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Case Report

Death Due to Accidental Diesel Poisoning

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Abstract:

Hydrocarbons are a group of organic substances made up of hydrogen and carbon molecules. Among the various groups of hydrocarbons, the commonly used fuels like petrol and diesel are classified under aliphatic hydrocarbons. Diesel is fuel for engines in automobiles and for electricity generators.

We present a case of a 55-year-old male, an auto driver by occupation who accidentally ingested diesel and got admitted to GGH, Nizamabad. He developed aspiration pneumonia and expired, following which an autopsy was conducted in this case. In this case, viscera were preserved for chemical analysis & lungs were preserved for histopathological examination. The chemical analysis of viscera revealed traces of inflammable hydrocarbons and on histopathological examination of the lungs indicated aspiration pneumonia. On HPE of the lungs, it was observed that there was intra-alveolar serous accumulation and lymphomono nuclear inflammatory cell infiltrates. Additionally, the alveolar septa showed thickening and congestion, indicating the presence of aspiration pneumonia.

Hydrocarbon exposure leads to a spectrum of manifestations like pneumonitis, myocarditis, skin lesions, mediastinitis, and Parkinson's disease. Diesel is considered to have a relatively low aspiration potential compared to other substances, and fatal cases of diesel poisoning due to aspiration are uncommon.

In cases of chemical pneumonitis caused by hydrocarbon poisoning, the respiratory rate serves as a crucial prognostic factor for determining mortality. A high respiratory rate at the time of presentation is associated with poor outcomes and an increased risk of fatality. The development of Acute Respiratory Distress Syndrome (ARDS) is the ultimate pathway of toxicity in these cases, as observed during autopsy examinations.

Keywords: Hydrocarbons, Pneumonitis, Aspiration, Surface Active Agents, Diesel.

Introduction:

Hydrocarbons are a group of organic substances that are made up of carbon and hydrogen molecules. Hydrocarbons are classified as aliphatic hydrocarbons, aromatic hydrocarbons, halogenated hydrocarbons, cycloparaffins, and alkenes. Gasoline or petrol, kerosene, and diesel belong to the group of aliphatic hydrocarbons. Diesel is distilled from petrol and is used as a fuel for engines in automobiles and electricity generators. The exposure to diesel can occur by various means like ingestion, inhalation, intravenous injection, and through the dermal route. Such exposure is common among workers inthe petrochemical industry and automobile workshops. The exposure to petrochemical substances is extremely high among the rural Indian population as people tend to siphon the fuel using rubber tubes by creating a suction effect with mouth and handling all the equipment with bare hands.

According to the available literature on

hydrocarbon poisoning, common hydrocarbons that are known to pose potential risks of aspiration include gasoline (petrol), kerosene, mineral seal oil, and turpentine. On the other hand, diesel, which falls under aliphatic hydrocarbons, is generally considered to have a lower propensity for aspiration and cases of fatal diesel poisoning due to aspiration are relatively rare. Occasionally, adults and older children consume liquid hydrocarbons placed in an unlabeled can or bottle which results in accidental ingestion.

Intoxication through ingestion and aspiration of hydrocarbons leads to pneumonia which rarely leads to fatal outcomes. Pneumonitis induced by aspiration of diesel was first reported by Laughlen in 1925 and since then there have been several incidents of diesel poisoning being reported in the literature.[1]

Recreational exposure to hydrocarbons is becoming more common nowadays. The inhaling of hydrocarbons or other volatile solvents produces a transient state of euphoria for the abusers, that drives them to abuse these substances. The majority of patients who ingest hydrocarbons and develop chemical pneumonia typically experience a complete recovery, with death occurring in less than 1% of cases. [2] The general respiratory symptoms after exposure to hydrocarbons usually appear in the first hours after exposure and they diminish usually after an interval between 2 and 8 days.[3] As for the treatment part, in pneumonitis as a result of hydrocarbon exposure,the use of steroids is thought to limit inflammation and fibrosis.[4] As stated above, the practice of siphoning diesel is common in rural communities which leads to accidental aspiration and ingestion of diesel. The management in such cases is supportive and patients may need prolonged hospitalization. [5] Death due to exposure to diesel is rare and we report a case of death due to fatal diesel poisoning.

