

Case Report :

Severe Chemical Pneumonitis Following Accidental Ingestion Of Diesel

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Received: Nov, 12, 2017; **Reviewed:** Dec 11, 2017; **Accepted:** Dec 29, 2017

Abstract:

Accidental Hydrocarbon fluid ingestion is common in children. We report a case of accidental ingestion of motor oil fuel - diesel by a previously healthy 3-year-old female child resulting in bilateral chemical pneumonitis. Extensive pneumonic air space consolidation in all lobes of both lungs were seen only through computed tomography of the chest. Patient received antibiotics, bronchodilators, steroids and symptomatic and supportive care without any mechanical ventilator support. After 11 days patient's symptom had subsided and discharged. On follow up after 2 weeks there was marked resolution of the initial symptoms.

Keywords: *diesel ingestion,*

Introduction:

Diesel is commonly used as a motor fuel and also the most common used for fuel operated generator. Sometimes, these fuel are kept in water containers and has led to its accidental ingestion by children who often thought is water. Accidental ingestion of hydrocarbon is common problem in children leading to aspiration of the fluid. Aspiration of diesel can lead to a direct insult to the broncho-alveolar endothelium and initiates an intense inflammatory reaction. Clinico-radiological features are nonspecific. The clinical and radiologic manifestations may range from asymptomatic focal inflammatory reaction with

few or no radiologic abnormalities to severe, sometimes fatal, 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. Reports of diesel aspiration followed by chemical pneumonitis and its management have rarely been published. We present one such case of severe diesel induced chemical pneumonitis treated successfully at our centre.

Case Report:

A 3 year old female patient had accidentally consumed a small amount of diesel oil inadvertently which was left around by the grandparent in a glass. She had been hospitalized in critical care 20 days ago and treated with antibiotics. She was discharged from the previous hospital as she clinically stable. However, She was brought to our hospital with complaints of shortness of breath since 1 day. The child had a history of NICU stay for 5 days as she developed cyanosis 4 hours after the delivery. She had motor developmental delay. On physical examination, it was determined that weight and height was below 25th percentile, temp: 98.7 °C, pulse: 114 beats/min., respiratory rate: 56/min, SaO₂ : 96%, there were subcostal and intercostal retractions, on auscultation respiratory sounds were equal on both the sides with no adventitious sounds but patient had bronchial breathing; CNS examination showed

mild muscle wasting of lower limbs with reduced power and reflexes, child had difficulty in walking without support, and other systems' examinations were normal. In the chest radiography, there was complete bilateral consolidation involving air bronchogram. Thorax CT revealed extensive pneumonic air space consolidation in all lobes of both lungs. The child was started on initial treatment consisting of oxygen inhalation, iv antibiotics, inhaled steroids and salbutamol, and supportive measures. Since the child had such extensive consolidation. Since the child had no improvement and intermittently would have bouts of cough leading to severe respiratory distress the child was started on fluticasone and salmeterol combination inhaler and Salbutamol MDI. Injectable antibiotic was discontinued and was started on oral azithromycin for its anti-inflammatory property along with high dose oral prednisolone which was gradually tapered and omitted. The patient was discharged from the hospital 11 days later when her symptoms got prominently better but still on medication. The patient was followed up at regular intervals and showed a marked improvement clinically.

Discussion:

Accidental ingestion of hydrocarbons (AIH) is important in the field of paediatrics as they very prone to the side effects and they suffer mainly due to their inquisitiveness and this has been reported in the study in the best institutes of the country.¹ The incidence of accidental poisoning in children ranges from 7-8%.^{1,2} AIH is seen in children mostly who are younger than 5 years. Accidental ingestion of these hydrocarbons can lead to chemical pneumonitis which 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 or as a motor fuel in generators are hydrocarbons. Highly volatile with low viscosity and lower surface tension hydrocarbons are more likely to be inhaled or aspirated into the respiratory system.³ 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

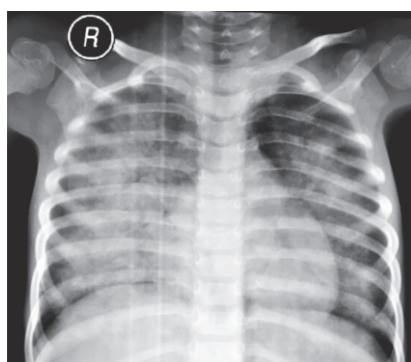


Figure 1. Chest X-Ray of patient showing bilateral consolidation which was taken after 25 days of ingestion

