Case Report

"Diesel siphoner's lung": Exogenous lipoid pneumonia following hydrocarbon aspiration

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ABSTRACT

Lipoid pneumonia is an unusual and uncommon form of pneumonia caused by aspiration of fatty substances. Hydrocarbon pneumonitis following aspiration of diesel is a form of exogenous lipoid pneumonia wherein, aspirated diesel reaches the alveoli rapidly without evoking any significant cough, but initiates an intense inflammatory reaction in the pulmonary parenchyma. This is a rarely described clinical scenario, although the practice of diesel siphonage from automobiles is a common practice in developing countries. We herein describe a 40-year-old male patient, in whom the diagnosis of lipoid pneumonia was delayed for a long duration and highlight the importance of taking a detailed occupational exposure history in patients with non-resolving pneumonia to rule out the underlying possibility of this rare clinical entity.

KEY WORDS: Aspiration, bronchoscopy, lipoid pneumonia

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INTRODUCTION

Lipoid pneumonia is an uncommon form of pneumonia caused by aspiration of fatty substances. Though it has been recognized since 1925 when it was first reported by Laughlen,[1] unfamiliarity with this condition is a frequent cause of under diagnosis. The absence of specific clinico-radiological features makes diagnosis difficult unless a strong suspicion is held and a thorough history is obtained. Definitive diagnosis is achieved by the presence of intra-alveolar lipid and lipid laden macrophages on histopathology.

Although data on the precise incidence of lipoid pneumonia are lacking, its incidence has been reported to be 1.0-2.5% in autopsy studies.[2] The condition has been classified into exogenous and endogenous types based on the mode of lipid acquisition. The exogenous type is commoner and associated with aspiration of fatty substances whereas the endogenous type is usually secondary to bronchial obstruction or following chronic infections or due to errors of lipid metabolism.[3] A high index of suspicion and early diagnosis can help halt the progression of disease by avoiding further exposure to the offending agents and allow appropriate and timely management. Herein, we present the case of a middle aged gentleman in whom the diagnosis of lipoid pneumonia due to diesel aspiration was delayed. Establishment of a correct diagnosis led to the institution of appropriate management, which was followed by patient recovery.

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CASE REPORT

A 40-year-old male patient presented with a history of recurrent episodes of fever associated with cough and expectoration of 18 months duration. There was associated history of streaky hemoptysis on and off. There was no history of loss of weight, chest pain, wheezing or shortness of breath. Patient was a smoker with a smoking index of 300; however, no other significant personal or occupational exposure history was forthcoming. There was no past history of tuberculosis. He had been treated with multiple antibiotics over the past 1 year and empiric antitubercular therapy too had been administered, without any symptomatic relief. General physical examination was unremarkable. On chest examination, scattered crepitations were audible bilaterally. Rest of the systemic

examination was normal. Baseline laboratory parameters were within normal limits. There was no leukocytosis. Liver and renal function tests and arterial blood gases were within normal limits. Sputum cultures were negative for bacterial, mycobacterial or fungal growth.

Chest radiograph [Figure 1] demonstrated bilateral pulmonary infiltrates, more on the left lower zone and computed tomography (CT) of the thorax [Figure 2] showed multifocal areas of low attenuation consolidation predominantly in the left lower lobe. There was no mediastinal lymph node enlargement, pneumothorax or pleural/pericardial effusion. Flexible bronchoscopy was performed, which demonstrated normal bronchial anatomy with no endobronchial growth or abnormality. Examination of the bronchoalveolar lavage (BAL) fluid and bronchoscopic lung biopsy demonstrated lipid laden macrophages in the alveoli and interstitium, confirming the diagnosis of lipoid pneumonia [Figure 3]. On subsequent enquiry, patient recollected history of multiple episodes of aspiration while siphoning diesel out of cars, when he used to work as an automobile mechanic. He had left the job 2 years back. A final diagnosis of exogenous lipoid pneumonia due to recurrent diesel aspiration was made. Patient improved symptomatically after bronchoalveolar lavage and was advised to follow-up regularly.

