# Chemical pneumonitis following diesel oil siphonage: A case report and review of literature

**Sujeet Raina[1,*], Ansari Mohd Shoeb[2], Rajesh Sharma[3], Amandeep Raghuvanshi[4]**

[1]Assistant Professor, [2]PG Student, [3]Professor, [4]Senior Resident, Dept. of Medicine, Dr. RPGMC, Kangra, Himachal Pradesh

***Corresponding Author:**
Email: sujeetraina@gmail.com

## Abstract

Reports of diesel aspiration followed by chemical pneumonitis and its management have rarely been published. Details of clinical features, imaging findings, appropriate management and outcome of this condition are available in literature in the form of individual case reports. We present a case of diesel aspiration induced chemical pneumonitis that was successfully treated in our medicine ward.

**Keywords:** Aspiration; Chemical; Diesel; Pneumonia

## Introduction

Manual siphonage of fuel from the motor vehicles fuel tanks is a common practice in Asia including India and Middle East[1,2]. Diesel aspiration leads to a direct insult to the broncho-alveloar endothelium and initiates an intense inflammatory reaction. Clinico-radiological features are nonspecific[1]. The clinical spectrum varies from being asymptomatic to chemical pneumonitis and its complications. The exact prevalence of the entity is unknown, but may be more common than reported. Large data on clinical features, imaging findings, appropriate management and outcome of this condition is lacking. No treatment based randomized control trial have been performed as the numbers of cases are less. We report a patient who developed chemical pneumonitis following manual siphonage of diesel and was successfully treated. The literature on this clinical condition is also reviewed. Data on precise incidence of chemical pneumonitis from India are lacking.

## Case Report

A 42 year old male auto mechanic by profession attended the emergency at 6.30pm with the history of accidental diesel oil aspiration during siphonage of motor vehicle at around 11 am on the same day while he was working in his garage. He presented with the chief complaints of difficulty in breathing, retrosternal burning sensation and chest pain which started immediately after the aspiration. There was no history of wheeze, fever, cough, hemoptysis and altered sensorium. No significant past history was present. On examination patient was fully conscious and hemodynamically stable. His pulse rate was 94/minute; and blood pressure was 126/64 mmHg; respiratory rate was 25/minute and SpO2 was 94% at room air. There was no cyanosis. Chest wall tenderness was present. On chest auscultation fine inspiratory crackles were audible in infrascapular area bilaterally. Rest of the examination was unremarkable. Baseline hemogram, renal and liver function tests were normal. ABG analysis and electrocardiogram were normal. X-ray chest PA view revealed bilateral lower zone patchy opacities (Fig. 1A). The diagnosis of chemical pneumonitis was made and patient was started on injection hydrocortisone; injection ceftriaxone and injection diclofenac sodium. Patient developed fever and hemoptysis during hospital stay which persisted till day five of admission. The chest pain and tenderness continued till day seven of admission. Contrast enhanced computed tomography of chest was done which revealed bilateral symmetrical areas of ill-defined nodules with tree in bud pattern and patchy area of consolidation and collapse in bilateral upper lobe, lingular lobe and right middle lobe. Total leucocyte count increased from 9000/cmm at baseline to 15000/cmm on day seven and 22,000 on day nine. Radiological extension of lesions on chest X-ray was observed on seventh day of admission (Fig. 1B). Sputum and blood cultures were sterile. Piperacillin tazobactam replaced ceftriaxone on seventh day. Patient recovered and was discharged after two weeks of admission. Radiological clearance was observed on discharge (Fig. 1C).

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

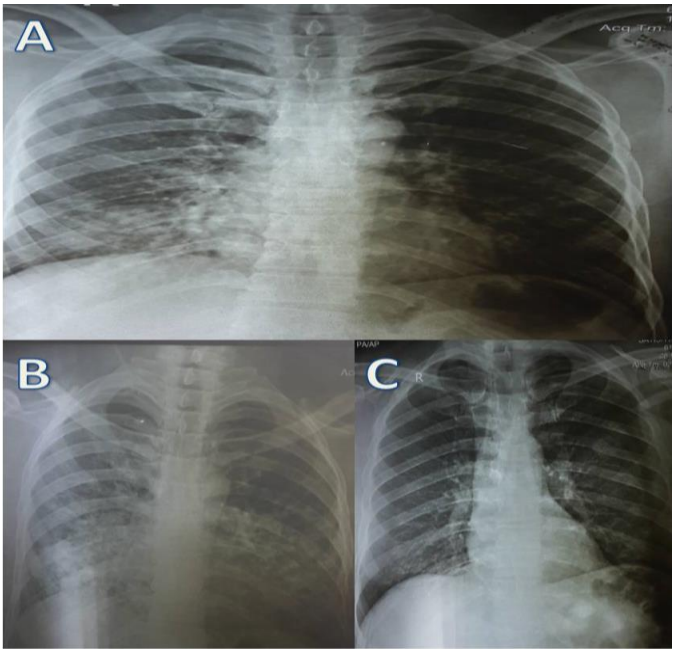


**Fig. 1: Chest X-ray PA view of patient A) taken on admission day showing lower zone opacities B) Day 7 post aspiration showing further increase in the alveolar opacities C) At discharge showing clearance**