Case Report:

A 55-year-old male, an auto driver by occupation, had an accidental ingestion of about 50ml of diesel at his residence.After accidentally ingesting the substance, the patient attempted to induce vomiting manually several times in an effort to expel it. Additionally, the patient consumed turmeric water in an attempt to alleviate the effects of the ingestion. However, the patient developed a burning sensation in the abdomen following which he was brought to the casualty of Government General Hospital, Nizamabad.

On examination,SpO2 was found to be 94% at room air,BP was found to be 80/50mmHg, respiratory rate was 28/min, CVS: NAD, RS: bilateral basal crepitations present. GCS 15/15, Lab values were as follows, FBS- 136mg/dl, blood urea- 70mg/dl, Serum creatinine- 2.4mg/dl, Serum bilirubin- 0.8mg/dl, SGPT- 17 U/L, SGOT- 25 U/L, Hb- 13.2mg/dl, TLC- 4,100/mm3, Platelets- 1.8Lakhs/mm3, DLC: Neutrophils-59%, Lymphocytes- 35%, Eosinophils- 02%, Monocytes- 04%, Basophils- 0%. Serum electrolytes were as follows: Sodium- 138mg/dl, Potassium- 3.5mg/dl, Chlorine- 110mg/dl. The ABG report was suggestive of Type I respiratory failure. The patient was kept on 6 litres of oxygen initially and was subsequently shiftedtoNIV support due to tachypnea. However, due to the SpO2 levels dropping to 60% and showing no signs of improvement, the decision was made to intubate the patient. The patient succumbed to the complications of diesel poisoning within 3 days and an autopsy was conducted.

During the autopsy, there were no external injuries on the body. All the visceral organs including lungs, liver, spleen, kidneys, and adrenals were congested. The stomach contained 100ml of a brown viscous fluid with a pungent smell and the mucosa was congested. The internal organs (viscera) were preserved for chemical analysis at the Forensic Science Laboratory in Hyderabad, while the lungs were preserved in 10% formalin for histopathological examination at the Department of Pathology, GGH, Nizamabad. The chemical analysis conducted at the Forensic Science Laboratory revealed the presence of inflammable hydrocarbons in the examined viscera. On histopathological examination of the lungs, it was observed that there was intra-alveolar serous accumulation and lymphomononuclear inflammatory cell infiltrates. Additionally, the alveolar septa

showed thickening and congestion, indicating the presence of aspiration pneumonia. (Figures 1-3)

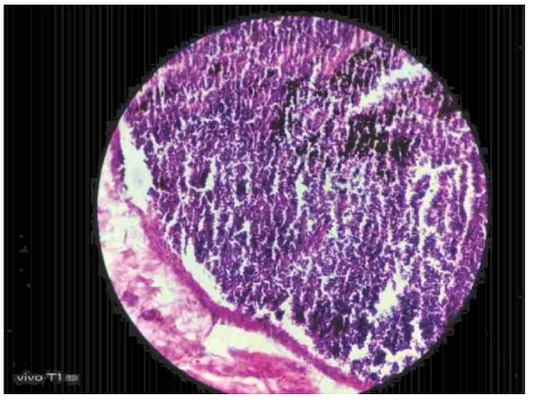

Figure 1 : Lung Photomicrograph, (H& E,20X) showing lymphomono nuclear infiltration

