Volume : 2 | Issue : 9 | Sept 2013 • ISSN No 2277 - 8160

Research Paper | Medical Science

Hydrocarbon Pneumonia Following Diesel Siphonage

Adesh Kumar	Associate Professor Department of Pulmonary Medicine, Rural Institute of Medical Sciences & Research Safai, Etawa U.P.India.
Ashish Kumar Gupta	Senior Resident Department of Pulmonary Medicine, Rural Institute of Medical Sciences & Research Safai, Etawa U.P.India.
Mohd Haroon Khan	Assistant Professor Department of Community Medicine, Rohilkhand Medical College, Bareilly,(U.P). India.

ABSTRACT *In our country, siphoning of diesel/petrol from fuel tanks is a common practice. We describe a 60-year-old farmer, who accidentally aspirated fuel while siphoning from his generator. A diagnosis of diesel induced aspiration pneumonia was confirmed by the presence of foam cells on bronchial biopsy. The patient showed gradual recovery with the symptomatic therapy. Diesel aspiration leading to bilateral pneumonitis is yet to be reported in our country.*

KEYWORDS: Pneumonia, diesal, bronchoscopy, HRCT thorax

Introduction-

Lipoid pneumonia is an unusual cause of respiratory symptoms and has been reported in all age groups. Initial reports of LP predominantly included children – often with local anatomic defects like cleft palate or debilitated adults, but several reports indicate that it can occur in healthy individuals as well [1,2]. There are reports of LP associated with aspirating or inhaling mineral oil, oil based laxatives, lip balm, lip glow etc [2,3]. In our country siphoning of various mineral oils (like diesel) from containers is a common practice and may be an important risk factor for LP. Other risk factors described in infants and small children are use of traditional folk remedies like, use of oily nasal drops, forceful animal fats feeding, such as "ghee", to establish regular bowel habits or transnasal use to treat cough and cold [4,5]. This entity does not have characteristic clinical or radiological features. Therefore, awareness of this type of pneumonia is important as diagnosis requires a high degree of clinical suspicion. It should be considered in the differential diagnoses of any acute or chronic respiratory condition in cases with history of exposure to fatty substances. The diagnosis of acute LP, in absence of history of exposure tofatty substances, may be very difficult. However, early diagnosis and timely treatment is important for better outcome. In suspected cases, diagnosis can be established by demonstrating lipid laden macrophages in various specimens like sputum, bronchoalveolar lavage, lung biopsy [6,7].

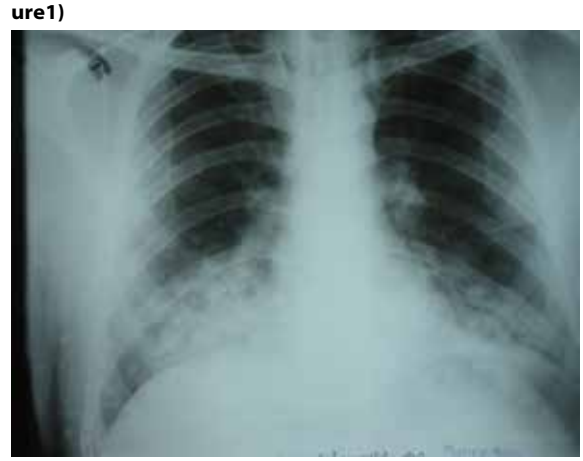
Case summary-

A 60-year-old farmer was referred to our Institute with a history of accidental diesel aspiration while siphoning from the fuel tank of GENERATOR, which occurred SEVEN days prior to presentation. The patient complained of progressively increasing breathlessness at rest without wheeze, along with mild, non-radiating pain on BI-LATERAL LOWER chest. He developed a productive cough with frothy expectoration that was non-foul smelling. The patient had nausea but there was no history of hemoptysis, hematemesis, dysphagia or hoarseness of voice. He had received symptomatic treatment at a private hospital for three days without relief.