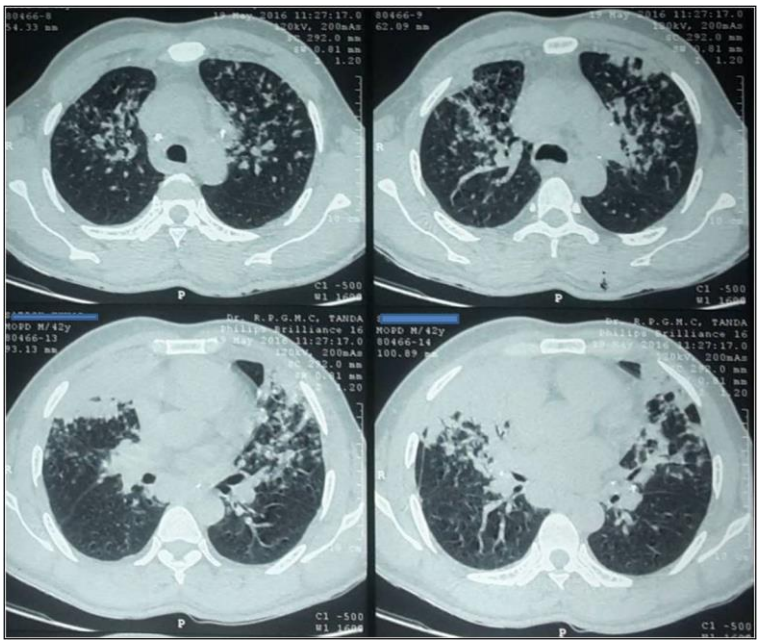

**Fig. 2: Axial CT (lung window) showing multiple ill-defined nodular densities, ground glass opacities with patchy areas of consolidation and tree in bud pattern**

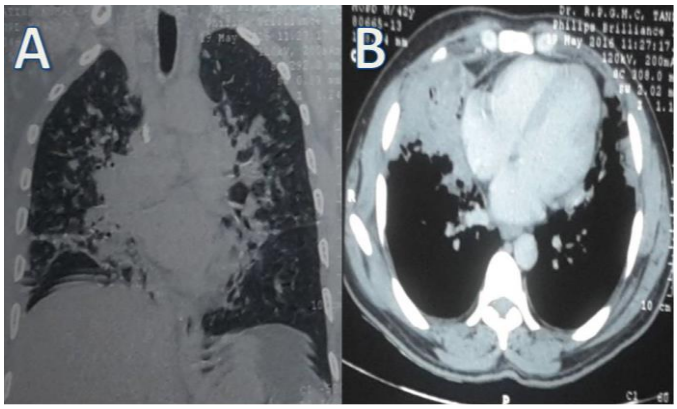


**Fig. 3: A) Coronal CT (lung window) B) axial CT(mediastinal window) showing multiple ill- defined nodular densities, ground glass opacities with patchy areas of consolidation**

## Discussion

Chemical pneumonitis is caused by a direct insult to the broncho-alveolar endothelium by the exposure to a foreign substance, solid particles or liquid. Petroleum product like diesel and petrol, used as fuel in transport vehicles are hydrocarbons. Hydrocarbon aspiration can lead to chemical pneumonitis (lipiodal or hydrocarbon pneumonitis) following the manual siphoning from fuel tanks. Highly volatile with low viscosity and lower surface tension hydrocarbons are more likely to be inhaled or aspirated into the respiratory system.[3]Diesel is a complex mixture of liquid hydrocarbons produced from the fractional distillation of crude oil, to which various chemical additives like detergents, smoke suppressants, flow improvers etc. are added.[4]Diesel oil has a low viscosity, is highly volatile, insoluble in water and a potential hydrocarbon to cause severe progressive lung tissue inflammation. Aspirated hydrocarbons like diesel are non- irritating, hydrophobic and are not absorbed thus reach broncho-alveolar spaces without evoking cough reflex. In the lower airways they impair mucociliary clearance which further diminishes their expulsion. The lipid deposited cannot be metabolized due to lack of enzymes in humans. Direct contact in the alveolar membranes induces edema, haemorrhage and decreased surfactant production. Macrophages get activated and inflammatory cytokines are released resulting in an inflammatory reaction. The activated macrophages phagocytose the emulsified lipid in the alveoli which may remain for a long time. In addition to their role in pathogenesis, the detection of lipid containing cells or foamy cells help in establishing diagnosis.[5,6] The clinical manifestations are nonspecific and differ among patients. The spectrum varies from asymptomatic focal inflammatory reaction with few or no radiologic abnormalities to an acute illness resembling infectious pneumonia or acute respiratory distress syndrome (massive exposure) and chronic respiratory disease (chronic, recurrent, low dose exposure). Patients usually present with features of acute chemical pneumonitis in the form of fever, cough, chest pain, hemoptysis and dyspnoea within few hours following accidental aspiration of diesel while siphoning. Chest pain may be severe. Tenderness of chest wall is observed which could be related to chemical pleuritis.[3] Later, these patients may develop cardiomyopathies or fatal cardiac arrhythmias.[1,7] 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.[8]

