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CLINICAL PROFILE AND OUTCOMES OF KEROSENE POISONING IN CHILDREN OF MOSUL, IRAQ

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ABSTRACT

A study was conducted on 64 children who accidentally ingested kerosene between June 2021 and June 2024. Most of these children were between one and three years old, and boys were three times more likely to be affected than girls. Common symptoms included coughing, difficulty breathing, vomiting, and drowsiness. Fever often developed later, typically within six hours of ingestion. A significant number of children (75%) developed kerosene aspiration pneumonia within six hours. This was primarily diagnosed through X-rays, as clinical symptoms were not always obvious. The most common finding on X-rays was fluid in the lower right lung. Vomiting was a major risk factor for pneumonia, occurring in 85.4% of children who developed the condition. All patients received antibiotics, but these did not reduce the duration of fever. Unfortunately, one child deteriorated rapidly and passed away soon after arrival. However, the remaining patients improved clinically and radiologically over a period of 3-10 days. Complete recovery on X-rays took longer, with some children taking up to 28 days.

KEYWORDS: Kerosene ingestion; Childhood poisoning; Aspiration pneumonia; Clinical outcomes.

INTRODUCTION

Kerosene ingestion is a leading cause of accidental poisoning and subsequent morbidity and mortality in young children, particularly in developing countries across Africa, India, Iraq, and other regions where it is widely used for lighting, cooking, and heating.[1–3] Hydrocarbons (petroleum distillates) comprise a vast array of chemicals found in thousands of commercial products, including gasoline, kerosene, benzene, lamp oil, lighter fluid, paint thinners, furniture polish, and cleaning agents.[4,5] These products, being fat solvents, disrupt nerve function, potentially causing depression, convulsions, and coma.

Hydrocarbon toxicity stems from two primary mechanisms: local toxicity, involving chemical burns to the mouth, throat, and skin, with potentially severe lung damage[4, 7]; and systemic toxicity, affecting the central nervous system and viscera, resulting in gastrointestinal symptoms, liver toxicity, cardiomyopathy, and renal toxicity. Aspiration into the respiratory tract is the major hazard of accidental hydrocarbon ingestion[4–9], accounting for almost all deaths. Toxicity is related to viscosity; high-viscosity, low-volatile products are generally less toxic upon ingestion, while low surface tension, low viscosity, and high-volatile products pose a greater risk of pulmonary symptoms.[9,10] Hydrocarbons are categorized by their risk profile, with some (e.g., asphalt, tar, motor oil) being essentially non-toxic in most cases. Pulmonary involvement follows aspiration and inhalation during swallowing, spreading from the hypopharynx to the trachea, rather than through gastrointestinal absorption.[7, 9] Vomiting significantly increases this risk of aspiration.[3–5] The resulting lung injury manifests as diffuse acute alveolitis, necrotizing bronchopneumonia, interstitial inflammation, and pulmonary edema, lasting 3–10 days and potentially transitioning into a chronic inflammatory response resolving over several weeks.[1, 7] Surfactant damage can also lead to alveolar collapse and airway closure.

Central nervous system (CNS) depression, caused by the absorbed hydrocarbons, varies in severity with the amount absorbed[6] While both systemic and direct brain toxicity contribute, cerebral hypoxia secondary to respiratory insufficiency often plays the major role[6]

Low molecular weight, low-viscosity hydrocarbons (e.g., benzene, gasoline, mineral spirits) are more likely to cause necrotizing CNS effects than kerosene.[8, 9] Severe pulmonary involvement frequently accompanies CNS complications (Majeed, HA et al.). Aromatic and halogenated hydrocarbons are also absorbed, causing liver, heart, and kidney damage[11] Specific examples include hepatotoxicity from carbon tetrachloride, myocardial sensitization and dysrhythmia risk from volatile hydrocarbons, and renal complications such as tubular acidosis and failure.[3, 11] Bone marrow suppression, skeletal muscle damage, methemoglobinemia (from nitrobenzene, aniline, and related compounds), and intravascular hemolysis may also occur.[1, 3, 4]

