Diesel Aspiration Induced Chemical Pneumonitis : A Case Report

**KEYWORDS**

Chemical pneumonitis, diesel aspiration, aspiration pneumonia

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**ABSTRACT**

Chemical pneumonitis is caused by a direct chemical insult due to the entry of a foreign substance, solid or liquid, into the respiratory tract. The clinical and radiologic manifestations are from asymptomatic focal inflammatory reaction with few or no radiologic abnormalities to severe complications. The clinical history is important in diagnosing 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. The most common predisposing factors for aspiration in adults are alcoholism, stroke, seizures, loss of consciousness and neuromuscular disorders. Chest radiography is readily available and inexpensive as the primary imaging modality and also for monitoring. Sonography may sometimes be useful to detect pleural fluid and underlying lung consolidation. CT is the modality of choice to resolve details.

**Introduction:**

Chemical pneumonitis is caused by a direct chemical insult to the broncho-alveolar endothelium by the entry of a foreign substance, solid particles or liquid, into the respiratory tract. The clinical and radiologic manifestations may range from asymptomatic focal inflammatory reaction with few or no radiologic abnormalities to severe, sometimes fatal, complications. The clinical history is crucial in diagnosing 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. Reports of diesel aspiration followed by chemical pneumonitis and its management have rarely been published. We present a case of severe diesel induced chemical pneumonitis that was successfully treated in our pulmonary care unit (PCU).

**Predisposing factors:**

- alcohol intoxication
- general anesthesia
- unconsciousness/coma
- structural abnormality of larynx/pharynx/oesophagus
- neuromuscular/swallowing disorders

Primary Imaging examination: Chest radiography is readily available and inexpensive. Postero-anterior and lateral views are the commonly used imaging modality to diagnose, evaluate and monitor aspiration pneumonia and its complications. Ultrasound is useful for monitoring pleural effusion with collapsed-consolidated basal segments. Computed tomography (CT) is useful for resolution of details of pulmonary architecture and differentiation of pleural from pulmonary collections. Unlike radiography, CT is capable of differentiating fluid from solid tissue and detection of hyperemia/inflammation by contrast enhancement; thus it is ideal for displaying detailed features of aspiration pneumonia – consolidation, broncho-alveolar exudates, encysted fluid collections, abscesses, pleural effusion or empyema. CT precisely delineates the lobar/segmental location of the lesion. A foreign body in the tracheo-bronchial tree and associated atelectasis/consolidation can be easily defined with a CT scan.

**Clinical presentation:**

Patients with chemical pneumonia may present with an abrupt development of symptoms within a few minutes or as a gradual, insidious onset up to 2 hours after the aspiration event. It may start with mild dyspnea, rapid breathing, audible wheezing, and cough with pink or frothy sputum. Diesel being relatively heavy and inert easily settles in the basal parts of lung, particularly right middle lobe, on aspiration in upright posture and produces basal consolidation/exudates. The clinical course in case of diesel aspiration is comparatively slow and indolent.

Findings on physical examination may include tachypnea, tachycardia, fever, respiratory rales, wheezing, and occasionally cyanosis.

**Case Report:**

A 55 years male worker in an automobile workshop was trying to siphon off diesel from a container by sucking through a tube. There was accidental aspiration into the lung, following which the patient developed breathlessness, cough, nausea and vomiting.

On clinical examination there was tachypnea, tachycardia and reduced air entry into the right lung on auscultation. Chest radiograph revealed confluent alveolar opacities in right mid and lower zones silhouetting the cardiac and diaphragmatic outlines, suggestive of predominantly right middle & lower lobe consolidation resulting from chemical pneumonitis. A few patchy opacities were also noted in the left lower zone.
On ultrasound scanning there was a moderate amount of right pleural effusion with underlying basal lung consolidation. Left lung was nearly normal; no pleural effusion was seen on the left. A paracentesis of the right pleural fluid was done, for diagnostic purposes. This was exudative and revealed no organism.

CT scan done 2 days after diesel aspiration revealed features of chemical pneumonitis in the form of focal ground glass opacities and multiloculated collections involving both the lungs, the right lung more than left, along with effusion in the right pleural cavity with loculated extension into the minor fissure.

