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BILATERAL EMPYEMA FOLLOWING KEROSENE POISONING - A CASE REPORT

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Abstract : Kerosene is a highly volatile hydrocarbon with aspiration hazard. The toxic effects following ingestion or inhalation are predominantly related to the direct pulmonary damage and subsequent inflammation, resulting in chemical pneumonitis. Successful management of hydrocarbon aspiration requires recognition of pulmonary toxicity and rapid initiation of appropriate supportive care. Empyema following kerosene poisoning is very rare. We report a case of bilateral empyema following kerosene ingestion in a 16 year old girl from South India.

Keyword : Kerosene, Chemical pneumonitis, Bilateral empyema

INTRODUCTION

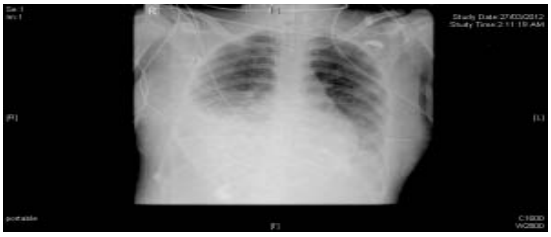
Kerosene poisoning is a commonly encountered emergency in general medical practice. Kerosene is a low viscosity liquid hydrocarbon compound with aspiration hazard. Poisoning is usually due to inhalation of fumes or ingestion of small amounts accidentally or with suicidal intent. The highest rates of morbidity and mortality result from accidental ingestion by children younger than 5 years [1,2]. The major impact is on the respiratory system and the central nervous system. Kerosene affects the respiratory system, primarily by direct toxic injury and subsequent inflammation, resulting in chemical pneumonitis. Secondary bacterial or viral infection, lipoid pneumonia, pneumatoceles, and pleural effusion are the pulmonary complications of kerosene poisoning described in literature. Empyema following kerosene poisoning is a very rare complication [3,9].

CASE REPORT

16- year- old girl from South India was brought to the emergency ward with a history of alleged ingestion of approximately 10 mL of kerosene about 12 hours back with an intention of deliberate self-harm, following an altercation with her parents. She was taken to a nearby hospital within 2 hours, where emesis was induced. She developed worsening breathing difficulty subsequently, and was referred to our centre for further management. In the Emergency ward, she was found to be hypoxaemic, was started on supplemental oxygen therapy, and was admitted to the acute medical At admission, she was drowsy. She was normotensive with tachycardia and tachypnoea. Blood pressure was normal.
Respiratory system examination revealed bilateral crackles. Other systemic examination was unremarkable.

On evaluation, her total leukocyte count was 13400 /cu mm with 80% neutrophils. Liver and renal function tests were normal. Initial chest radiograph revealed parenchymal infiltrates involving bilateral lower lung zones [**Image 1**]. A diagnosis of kerosene poisoning with chemical pneumonitis was made, and she was continued on supplemental oxygen with close monitoring for worsening of respiratory distress.