Clinical features of hydrocarbon poisoning begin with mouth and throat burning, choking, coughing, nausea, vomiting, hemoptysis, and potentially coma.[8] Respiratory distress (tachypnea, nasal flaring, retractions, grunting, tachycardia, and cardiac decompensation) signals severe pulmonary involvement.[11] CNS depression manifests as weakness, hypotonia, dizziness, confusion, lethargy, irritability, agitation, or convulsions.[8] Gastrointestinal symptoms (abdominal pain), liver and kidney dysfunction, and rhabdomyolysis may also develop, with sudden death from myocardial irritability and ventricular fibrillation possible.[11] Late sequelae include weakness, weight loss, anemia, peripheral neuropathy, psychosis, and cerebral or cerebellar atrophy.[6–11] Kerosene pneumonitis specifically presents with immediate coughing and vomiting, followed within hours by fever (38–40°C).[14] Less extensive aspiration may delay pulmonary symptom onset by 12–24 hours.[14, 15] Dyspnea, transient cyanosis, diminished resonance on percussion, and abnormal breath sounds (suppressed, tubular, or crackles) may be observed.[14, 16]

Radiological examination (chest x-rays) often reveals pneumonic involvement more readily than physical findings, showing minimal changes initially but rapidly progressing to extensive infiltrates.[14] Chest x-rays might also show punctate or mottled densities in the perihilar region extending into the midlung field, basal pneumonitis, atelectasis, and consolidation.[16] Air esophagogram, gaseous gastric distension, and double gastric fluid levels may be seen.[14, 16] Complications like pneumothorax, subcutaneous emphysema, pleural effusion (including empyema), and pneumatocele may develop later.[4,14,16,19,20] A poor correlation exists between physical findings and radiological abnormalities[8, 16], with radiological changes usually resolving within 10–14 days.[21, 22]

Laboratory investigations may show leukocytosis, although this is often misleading as bacteria are usually absent in aspiration pneumonitis[4] Pulmonary function tests years later may reveal small airway abnormalities with or without elastic recoil loss, correlating with the severity of the acute insult and potentially increasing the risk of chronic lung disease in adulthood.[12, 16, 23, 24]

Treatment and hospitalization are warranted for symptomatic patients (at initial examination or within 6 hours of observation), those with abnormal chest x-rays, or those who ingested particularly toxic agents (e.g., furniture polish).[14–16] Hospital stays averages 3–5 days. Asymptomatic patients with normal chest x-rays after 6 hours can be observed at home with instructions to return if respiratory symptoms develop.[16] Treatment is primarily supportive, with oxygen, respiratory physiotherapy, and caloric intake aiding recovery. Antibiotics are only indicated for confirmed secondary bacterial infections.[13, 14, 15] Eliminating hydrocarbons through induced vomiting or gastric lavage is contraindicated due to the aspiration risk, especially hours after ingestion.[8, 11, 14, 16] Nasogastric suction may be considered for large ingestions if endotracheal intubation is possible without inducing vomiting.[14] Antimicrobial therapy is generally not routinely recommended[14] but may be necessary for secondary bacterial infections (fever reappearing on days 3–5), potentially using penicillin and tobramycin or adding anaerobic coverage (metronidazole).[14] Corticosteroids are not beneficial and may be harmful[11, 14, 15, 17] Ventilation and oxygen therapy (including continuous positive airway pressure and extracorporeal membrane oxygenation) are important in severe cases.[14, 16, 18]

Most children recover fully[14, 15], but some progress to respiratory failure and death. Prognosis depends on the amount ingested or aspirated and the quality of medical care.[14] Prevention relies on education for both medical professionals and the public, emphasizing safe kerosene storage out of the reach of children[1, 16] This study aims to evaluate children presenting after kerosene ingestion, assessing their clinical and radiological findings according to the time of presentation.

PATIENTS AND METHODS

This prospective study investigated the clinical characteristics and outcomes of 64 children who were regularly seen at our healthcare facilities between June 2021 and June 2024. All participants presented to the casualty unit and underwent a minimum of six hours of observation. Detailed medical histories were obtained from parents using a structured questionnaire, which included information on family size, maternal occupation, and the method of kerosene storage within the home. This latter variable was included due to its potential association with respiratory illness in this population. Each child received a comprehensive physical examination focusing on the presence and severity of symptoms indicative of respiratory compromise, specifically cough, dyspnea (shortness of breath), cyanosis (bluish discoloration of the skin), drowsiness, vomiting, and signs of lung consolidation (areas of compacted lung tissue).