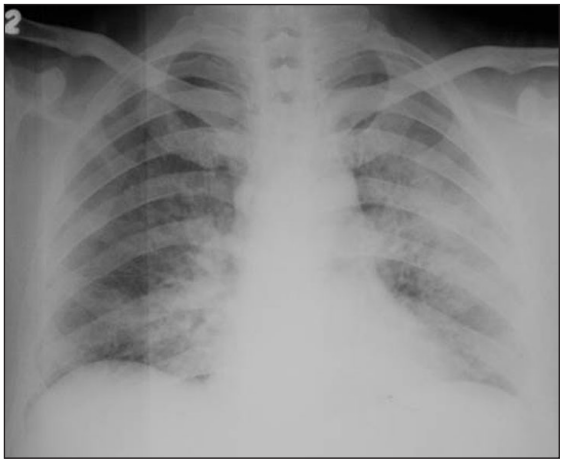

Figure 1: Posteroanterior chest radiograph demonstrating bilateral alveolar infiltrates, more in the left lower zone

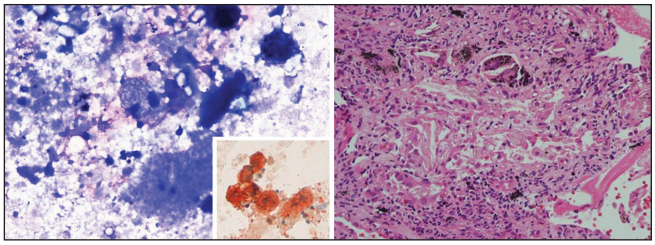

Figure 3: Cytopathological examination of the bronchoalveolar lavage fluid (left panel) showing the presence of foamy macrophages, which stain positive with fat stain (inset). Bronchoscopic lung biopsy (right panel) demonstrating giant cell reaction along with numerous foamy lipid laden macrophages

After 2 months, patient presented with recurrence of fever associated with cough and expectoration. There was no symptomatic relief with a course of broad spectrum antibiotics and chest radiograph showed new onset infiltrates in the right lower zone. BAL fluid analysis demonstrated lipid laden macrophages. Stain for acid fact bacilli or fungus was negative. In view of recurrent symptomatic deterioration and no evidence of uncontrolled infection, patient was initiated on treatment with oral prednisolone (0.5 mg/kg/day), which was tapered over 6 weeks duration. A follow-up chest radiograph [Figure 4] showed complete resolution of the radiographic abnormalities.

DISCUSSION

Lipoid pneumonia is an unusual cause of pneumonia and has been reported in various age groups in different clinical settings. Initial reports of exogenous lipoid pneumonia included children treated with mineral oil based nasal drops for the common cold and adults with oily laxative abuse.[1] Other reported risk factors include nasopharyngeal anatomic defects or neuromuscular weakness predisposing to aspiration, occupational exposure in fire eaters[4] and forceful feeding of animal fat in children with a view to regularize bowel habits.[5]

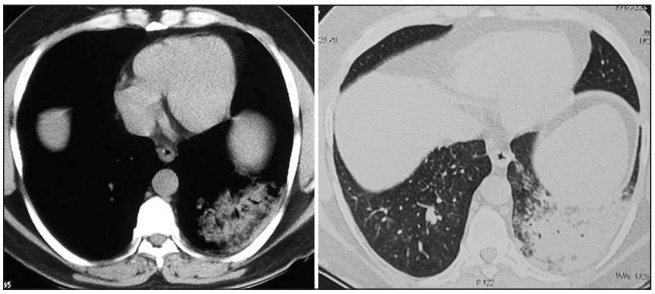

Figure 2: Computed tomography scan of the thorax demonstrating left lower lobe consolidation. Low-density areas are seen in the consolidated lung in the mediastinal window (left panel)

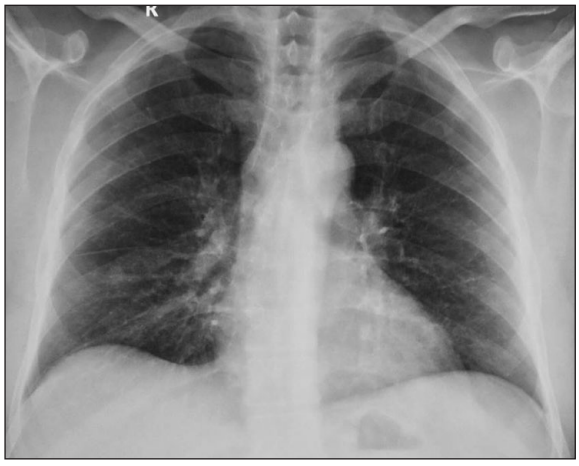

Figure 4: Chest radiograph demonstrating resolution of the pulmonary infiltrates subsequent to treatment with oral prednisolone

