

Lipoid Pneumonia Following Aspiration of Ghee (animal fat) in an Omani Infant

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Abstract

We are reporting, what we believe, is the first case of biopsy proven exogenous lipid pneumonia in an Omani infant. Exogenous lipid pneumonia is caused by aspiration of oily products. One reason is the traditional practice of giving infants oily products (Ghee) as in our patient.

Keywords: Pneumonia, Lipid, bronchoalveolar lavage, exogenous lipid pneumonia, lipid laden macrophages

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Introduction

Lipoid pneumonia caused by aspiration of oily products poses a diagnostic challenge, both for the clinician and the radiologist. Clinically it is difficult to differentiate from other causes of pneumonia. Lipoid pneumonia became very uncommon since the decline in the use of oil based medications; however it is still reported in patients treated with liquid paraffin (mineral Oil) for constipation.^{1,2} One reason for this form of pneumonia in some developing countries is the traditional practice of giving infants oily products for various reasons.³⁻⁷ We report, what we believe, is the first case of biopsy proven lipid pneumonia in an Omani infant.

Case report

A forty-five day old Omani female infant presented to a secondary hospital with a few days history of fever, cough, and rapid breathing. Examination revealed her to be in significant respiratory distress, requiring high flow of oxygen to maintain normal oxygen saturation. She had bilateral decreased breath sounds and rales upon auscultation of her chest. The chest x-ray (CXR) showed bilateral extensive consolidation involving mainly the lower lobes of the lungs. Her Full Blood Count (FBC) showed significant leukocytosis and neutrophilia with WBC $27 \times 10^9/L$, and absolute neutrophil count $20 \times 10^9/L$. Blood cultures showed no growth of any organisms. She was managed in the Pediatric Intensive Care Unit (PICU) with broad spectrum antibiotics and non-invasive ventilatory support with continuous positive airway pressure (CPAP). Despite the use of different courses of antibiotics over a period of 25 days, she only showed minimal clinical improvement and was failing to thrive. Therefore, she was referred to Sultan Qaboos University Hospital (SQUH) to rule out immunodeficiency.

Upon arrival at SQUH, the infant was in respiratory distress with tachypnea, intercostal and subcostal recessions, and requiring 5L/minute of oxygen to keep her oxygen saturation above 90%. Examination of the chest revealed decreased breath sounds with diffuse rales bilaterally. A repeat CXR showed similar findings to the previous

x-ray (Figure 1). Computed tomography (CT) scan (Figure 2) revealed extensive bilateral consolidation in right upper lobe and middle lobes as well as lower lobes and no anatomical abnormalities. She was managed in PICU with non-invasive ventilatory support and broad spectrum antibiotics including antifungal agents for three more weeks.



Figure 1: Plain chest x-ray showing bilateral extensive consolidations.

An extensive battery of investigations was carried out including a complete immunological work up which was normal. Nasal brush biopsy was not suggestive of Primary Ciliary Dyskinesia. A barium swallow showed no tracheo-esophageal fistula but mild gastroesophageal reflux. Bronchoscopy did not reveal any significant anatomical abnormalities or foreign bodies and the bronchoalveolar lavage aspirate (BAL) showed epithelial and inflammatory cells as well as macrophages. BAL was sent for microbiological studies including Gram and Ziehl-Neelsen staining, and bacterial and fungal cultures. All the results were unremarkable. Serological and Polymerase Chain Reaction were carried out to test for viruses including Cytomegalovirus, Epstein-Barr virus and Herpes Simplex Virus, all were negative. Sweat chloride was normal.



Figure 2: CT scan of the lung showing extensive bilateral dependent lung consolidation

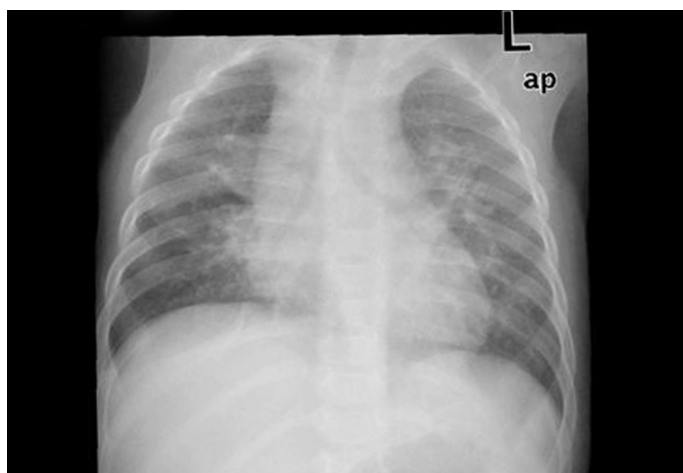


Figure 3: Plain chest x-ray 6 months after treatment showing almost clear lung fields.