Figure 2 : Lung Photomicrograph, (H & E, 20X) showing alveolar septal congestion

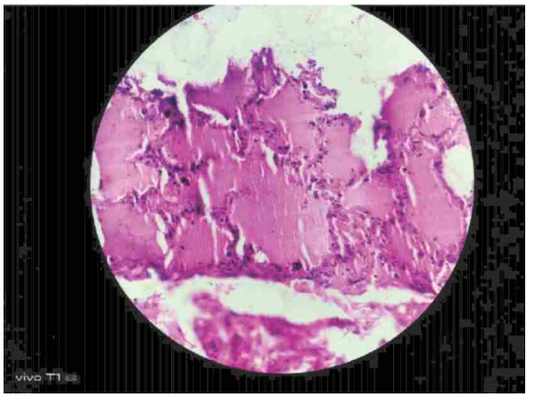

Figure 3 : Lung Photomicrograph, (H & E, 40X) showing intra alveolar serous accumulation

Discussion:

Ingestion of hydrocarbons leads to toxicity affecting many organ systems among which the pulmonary system is the most commonly affected.[6] The specific toxicity of each hydrocarbon is based upon its chemical properties whereas the organ which would be exposed to toxicity depends upon the dose and route of ingestion. Most of the aliphatic hydrocarbons are absorbed in small amounts from the gastrointestinal tract whereas aspiration occurs frequently either initially or in a semi-delayed fashion as the patient coughs or vomits and it may even lead to severe pneumonitis. Several studies have shown that exposure to hydrocarbon solvents is a risk factor for the earlier onset of symptoms of Parkinson's Disease (PD) and paving the way for a severe course, which also suggests that hydrocarbon solvents may have a role in the etiopathogenesis of PD.[7]

Hydrocarbon poisoning may also lead to unusual clinical manifestations like encephalopathy, renal tubular acidosis, skin lesions, and myocarditis.[8] Diesel has a high potential for aspiration due to its low viscosity and high volatility.[9] Aspiration of diesel may cause chemical pneumonitis which presents nonspecifically with breathlessness, cough, chest pain, and hemoptysis as symptoms.[10] Among other hydrocarbons, accidental poisoning of kerosene is common in children leading to aspiration pneumonitis. Safe storage of toxic compounds away from children, avoiding storage of kerosene and other fuels in beverage bottles, and parental supervision are a few interventions to prevent accidental poisonings in children.[11]

Hydrocarbon pneumonitis mostly affects the type 2 pneumocytes in alveoli which results in a decrease in surfactant production leading to alveolar collapse, ventilation-perfusion mismatch, and hypoxemia. Hemorrhagic alveolitis can also occur subsequently which peaks 3 days after ingestion.[12] Hydrocarbon aspiration can result in various pulmonary complications, including interstitial inflammation, intra-alveolar haemorrhage, edema, hyperemia, bronchial necrosis, and vascular necrosis. In rare cases, it may lead to the development of pneumothorax or bronchopleural

fistula. There have been reported cases where ingestion of kerosene has been associated with the development of pneumatoceles.[13] The common modes of exposure to hydrocarbons are ingestion and inhalation, which can lead to chemical pneumonitis. However, it is worth noting that rare cases of chemical pneumonitis have been reported due to the intravenous injection of hydrocarbons.[14] Ingestion of diesel may at times lead to perforation of the esophagus and pneumomediastinum and that may lead to mediastinitis. Mediastinitis carries a high risk of mortality and can be diagnosed by a CT scan, which typically shows sternal separation, sub-sternal fluid collection, or air-fluid levels within the mediastinum.[15] In hydrocarbon ingestion, the oily particles that are aspired into the airways do not stimulate the coughing reflex and they reach the inferior respiratory tract. Furthermore, the mucociliary clearance is disturbed and that prevents the evacuation of particles gathered in the alveolar macrophages. Pneumonitis developing because of hydrocarbon exposure shows certain characteristic findings on CT scan like air space consolidation predominantly in the right middle lobe and areas of low attenuation within the consolidation.[16] In cases of hydrocarbon pneumonia developing due to white spirit aspiration, HRCT reveals patchy opacities of coalescing masses with well-defined walls in the middle lobe, lingula, and lower lobes.[17]