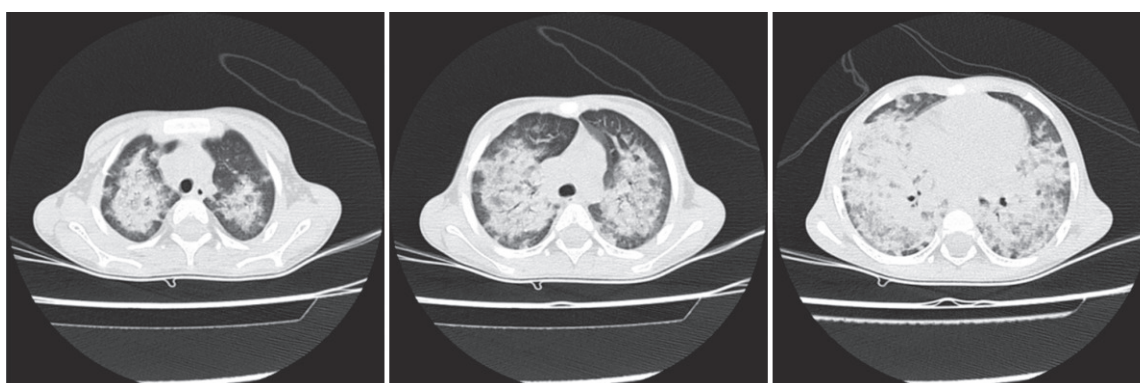


Figure 2. CT Thorax showing Extensive pneumonic air space consolidation in all lobes of both lungs.

without evoking cough reflex. They can then vaporize and displace the oxygen, which can lead to a transient state of hypoxemia. 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 type II pneumocytes are the most affected, resulting in decreased production of surfactant. This decrease in surfactant, results in alveolar collapse, a ventilation-perfusion mismatch, and eventually hypoxemia. The end result of HC aspiration is interstitial inflammation, intra-alveolar hemorrhage and edema, hyperemia, bronchial necrosis, and vascular necrosis.

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. Chest pain may be severe. Tenderness of chest wall is observed which could be related to chemical pleuritis.³ HCs create a burning sensation as they irritate the gastrointestinal mucosa. Vomiting has been reported in up to one third of all HC exposures as in our study also. The chlorinated HCs are hepatotoxic due to free radical formation unlike the aliphatic HCs in our study. The common histopathologic pattern is centrilobular (zone III) necrosis. Liver function test results can be

derranged within 24 hours after ingestion of the HC, and clinically apparent jaundice can be seen within 48-96 hours. Later, these patients may develop cardiomyopathies or fatal cardiac arrhythmias.^{7,11} 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

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.¹² 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.⁹ The definitive diagnosis is made by demonstrating lipid laden macrophages in BAL fluid and in the alveoli or interstitium in bronchoscopic lung biopsy.¹⁰

Management for the treatment of hydrocarbon ingestion is not well established. 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

our case has been clinically better for few days of initial stormy course of chemical pneumonia was better for few days and again had respiratory failure. This may be due to residual chemical effects in lung tissue. It has been observed that in few cases, prolonged lung pathology is evident even for six months. Our case, required

steroids as she had second episode of respiratory failure.

Primary prevention of chemical pneumonitis due to accidental ingestion can be difficult in India because of the inquisitiveness of the children especially children below 5 years of age. There is a need to educate people regarding this habit and its terrible consequences.

Conclusion:

accidental hydrocarbon poisoning can cause repeated respiratory problems in few cases. Some of them requires systemic and inhaled steroids. Regular followup is required in these cases.

Contribution :

NCM- Revising manuscript, Checking MRI/CT plates,;NJ,JC-Collection & analysis of data, writing manuscript, VH- Revising manuscript,, PK-Collection of data

Conflict of Interest: None

Source of Funding :Nil

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Present case showed initial improvement as normally seen in most of patients with hydrocarbon poisoning. But due to residual diesel in lung, deteriorated fastly and required systemic steroids. Prolonged chemical pneumonitis can be seen sometimes upto six months.