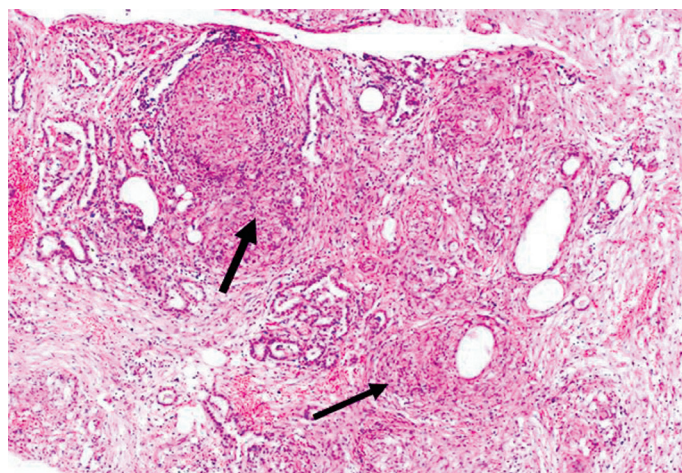


Figure 4: Low power H&E view of the lung biopsy showing a large collection of Vacuolated macrophages, inflammatory cells and extensive fibrosis

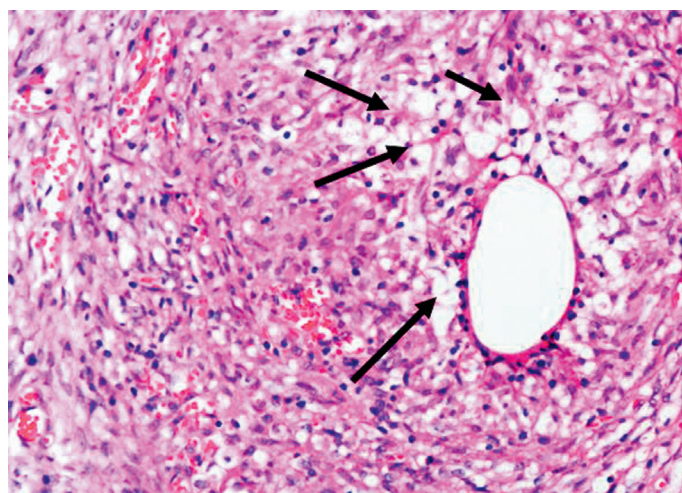


Figure 5: Lung biopsy showing a collection of macrophages with lipid vacuoles Arros (H&E x 20)

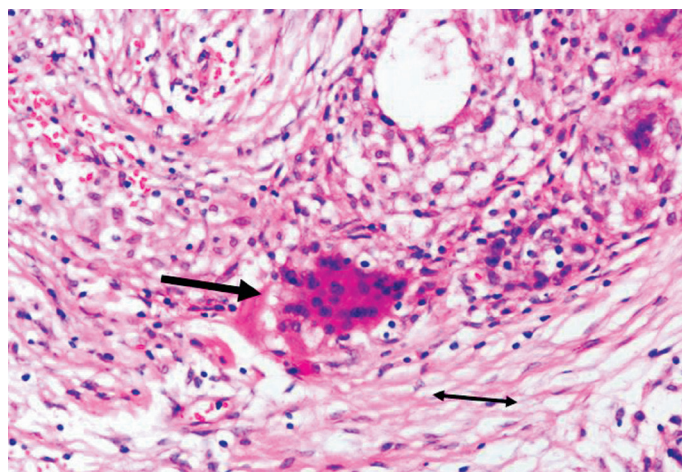


Figure 6: Lung biopsy showing a foreign body giant cells (one head arrow), extensive interstitial fibrosis (double headed arrow) H&E x 20

Subsequently around day 50 of admission in SQUH, as the patient showed mild clinical improvement and no radiological improvement an open lung biopsy was performed in Royal Hospital. This revealed extensive macrophages with lipid vacuoles, foreign body giant cells, fibrosis and inflammatory cells all suggestive of exogenous lipoid pneumonia. (Figures 4-6).