Diagnostic evaluation for hydrocarbon exposure includes various modalities. X-ray imaging can reveal early changes within 30 minutes of exposure, characterized by perihilar densities, broncho vascular markings, bibasilar infiltrates, and pneumonic consolidation. In early erect X-rays, the presence of a "double bubble" sign in the stomach may be indicative. ABG analysis helps assess hypoxemia, reflecting decreased oxygen levels in the blood. Blood tests often demonstrate leukocytosis within the initial 48 hours post-exposure. ECG and continuous cardiac monitoring are recommended for assessing cardiac function. Biochemical markers such as BUN, creatinine, serum CPK, and liver enzymes provide additional insights into the patient's condition.

Patients manifest various signs and symptoms following hydrocarbon exposure. These may include lethargy, the presence of abnormal lung sounds such as rhonchi and rales, retractions (intercostal and subcostal), cyanosis, leukocytosis, and fever typically observed within approximately 4 hours post-exposure. *Tachypnea (Respiratory Rate)*serves as an important clinical indicator of potential toxicity. Close monitoring of these clinical manifestations aids in assessing the severity and progression of hydrocarbon-related complications.

In cases of hydrocarbon toxicity, the following management approaches are employed. The mainstay of management is supportive which includes supplemental oxygen and mechanical ventilation. The use of antibiotics is necessary if there are any signs suggestive of super-added infections. The role of steroids in affecting mortality is not yet established. However, in severe chemical pneumonitis, practitioners routinely prescribe them for the benefit of patients. Immediate endotracheal intubation is crucial for ensuring adequate airway protection and ventilation. Symptomatic patients receive high-flow oxygen therapy. For pulmonary edema and acute lung injury, continuous positive airway pressure (CPAP) is employed, and in severe cases, intermittent positive pressure ventilation (IPPV) with positive end-expiratory pressure (PEEP) may be considered. Extracorporeal membrane oxygenation (ECMO) can be utilized as an effective option when other interventions fail to improve severe pulmonary toxicity. Bronchodilators, such as salbutamol, may be administered.

Decontamination procedures vary depending on the exposure route. In cases of dermal exposure, contaminated clothing is removed, and the affected area is washed with disinfectant soap and water. Emesis induction is contraindicated. For significant ingestions, stomach lavage can be attempted after intubation, while activated charcoal may also be administered. Pulse steroid therapy may be considered in the advanced stage of adult respiratory distress syndrome. In critically ill patients where pulmonary cultures cannot be obtained, empirical broad-spectrum prophylactic antibiotic therapy is necessary.

Caution must be exercised when using adrenaline and other sympathomimetics, as they can potentially precipitate ventricular arrhythmias. In cases of frequent or prolonged convulsions, intravenous diazepam, lorazepam, or midazolam can be administered. Phenobarbital sodium can be used as a second-line therapy, and slow intravenous administration of phenytoin can be an alternative. If convulsions persist, referral to intensive care, general anaesthesia, intubation, and ventilation may be necessary.[18]

In the present case, the storing of diesel in a PET bottle with the label of a beverage led to accidental consumption of diesel, and subsequent attempts of induced vomiting by the deceased individual led to aspiration, pneumonitis, and ARDSsequelae.

Conclusion:

Ingestion and aspiration of hydrocarbons have a direct impact on the pulmonary parenchyma and can lead to fatal outcomes. Timely and appropriate treatment following exposure plays a crucial role in preventing death and improving patient outcomes. In this particular case, death occurred as a result of aspiration pneumonia. During the histopathological examination, we were unable to identify lipoid cells due to the considerable passage of time since the toxic exposure had taken place. The crucial prognostic factor which is helpful in gauging mortality in chemical pneumonitis due to hydrocarbon poisoning is the 'respiratory rate'. A high RR at the time of presentation leads to fatal outcomes. The final common pathway of toxicity is the Acute Respiratory Distress Syndrome (ARDS) lung for autopsy purposes.

Conflicts of interest : None to declare

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