Figure (2): CT Scanogram showing confluent alveolar opacities in right mid and lower zones silhouetting right cardiac border suggestive of consolidation with compensatory hyperinflation of left lung.

Figure (3), (4), (5): Axial and coronal CT (lung window) showing multiple nodular densities, ground glass opacities, septal thickening and loculated collections in both lungs.
interval showing gradual resolution of the chemical pneumonitis in the form of reduction in the size of the alveolar opacities and consolidation in right mid and lower zone and disappearance of left side opacities.

Fig (10): Chest radiograph erect on day 3 after aspiration showing well defined loculated collection and confluent alveolar opacities in right mid and lower zone silhouetting cardiac and diaphragmatic outlines on right.

Fig (11): Chest radiograph erect on day 5 after aspiration showing reduction in the alveolar opacities and consolidation in right mid and lower zone.

Fig (12): Chest radiograph taken on day 10 post aspiration showing further reduction in the alveolar opacities.
and consolidation with clearance of right pleural effusion.

Management:
The patient was admitted to the pulmonary care unit and treated with high concentrations of oxygen and frequent nebulization with bronchodilators. Antibiotic coverage and NSAIDs were given to reduce the infection and inflammation. Follow up chest radiographs were obtained every 5 and 10 days.

The patient's clinical condition improved slowly. Serial chest radiographs showed reduction in the consolidations and fluid collections.

Discussion:
4 Chemical pneumonitis has been reported after exposure to a variety of chemicals and respiratory irritants. Diesel aspiration causes injury to bronchial and alveolar epithelial cells directly from tissue asphyxiation or from cytokine or cellular mediated inflammatory response. The epithelial cell injury causes reduction of mucociliary clearance of inhaled particulates, as also leakage of fluid between intercellular junction. The sloughing of dead epithelium leads to airway obstruction. Airway edema results as much from epithelial junction leak as it does from inflammatory response to cytokines and mediators released in response to cellular injury. The inflammatory mediators cause inflammation, edema, and airway smooth muscle contraction which interferes with clearance of the chemical. 6 Adult respiratory distress syndrome (ARDS) may result from a high level of exposure to an irritant liquid, gas, smoke, fume or vapour either at home, at the workplace or in the general environment. The frequency of developing ARDS after an inhalational exposure to an irritant substance has been difficult to quantify because patient-specific information on the magnitude and duration of exposure at the time of an inhalational accident is not often available.

Petroleum diesel is a complex mixture of liquid hydrocarbons. The practice of manual siphoning of diesel from fuel tanks is common in developing countries but hydrocarbon pneumonitis due to diesel siphonage is rarely reported.

After aspiration, hydrocarbons do not get absorbed in the airways and reach alveoli rapidly without evoking cough. In alveoli, they induce oedema, tissue damage and surfactant destruction. The pathologic changes result from inflammatory reaction due to activation of macrophages and release of inflammatory cytokines. The symptoms of acute hydrocarbon pneumonitis are non-specific. The typical clinical manifestations of acute exogenous chemical pneumonia include breathlessness, cough and low grade fever which usually resolve with supportive treatment. In our case chest pain and breathlessness were the predominant respiratory symptoms. The activated macrophages phagocytose the emulsified lipid in the alveoli and detection of these lipid containing cells or foamy cells through appropriate staining techniques is diagnostic of lipoid pneumonia. In hydrocarbon pneumonitis, bronchoscopy is useful for obtaining specimens and for suctioning the irritant from the site and also viewing the inflammatory changes. In our case, bronchoscopy was not performed.

Chest CT features of hydrocarbon pneumonitis after diesel aspiration/ingestion is rarely documented and most cases show bilateral necrotic air-space consolidations predominantly involving the right middle lobe. HRCT of chest is the imaging technique of choice as it may show typical appearances of exogenous lipoid pneumonia like consolidation of low attenuation with ‘crazy paving’ pattern. In our patient, the HRCT of chest showed areas of ground glass appearance, bilateral patchy consolidation, septal thickening and nodular densities predominantly involving the lingula and right middle lobe. Resolution of radiologic opacities following clinical recovery usually occurs between 10 days to 3 weeks. We used antibiotic and bronchodilator and high concentrations of oxygen for treatment.

REFERENCE