On examination, the patient was febrile with a pulse rate of 110/MIN, regular, good volume with a blood pressure of 130/78mmHg. He was dyspnoeic with a respiratory rate of 28/min. There was no cyanosis or pedal edema and JVP was not raised. Respiratory movements were equal on both sides of the chest. However, the percussion note was impaired bilaterally in the inframammary, infraxillary and infrascapular areas. On auscultation, vesicular breath sounds, though audible, were greatly diminished in intensity in these areas along with coarse, basal, inspiratory crepitations.

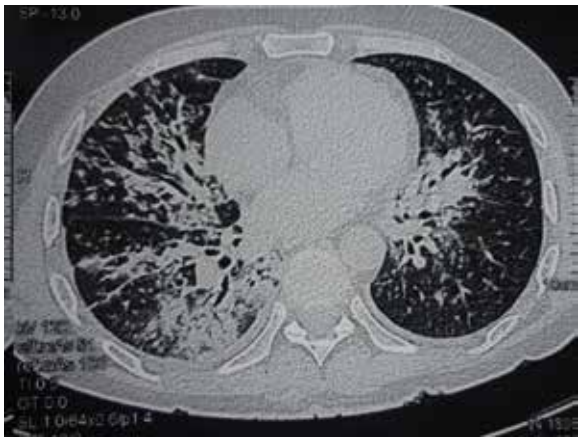
The haemogram revealed a total leucocyte count of 3200 cells/mm3 with 57% polymorphs and 36% lymphocytes. A chest roentgenogram revealed bilateral, non-segmental, homogeneous opacities in the lower zones (Figure 1). Blood gas analysis showed a pH of 7.477, pCO2 41 mm, and pO2 45mm with bicarbonates of 29.6 mmol. oxygen saturation (SaO2) of 92%. Blood urea (56 mg/dl) and creatinine (1.75 mg/dl) were normal. Liver function tests showed normal bilirubin and mild hepatitis with SGOT and SGPT of 70 and 70iu, respectively. ECG was within normal limits. A provisional diagnosis of aspiration pneumonitis was made and the patient was treated with oxygen inhalation, analgesics and levoflxacin500mg daily with interavenous amoxicillin-clavalunic acid1.2 gm thrice daily along with interavenous hydrocortisone 200mg thrice daily.

Chest xray shows bi-lateral lower zone consolidation(figure1)



(figure-1 Chest radiograph showing bilateral patchy opacities)

Patient is subjected to Computer Tomography (CT scan) of chest which revealed consolidation in the right middle lobe and lower lobe and also left lower lobe.(figure 2)



Patient underwent fiberoptic bronchoscopy(FOB) with bronchoalveolar lavage(BAL) and transbronchial lung biopsy from right lower lobe. fob showed normal airways.staining and culture for mycobacteria and fungi were negative.BAL fluid cytology showed multiple lipid laden macrophage.

DISCUSSION-

Exposure to hydrocarbons can happen in many forms, but it causes the most destruction with aspiration [8].Siphonage of fuel from the motor vehicles is a very common practice in our country. Accidental aspiration of diesel/petrol during siphoning can result as a result of direct inhalation or may follow ingestion(9). Symptomatic involvement of the pulmonary system occurs in the form of pulmonary oedema, atelectasis and consolidation, and is often the most common presentation of hydrocarbon toxicity(10) The toxic potential of hydrocarbons is directly related to their physical properties. Highly volatile with low viscosity and lower surface tension hydrocarbons are more likely to be inhaled or aspirated into the respiratory system [8]. The hydrophobic nature of them let them to penetrate deep into the tracheobronchial tree. Direct contact with alveolar membranes can lead to hemorrhage, hyperemia and edema. They provoke the activation of macrophages, leading to an increased release of cytokines and a prolonged inflammatory reaction [11]. The type II pneumocytes are most affected, resulting in a decreased surfactant production. The surfactant layer, which is composed of lipids, is made soluble by hydrocarbons, causing further damage [12]. The end results of hydrocarbon aspiration are necrosing chemical pneumonitis, atelectasis, pleural effusion and hypoxemia [13]. The radiological lesions are generally out of proportion to the clinical findings. Radiographic findings include unilateral or bilateral lung consolidation, pneumatoceles, pleural effusion, atelectasis and spontaneous pneumothorax [14]. Hydrocarbon pneumonia is a pseudo-infectious lung disease with a severe release of inflammatory cytokines with macrophage activation. The inflammatory nature of the disease suggests that steroid treatment can be useful in these patients. However there have been conflicting reports about the effectiveness of steroids [15]. Our patient had significantly improved by steroid treatment. The conflicting reports on the use of steroids could be due to different intensity of exposures and complications of pneumonitis such as microbial superinfection and acute respiratory failure. Prophylactic antibiotic therapy is usually applied in these cases. A prophylactic antibiotic therapy was also begun in our patient since he was admitted 7 days after the event. Because pathogens were not detected in bronchial samples we discontinued antibiotic treatment. The prognosis is generally good in hydrocarbon aspiration and our patient completely improved after 15 days of treatment. However some studies reported that some complications may develop, which included abscess, bronchopulmonary fistula, bacterial superinfection and acute respiratory failure and death.