The clinical history is crucial in early recognition of aspiration pneumonia. The nature of the aspirated material, the quantity of aspirated material, and the time course of the event influence the size and distribution of the lung parenchymal abnormalities.More commonly affected parts of lungs includes right middle lobe; however in most of the case reports from the Indian subcontinent, lower lobe predominance can be noticed.[9]

Radiologic manifestations are nonspecific. They are unilateral or bilateral consolidation, ground glass opacities and airspace nodules. Rarely crazy paving pattern, pneumatoceles and fat containing masses (paraffinomas) have been reported. Pulmonary abscess, cavity, Pleural effusion, atelectasis, pneumothorax, broncho-pleural fistulae may be observed.[2] The most characteristic finding on CT is the presence of a low density consolidation/areas of fat attenuation (−30 to −150 HU). Resolution of radiologic opacities following clinical recovery usually occurs between two weeks to eight months.[10] The definitive diagnosis is made by demonstrating lipid laden macrophages in BAL fluid and in the alveoli or interstitium in bronchoscopic lung biopsy.[11]

Management is not well-established and experience is from anecdotal case reports.[1] Avoiding further exposure to the offending agent is the most important treatment option. Role of steroid in treatment is rational as the illness is of inflammatory nature. They help in limiting the inflammation thus prevent fibrosis. However there have been conflicting reports about the effectiveness of steroids. The conflicting reports on the use of steroids could be due to different intensity of

exposures and complications of pneumonitis such as microbial super infection and acute respiratory failure. Prophylactic antibiotic therapy is usually applied in these cases.[4]

Primary prevention of chemical pneumonitis due to manual siphonage of fuel from the motor vehicles is difficult in India because of the widespread use of this practice by auto mechanics. There is a need to educate people regarding this habit and its terrible consequences. Relative sparing of lower lobes was observed on CT chest in our case as compared to lower lobe predominance observed in most of the case reports from India.

**Acknowledgements:** None

**Conflicts of interest:** None declared

## References

1. Khanna P, Devgan SC, Arora VK, Shah A. Hydrocarbon pneumonitis following diesel siphonage. Indian J Chest Dis Allied Sci. 2004;46:129-32.
2. Koc I, Dokme A. Pneumonia due to diesel fuel aspiration: A case report. Gaziantep Med J. 2015;21:62-4.
3. Sertogullarindan B, Bora A, Sayır F, Ozbay B. Hydrocarbon pneumonitis; Clinical and radiological variability. J Clin Anal Med. 2015;6:117-19.
4. Gowrinath K, Shanthi V, Sujatha V, Murali Mohan KV. Pneumonitis following diesel oil siphonage. Respir Med Case Rep. 2012;5:9-11.
5. Franquet T, Gomez-Santos D, Gimenez A, Torrubia S, Monill JM. Fire eater's pneumonia: radiographic and CT findings. J Comput Tomogr. 2000;24:448e50.
6. Grossi E, Crisanti E, Poletti G, Poletti V. Fire eater's pneumonitis. Monaldi Arch Chest Dis. 2006;65:59e61.
7. Kapoor D, Goel N, Singh M, Singh J. A rare case of fatal acute respiratory distress syndrome following diesel oil siphonage. Indian J Anaesth. 2013;57:625-27.
8. Venkatnarayan K, Madan K, Walia R, Kumar J, Jain D, Guleria R. "Diesel siphoner's lung": Exogenous lipoid pneumonia following hydrocarbon aspiration. Lung India. 2014;31:63-6.
9. Shrivastava MS, Palkar AV, Karnik ND. Hydrocarbon pneumonitis masquerading as acute lung injury. BMJ Case Rep. 2011; doi:10.1136/bcr.03.2011.4017.
10. Kitchen JM, O'Brien DE, McLaghlin AM. Perils of fire eating: an acute form of lipoid pneumonia or fire eater's lung. Thorax. 2008;63:409.
11. Frieman DG, Engelberg H, Merrit WH. Oil aspiration (Lipoid) pneumonia in adults. Arch Intern Med. 1940;66:11-38.