One of the common practices in developing countries is siphoning out mineral oil from automobiles as in our patient; however, hydrocarbon pneumonitis in this setting has been rarely reported.[6]

The pathophysiology of lipoid pneumonia is not completely understood. Aspirated oil particles are generally bland, non-irritating and do not stimulate the cough reflex pathway and thus reach the lower airways. Once in the airways they impair mucociliary clearance and this further impairs their expulsion. The lipid deposited cannot be metabolized due to lack of enzymes in humans and are emulsified and engulfed by alveolar macrophages, in which they can remain for a long time. They are released into the alveoli in a timely fashion following disruption of the macrophages, inciting a giant cell response leading to fibrosis and disruption of bronchial and alveolar structure.[7,8] Diesel is a complex hydrocarbon mixture produced from the fractional distillation of crude oil, to which various chemical additives are added.

Clinical presentation of lipoid pneumonia encompasses a spectrum of disorders ranging between chronic presentations such as insidious onset shortness of breath, fever, weight loss simulating chronic infections or interstitial lung disease, which follows chronic, recurrent, low dose exposure to the inciting agent[9] or an acute presentation with severe respiratory distress similar to acute respiratory distress syndrome usually following a massive exposure.[6] There have also been reports of cases simulating infectious pneumonia with fever, cough and pleuritic chest pain within a few hours following accidental aspiration of kerosene or diesel while siphoning.[10,11] Few cases of pleural effusions and empyema have also been reported as possible complications.[11] Though middle lobe is postulated to be the most common site of involvement owing to leaning forward position of the siphoners; in most of the case reports from the Indian subcontinent, lower lobe predominance can be noticed. One probable reason for the same might be the squatting posture assumed by patients while siphoning the mineral oils.[10] Our patient had a delayed presentation subsequent to the primary exposure, with no ongoing exposure to the offending agent. Radiologically lipoid pneumonia may manifest as alveolar consolidation, ground-glass opacities and airspace nodules. Rarely crazy paving pattern, pneumatoceles and fat containing masses (paraffinomas) have been reported. The most characteristic finding on CT is the presence of a low-density consolidation (−30 to −150 HU) which was seen in our patient.[12] The definitive diagnosis is made by demonstrating lipid laden macrophages in BAL fluid and in the alveoli or interstitium in bronchoscopic lung biopsy.[13] Treatment of lipoid pneumonia is not well-established and experience is from anecdotal case reports. Avoiding further exposure to the offending agent is the most important treatment option for exogenous lipoid pneumonia. Several authors have shown the utility of corticosteroids in slowing the inflammation.[14] Steroid therapy limits the inflammatory response and ongoing fibrosis. In a series of pediatric patients with lipoid pneumonia following nasal instillation of olive oil, administration of prednisolone was followed by complete clinical and radiological recovery.[15] Repeated whole lung lavage has been reported to be useful.[16] Use of fat emulsifiers like 0.05% polysorbate 80 in Ringer's lactate for lung lavage has also been reported.[17]

Lipid laden macrophages may remain dormant for a long duration and release the lipid again into the alveoli when they lyse thus inciting an inflammatory reaction. This is the likely mechanism for a delayed presentation in our patient.

CONCLUSION

Lipoid pneumonia should always be considered in the list of differential diagnoses while managing a patient with non-resolving pneumonia. A high index of suspicion is required to diagnose the condition as the history of exposure is not always forthcoming especially in chronic cases. Physicians should be aware of the clinico-radiological profile of this entity in order to avoid diagnostic delay. This can prevent prolonged and unnecessary use of antimicrobial therapy and prevention of exposure can lead to an asymptomatic recovery in many cases. In our opinion, it is important to create awareness regarding the possible serious adverse events associated with the practice of mineral oil siphonage and this practice should be abandoned.

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