized by pulmonary enzymes. When it reaches the alveolar space, it is phagocytosed by alveolar macrophages.[1,5] Although some of these cells penetrate the interstitial tissue and reach the peribronchial lymphatic vessels and hilar lymph nodes, most of the mineral oil remains in the alveoli, either free or within macrophages, which cannot metabolize it and therefore disintegrate, returning it to the airspace. The activation of macrophages in the airspace promotes cytokine release and an inflammatory reaction.[1,6] Initially, there is an inflammatory foreign body reaction. Subsequently, there is chronic interstitial inflammation that progresses to pulmonary fibrosis. This vicious cycle contributes to the chronicity of the disease, even years after discontinuation of the use of the product. In anatomic pathology studies, lipoid pneumonia is characterized by the presence of giant cells, alveolar/interstitial fibrosis, and chronic inflammation. Depending on the duration of the disease, lipid-laden alveolar macrophages, as well as normal alveolar wall and septa, can be initially found. Advanced lesions show large vacuoles and inflammatory infiltrate in the alveolar walls, bronchial walls, and septa. Older lesions are characterized by fibrosis and parenchymal destruction around large, lipid-laden vacuoles.[7]

Radiologically, acute lipoid pneumonia presents as bilateral pulmonary opacities, in segmental or lobar distribution, involving mainly the posterior and lower lobes. Other manifestations of lipoid pneumonia include nodules, pneumatoceles, and pleural effusion. Pneumomediastinum and pneumothorax are rare and indicate a poorer prognosis. Chest CT can reveal areas of alveolar consolidation with low attenuation and a ground-glass pattern.[4,5]

The diagnosis of lipoid pneumonia should be based on the following: a history of mineral oil ingestion; the risk factors described above; clinical and radiological findings; and demonstration of lipids in bronchoscopy, BAL, or lung biopsy specimens.[1,8] The presence of lipid-laden macrophages in BAL fluid is considered to be the most important finding for the diagnosis of lipoid pneumonia.[1,2,8] Although spontaneous resolution can occur after discontinuation of the use of mineral oil, complications can occur, including bacterial infection, progressive fibrosis, bronchiectasis, hemoptysis, and severe cases that progress to respiratory failure and death.[1,3,6] The best form of treatment has yet to be well established in the scientific literature. Various studies have demonstrated that treatment with multiple BALs is effective, has few risks, and leads to resolution of the clinical and radiological signs.[1,2,5,8,9] In patients with pneumonia that does not respond to treatment, the hypothesis of lipoid pneumonia should be considered in the differential diagnosis. We strongly support

the recommendation that mineral oil be used judiciously in the treatment of chronic constipation. This medication should not be prescribed to neonates or infants, who resist taking it, or to children with developmental delay, with or without dysphagia.

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Submitted: 12 February 2012. Accepted, after review: 1 March 2012.