With this biopsy result we question the parents about administering oil products to their daughter; it became apparent that her grandmother had fed her ghee (animal fat) during the first week of life as a tradition to ensure her well being.

Subsequently the infant was started on oral prednisolone 2mg/kg/day for a total of 3 weeks. Within one week of this treatment, she started to improve clinically, 2 weeks later she was off oxygen supplementation and upon discharging home at 3 weeks she was bottle feeding with marked clinical and radiological improvement. The steroids were tapered gradually over 9 weeks, followed by inhaled beclomethasone 250 mcg BID for 6 months. At 6 months follow up, she had no respiratory symptoms and was thriving very well. Her CXR was almost normal (Figure 3)

Discussion

Traditional feeding of ghee (animal fat) to newborns has been practiced over many generations in Oman to help weight gain in infants with the belief that maternal colostrum is unhealthy.³ This practice has declined greatly over the years. However, though the current frequency is unknown, this practice is apparently still present in some areas of Oman. Other countries where similar practices, for different beliefs and reasons, are seen are Saudi Arabia, India, Mexico and Ghana.³⁻⁸

Oil aspiration suppresses the cough reflex and inhibits the normal muco-ciliary clearance in the airways, thus depriving it from its primary defense mechanism. The chemically inert oils, such as vegetable oils are not hydrolyzed by lung lipases so it induces a 'foreign body' response and are cleared by the macrophages. However animal fats (ghee) are hydrolyzed into toxic fatty acids by lung lipases inducing a severe inflammatory necrotizing hemorrhagic reaction producing an interstitial proliferative granulomatous pneumonia.^{2,7}

Lipoid pneumonia was first described by Laughlin in a report published in 1925.⁹ The risk of developing lipoid pneumonia is higher in patients who are prone to get lung aspiration such as those with neurological disabilities. The clinical pattern of lipoid pneumonia in infants can be a chronic or acute pneumonic illness that is unresponsive to routine antimicrobial therapy. The clinical picture is usually less severe than the radiological picture would suggest.^{4,7}

The age of onset and clinical presentation of our patient were typical for a lower respiratory tract infection, which are also the

most common features in patients with lipoid pneumonia. These are cough, fever, tachypnea, respiratory distress with retractions, rales and failure to thrive.⁴ Despite treatment with adequate broad spectrum antibiotics as well as supportive therapy, there was no significant improvement of the patient which led us to seek other diagnoses.

The initial radiological findings are non-specific. The CT scan of the chest is reported to be helpful in differentiating lipoid pneumonia from other pneumonias, since fat has negative attenuation of the range of -150 to -60 Hounsfield units. However, in some patients the fat attenuation is not evident on CT scans because the values are averaged with the surrounding inflammatory exudates.^{4,10}

The BAL fluid may show lipid laden macrophages, and with clinical presentation as well as radiological correlation the diagnosis of lipoid pneumonia can be achieved.^{7,9} The first BAL fluid in our patient was not tested for lipid laden macrophages since we were not suspecting lipoid pneumonia. It was only after the lung biopsy had identified the diagnosis that a repeat BAL fluids obtained via endotracheal tube was positive for lipid laden macrophages.

In our case we got the history of ghee ingestion after the lung biopsy suggested exogenous lipoid pneumonia. The role of eliciting history of administration of ghee or other oil products must be emphasized as it gives an indication of the diagnosis and diagnostic tests that may be missed if such history is not obtained.

We treated our patient with oral steroid for a period of 3 weeks followed by tapering period of 9 weeks, and another 6 months of inhaled steroids. She had shown a clear and dramatic clinical response and complete radiological resolution of the pneumonia after many weeks of unsuccessful response to multiple intravenous broad-spectrum antibiotics and ICU care. Other authors have also reported the use of steroids with good response.^{4,7,11} We believe that steroids have a role in the treatment of exogenous lipoid pneumonia specially to those who are very sick.

Conclusion

Lipoid pneumonia secondary to aspiration of ghee or other oil products can be the cause of un-resolving or recurrent pneumonia in infants and young children. A strong clinical suspicion and direct question to care giver will help diagnose it early and direct to the right diagnostic tools and treatment.

Since the custom of giving ghee to newborns and infants is still common in some areas of Oman. There is a need to study the extent of this practice and to educate the community about the hazards of such practice.

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