Lipoid Pneumonia - An Unusual Cause of Acute Respiratory Distress Syndrome

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Background: Lipoid pneumonia is a rare form of pneumonia caused by aspiration of fatty substances. **Case characteristics**: Acute respiratory distress syndrome in an infant due to accidental aspiration of baby oil massage. **Intervention**: Large volume bronchoalveolar lavage. **Outcome**: Gradual recovery over a period of 5 months. **Message**: Aspiration of lipids cause prolonged and refractory hypoxemia.

Keywords: *Bronchoalveolar lavage, Fiberoptic bronchoscopy, Lipoid pneumonia.*

Exogenous lipoid pneumonia is an uncommon condition resulting from the aspiration or inhalation of fatty substances like mineral oils or petroleum jelly [1,2]. We report a child developing acute respiratory distress syndrome (ARDS) after accidental ingestion of baby massage oil. She was successfully treated with multiple bilateral large volume bronchoalveolar lavages (BAL) carried out with fiberoptic flexible bronchoscope.

CASE REPORT

A 10-month-old girl presented to us fifteen days after accidental ingestion of baby massage oil. Post-ingestion, she had vomited once, and developed progressive cough and dyspnea in the next three days. Her clinical condition worsened despite oral antibiotics and she was admitted elsewhere. In view of poor response to treatment, patient was referred to our hospital for further management.

At admission, she had severe respiratory distress and hypoxemia. Initial chest radiograph showed bilateral diffuse ground glass appearance suggestive of alveolar-interstitial pattern of opacities. She was electively intubated and ventilated with pressure regulated volume controlled mode. In view of persistent respiratory acidosis (pH 7.21), high plateau pressure (32 cmH_2O) and refractory hypoxemia (PaO_2/FiO_2 <150), she was shifted to high frequency oscillatory ventilation (HFO-V, Sensormedics 3100A). She required high mean airway pressure (27cm H_2O), FiO_2 0.7, amplitude 45, frequency 10Hz and inspiratory time 33%. In view of the hemodynamic instability, boluses of normal saline and dopamine infusion (10 mcg/kg/min) were used for 48 hours. Hypoxemia and respiratory acidosis improved gradually over next 72 hours, and she was shifted to conventional ventilation on day 4. The weaning trial failed on day 7 of admission and chest *X*-ray continued to show ground glass haziness of both lung fields. The sepsis screen sent at admission was negative. Endotracheal aspirate sent on admission was positive for lipid-laden macrophages; gram stain and culture were non-contributory.

On seventh day of admission, we performed fiberoptic flexible bronchoscopy through endotracheal tube. The tracheobronchial tree was smeared with pale yellow oily secretions. Large volume (50mL saline for each lung) Bronchoalveolar lavage (BAL) was done; it was positive for lipid-laden macrophages and the cultures were sterile. She was started on intravenous methylprednisolone (2 mg/kg/day)followed by its oral preparation for next 8 weeks. During her stay, contrast enhanced computed tomography of chest also showed bilateral ground glass haziness of both lungs, lower lobes more involved than upper lobes (***Fig.* 1**). Single lung large volume BAL was performed twice a week, while she was on invasive ventilation, followed by weekly BAL; the child was successfully extubated on day 11 of admission. She was electively weaned and extubated to non-invasive ventilation (BIPAP) with supplement oxygen. Over the next 4 months she was shifted from BIPAP to continuous positive airway pressure mode. She could be weaned from oxygen after 5 months of hospital stay. Patient developed clubbing of all fingers by end of 3 weeks of stay in the hospital.

She received oral prednisolone and azathioprine (2 mg/kg/day) for 9 months. Initially, prednisolone was started with 2 mg/kg/day and dose was gradually tapered. Six months after discharge, she had normal growth and development with reversal of clubbing and normal chest *X*-ray.

DISCUSSION

Acute exogenous lipoid pneumonia is due to acute ingestion of mineral, vegetable or animal fats [3]. Chronic exogenous lipid pneumonia usually results from repeated episodes of aspiration or inhalation of oils over an extended period. It is usually seen in children with anatomic (cleft palate) or functional defects [2]. The aspirated fat is phagocytosed by macrophages leading to destruction of alveoli. Most of the oil coalesces, forming large fat drops surrounded by fibrous tissue and giant cells, creating paraffinoma. The inflammatory response can destroy the alveolar walls and the interstitium, and the resultant fibrosis can occasionally progress to end-stage lung disease [2,3].

Acutely, patient typically presents with cough, dyspnea and low-grade fever. Crackles or rhonchi may be heard on auscultation of chest. Laboratory investigations reveal hypoxemia, leukocytosis and raised erythrocyte sedimentation rate [1,2]. Diagnosis is established on the basis of history of exposure with presence of lipid laden macrophages in respiratory samples such as BAL fluid or sputum [4]. Hadda, *et al*. [3] reported a case of a 20-year-old male who presented with acute respiratory distress syndrome following accidental mineral oil aspiration.

The key to treatment is identifying and discontinuing exposure to the offending agent, providing adequate supportive therapy and treating complications [1,2,5,6]. Use of azathioprine with steroids has been reported in pulmonary fibrosis patients to prevent progression of fibrosis [6,7]. Fibrosis is reported as an end result in lipoid pneumonia but azathioprine use for the treatment has not been earlier reported [1,2]. Steroids either alone or in combination with intravenous immunoglobulin and large volume bronchial lavage have been used with reasonable success [5,8-10].

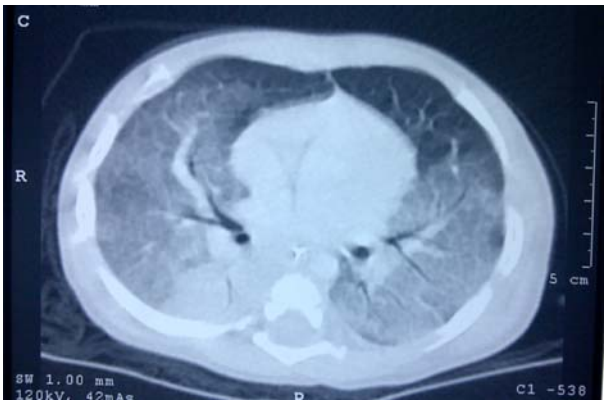

FIG. 1 *Contrast enhanced CT of chest (axial section in lung window) showing ground glass appearance, air trapping and consolidation.*

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