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Review article

Kerosene poisoning

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ABSTRACT

Accidental poisonings especially in children are relatively common due to inappropriate storage, easy availability and exploratory behavior of the child. Kerosene per se is not particularly poisonous. However, if a child or adult accidentally swallows kerosene, it can have multiple adverse health effects due to its aspiration into the tracheobronchial tree. Kerosene has been identified as the most common cause of accidental poisoning in various studies around the world. Ingestion of large quantity of kerosene is rare because of its foul smell and taste. Aspiration of kerosene usually occurs during swallowing. The complications of kerosene poisoning include hypoxia, pneumonitis, bacterial pneumonia, pneumatocele, pleural effusion, pneumothorax, subcutaneous emphysema and empyema. Timely recognition and management of pulmonary complication is a key to successful outcomes.

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1. Introduction

Kerosene is a liquid mixture of chemicals produced from the distillation of crude oil. It is a type of aliphatic hydrocarbon. The word kerosene comes from the Greek word 'keros', meaning 'wax'. Kerosene is still extensively used for cooking, heating and lighting in our country especially among the rural and the poor urban population. Therefore accidental poisoning by children is still relatively common due to inappropriate storage, exploratory behavior of the children and easy availability. Kerosene per se is not particularly poisonous. However, if a child or adult accidentally swallows kerosene, it can have multiple adverse health effects. Hydrocarbon poisoning including kerosene poisoning accounts for 5% of all accidental poisoning and about 25% of all deaths in children under 5 years of age [1,2]. The aspiration hazard of the hydrocarbons is determined by properties of volatility, surface

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tension and viscosity. The aspiration hazard of any hydrocarbon is inversely related to viscosity and surface tension and directly related to volatility [3,4]. Thus, kerosene oil with decreased viscosity, low surface tension, and high volatility is more likely to be aspirated and cause pulmonary injury. However, it rarely causes other systemic effects like central nervous system (CNS) or cardiovascular events.

2. Pathophysiology

Kerosene oil aspiration impacts predominantly the pulmonary tree particularly the distal airways. The primary mechanism of injury is its direct toxicity due to the physical and chemical properties of kerosene oil. Its physical properties of low viscosity, low surface tension and volatility aid its easy spread into the distal airways and penetration deep into the tracheobronchial tree thus causing maximum damage to the distal airways. Necrotizing pneumonia is the predominant pathological finding. Other findings include direct destruction of the airway epithelium, alveolar septae, and pulmonary capillaries, as well as secondary surfactant depletion. These changes cause segmental atelectasis, hemorrhagic exudative alveolitis, interstitial inflammation, and hyaline membrane formation very much like hyaline membrane disease of the newborn. The type II pneumocytes are most commonly affected, resulting in decreased surfactant production. This decrease in surfactant results in alveolar collapse, ventilation - perfusion mismatch, and hypoxemia and its associated consequences [5]. Incidence of hemorrhagic exudative alveolitis peaks 3 days after ingestion. Pneumothorax, pneumatocele, or bronchopleural fistula and pulmonary air-leak syndrome are other abnormalities which may be seen [6]. Inflammatory response is triggered due to chemical irritation and causes systemic response including temperature elevation, usually within hours of exposure not unlike sepsis.

3. Clinical features

Most patients who ingest kerosene oil are children who are initially asymptomatic but have a history of exposure given by the parents or relative. Symptomatic patients with respiratory distress and hypoxemia soon after ingestion typically progress rapidly to respiratory failure. Usually a peculiar petroleum distillate odor is discernible [7]. Urine and blood toxic screen is not easily available in our country (India) and does not aid in diagnosis and management of the patient.

Respiratory complaints may develop within 6 h or may be delayed in some cases for up to 48–72 h. Vomiting, whether spontaneous or induced, increases the chances of aspiration and chemical pneumonitis. Direct aspiration into trachea even in miniscule amount of kerosene oil can cause pneumonitis and ultimately death. Recovery usually takes place after about a week but may be prolonged if secondary bacterial pneumonia develops and complicates the clinical course.

Fever can occur as early as 30 min after ingestion and persists for several days. The severity and duration of fever usually correlates with the severity of respiratory involvement. It is a marker for superadded bacterial infection if present beyond 48 h of ingestion.

4. Physical examination

4.1. Vital signs

Patients with kerosene aspiration have fever at the time of presentation with associated tachycardia and tachypnea. Persistence of fever beyond 48 h suggests secondary bacterial infection. There may be signs of respiratory distress in form of nasal flaring, head bobbing and use of accessory muscles. Blood pressure may be abnormal due to the systemic inflammatory response. Pulse oximetry is an integral part of monitoring and may show presence of hypoxia.

4.2. Respiratory examination

Respiratory manifestations secondary to kerosene ingestion generally occur within 30 min, although it may be delayed. Immediate signs of aspiration include coughing, choking, gagging, and vomiting very similar to foreign body aspiration in children. Findings vary with the degree and extent of pulmonary injury and may include tachypnea, difficulty in breathing with objective signs of distress, wheezing, diminished resonance on percussion, bronchial breath sounds or crepitation depending on the pathology and stage of presentation.

5. Laboratory finding

5.1. Complete blood count

Neutrophilic leukocytosis occurs early in the clinical course. It is unrelated to the pulmonary pathology and persists for about a week. In cases of massive ingestion, the peripheral blood smear may reveal evidence of hemolysis. There may also be presence of hemoglobinuria and disseminated intravascular coagulopathy (DIC).

5.2. Ancillary studies

Children who ingest kerosene should receive a chest radiograph four to six hours after exposure or sooner if they show signs of pulmonary aspiration. Symptomatic patients should also undergo the following investigations:

1 Arterial blood gas: The initial blood gas findings typically show a mild respiratory alkalosis with hypoxemia. If uncorrected it will further lead to metabolic acidosis. If hypoxemia is not corrected, the patient will later develop a metabolic acidosis.
2 Liver and kidney function tests with serum electrolytes.
3 Urinalysis.

5.3. Radiological investigation

Imaging studies may show radiographic abnormalities on chest X-ray in 88 percent of patients within 2 h and by 12 h in 98%. These findings frequently precede the development of clinical findings. Initial radiograph findings consist of multiple, small, fluffy, patchy densities with ill-defined margins. These lesions become larger and coalesce as the injury progresses. There may be evidence of emphysematous changes or pneumothorax and pulmonary air leaks [8]. Radiographic abnormalities typically peak between two and eight hours after aspiration. The resolution of radiographic changes is gradual and usually lags behind clinical resolution, which usually occurs three to five days after aspiration in an uncomplicated course. There may be development of pneumatoceles during this latent period [9].

6. Management

The major toxicity of kerosene ingestion is pulmonary function impairment and therefore all initial attempts should be made to recognize and appropriately manage the same in a timely manner. Appropriate supportive care should be rapidly instituted. The

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