REFERENCES

1. Annobil SH, Benjamin B, Kameswaran M, Khan AR: Lipoid pneumonia in children following aspiration of animal fat (ghee). Ann Trop Paediatr 1991, 11(1):87-94. | 2. Burkhardt O, Merker HJ, Shakibaei M, Lode H. Electron microscopic findings in BAL of fire-eater after petroleum aspiration. Chest 2003;124(1):398-400 | 3. Dyer S. Hydrocarbons. In: W.olfson AB, Editor. Harwood-Nuss' Clinical Practice of Emergency Medicine. Philadelphila USA: Lippincott Williams & Wilkins; 2005. p. 1590-3. | 4. Franquet T, Gómez-Santos D, Giménez A, Torrubia S, Monill JM. Fire eater's pneu-monia: radiographic and CT findings. Comput Assist Tomogr 2000;24(3):448–50 | 5. Ferretti GR, Jankowski A, Rodiere M, Brichon PY, Brambilla C, Lantuejoul S: CT-guided biopsy of nonresolving focal air space consolidation. J Thorac Imaging 2008, 23(1):7-12. | 6. Gondouin A, Manzoni P, Ranfaing E, Brun J, Cadranel J, Sadoun D, Cordier JF, Depierre A, Dalphin JC: Exogenous lipid pneumonia: a retrospective multicentre study of 44 cases in France. Eur Respir J 1996, 9(7):1463-1469 | 7. Giammona ST. Effects of furniture polish on pulmonary surfactant. Am J Dis Child 1967;113(6):658-63. | 8. Hugosson CO, Riff EJ, Moore CC, Akhtar M, Tufenkeji HT: Lipoid pneumonia in infants: a radiological-pathological study. Pediatr Radiol 1991, 21(3):193-197. | 9. Levade T, Salvayre R, Dongay G, Dang QQ, Vieu C, Bessac A, Douste-Blazy L: Chemical analysis of the bronchoalveolar washing fluid in the diagnosis of liquid paraffin pneumonia. J Clin Chem Clin Biochem 1987,25(1):45-48. | 10. Meltzer E, Guranda L, Perelman M, Krupsky M, Vassilenko L, Sidi Y: Lipoid pneumonia: a preventable form of drug-induced lung injury. Eur J Intern Med 2005, 16(8):615-617. | 11. Olsen KR. Poisoning. In: Tierney LM, McPhee SJ, Papadakis MA, eds Current Medical Diagnosis and Treatment; 39th edn; International edition; McGraw-Hill Company; 2000: 1567-68 | 12. Rouse TE, Weese WC, Kazemi H. Gasoline ingestion. New Engl J Med 1974; 290 : 1092-93. | 13. Salm R, Hughes EW: A case of chronic paraffin pneumonitis. Thorax 1970, 25(6):762-768 | 14. Steele RW, Conklin RH, Mark HM. Corticosteroids and antibiotics for the treat¬ment of fulminant hydrocarbon aspiration. JAMA 1972; 219(11):1434-7. |