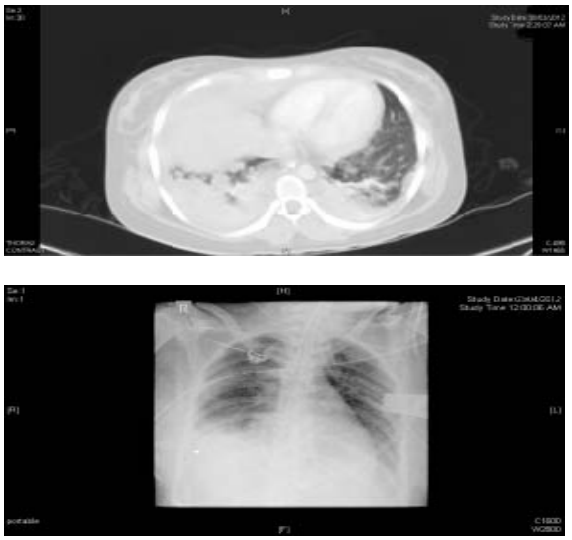
Image 1



On the second day, the respiratory distress and hypoxaemia worsened in the acute medical care ward, and she was shifted to the Medical High Dependency Unit, where she was electively intubated after a trial of non-invasive ventilation. Subsequently, on the fourth day of admission, she developed However, she continued to be febrile, and hypoxaemia persisted. Serial chest x rays showed worsening of pulmonary infiltrates. CT thorax done subsequently revealed bilateral loculated pleural effusion, subsegmental collapse-consolidation involving bilateral lower lobes and multiple lung parenchymal opacities suggestive of pneumonia [**Image 2**]. Intercostal drains were placed into the loculated effusions bilaterally and gross pus was drained [**Image 3**]. Gram stain of the pus revealed gram positive cocci; however theculture remained sterile. She was continued on injection Piperacillin-Tazobactum for a total duration of 2 weeks. Subsequently, her hypoxaemia resolved, lung infiltrates disappeared, and she was weaned off the ventilator after 12 days of mechanical ventilation.

Thereafter she was discharged in a stable state after removal of intercostal drains. At discharge, she was advised to continue oral Amoxycillin-Clavulanic acid to complete a total of 6 weeks of antibiotic therapy.

Image 2 Image 3



DISCUSSION

The toxic potential of hydrocarbons is directly related to their physical properties. Kerosene is a highly volatile compound , classified among hydrocarbons with aspiration hazard, of which the local toxic effects are largely due to direct pulmonary damage and subsequent inflammation [4]. Low viscosity and surface tension allow greater penetration into the distal airways, and facilitate spread over larger area of lung parenchyma resulting in chemical pneumonitis. The primary pathologic finding is severe necrotizing pneumonia. Secondary changes include atelectasis, interstitial inflammation, and hyaline membrane formation.

Clinical manifestations usually occur within 30 minutes of ingestion of the compound. However, it may be delayed for 12 to 24 hours. The common respiratory manifestations are cough, tachypnea, dyspnea, cyanosis, wheezing and crackles. Complications such as asphyxia, necrotizing chemical pneumonitis, lipoid pneumonia, secondary bacterial or viral infection, hemorrhagic pulmonary edema, pneumatoceles, pneumothorax, subcutaneous emphysema of the chest wall, and pleural effusion including empyema are described [3,5,6]. Patients with mild to moderate respiratory symptoms on presentation are at risk for respiratory failure over the subsequent 24 to 48 hours. Radiographic findings are evident within two hours of exposure in 88 percent and by 12 hours in 98 percent of the patients, and may precede the development of physical findings. Common findings include fine perihilar opacities, bibasal infiltrates, and atelectasis.

Aspiration pneumonia is the most common complication of hydrocarbon ingestion (40%) [7], the development of which is facilitated by the impairment of pulmonary defence mechanism and aspiration of oral micro-organisms [3]. Vomiting after hydrocarbon ingestion has been found to increase the risk of development of pneumonia [8]. The fact that vomiting was induced in our patient prior to bringing her to the hospital may have contributed to the development of pneumonia.

Fever occurring within hours of exposure usually results from inflammatory response to the chemical irritation, and does not warrant prophylactic use of antibiotics [1]. Features of secondary infection as evidenced by recurrence of fever after the first 48 hours, increasing infiltrates on serial chest radiographs, leukocytosis after the first 48 hours, and sputum or tracheal aspirate cultures positive for bacteria are indications for initiation of antibiotic therapy. Choice of antibiotic regimen should cover common gram positive and gram negative organisms, as well as anaerobes [10]. Corticosteroids have shown no beneficial effect in observational studies, and may even be harmful [11].

Treatment of empyema involves surgical drainage of the pus and institution of an appropriate antibiotic therapy for a total duration of 6-8 weeks.

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