A chest X-ray (CXR) was performed on every child six hours after their arrival at the casualty unit to assess radiological evidence of respiratory infection or other abnormalities. Complete blood counts (CBCs), including total white blood cell counts, were performed on a subset of 24 patients within the first 24 hours of admission. This smaller sample size for CBCs may reflect logistical constraints or resource allocation within the clinic.

Forty-eight of the 64 children required hospital admission due to the severity of their clinical presentation and/or radiological findings. These hospitalized children received daily monitoring throughout their stay. Data collected during their hospitalization included daily temperature measurements, detailed charting of their clinical progress, documentation of any complications that arose during their treatment, and the total duration of their hospital stay until discharge. This comprehensive data collection aimed to provide a thorough understanding of the disease course and potential risk factors contributing to severe respiratory illness in this pediatric population. The data analysis will likely explore the relationship between the collected variables and the severity of illness, need for hospitalization, and length of recovery.

RESULTS

Epidemiological aspect: The sample's age ranged from nine months to ten years, with most cases clustered between one and three years (mean age: 2.5 years), as shown in Figure 1. The male-to-female ratio was 3:1. A majority of cases, 35 (54.7%), were from urban areas. Additionally, 48 (75%) patients came from impoverished families, and 44 (68.8%) children lived in crowded households (six or more family members). Among the patients, 60 (93.7%) had mothers who were full-time housewives. In 18 (28.1%) cases, kerosene was stored in barrels, while in the remaining cases, it was kept in small containers such as milk or soft drink bottles, kettles, and drinking glasses, as outlined in Table 1. None of the families provided precise information on the volume ingested, although most claimed it was a small amount.

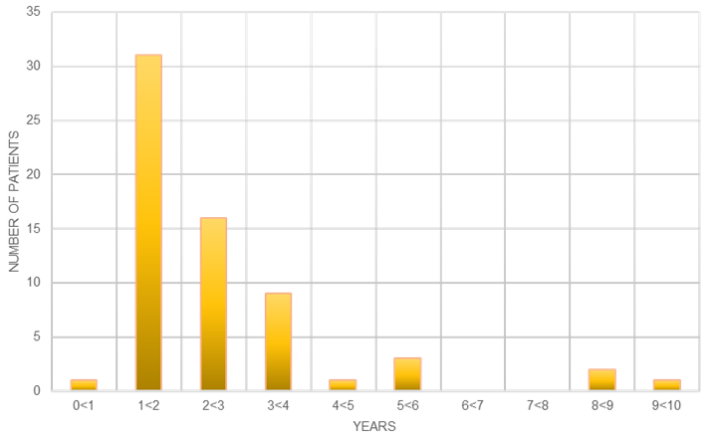

Figure 1. Age distribution of 64 children with kerosene poisoning admitted to causality unit.

Table 1: Epidemiological characters of the study group.

Characters of patients		No.	%
Sex	Male	48	75
	Female	16	25
Residence	Urban	35	54.7
	Rural	29	45.3
Family size	<6	20	31.2
	6+	44	68.8
Socioeconomic	Poor	48	75
	Good	16	25
Mother occupation	Housewife	60	93.75
	Worker	4	6.25
Kerosene storage	Small container	46	71.9
	Barrels	8	28.1

Clinical Features: Cough was the most common symptom, reported in 55 (85.9%) patients, followed by dyspnea in 51 (79.7%) patients. Other clinical signs observed within six hours of ingestion included vomiting, a kerosene odor, drowsiness, and skin changes, as detailed in Table 2. Reduced air entry, rhonchi, and crepitation were noted in a minority of cases (17.2%), primarily affecting the right side. One patient deteriorated rapidly, progressing to coma and death within minutes of admission. Among the 48 admitted patients, the clinical course was characterized by fever (ranging from 38–39.5°C) lasting several days, as well as

constipation, abdominal pain, and chest pain, as outlined in Table 3. Vomiting showed a significant association with the development of pneumonitis (OR: 4.48, P=0.01; Table 4). White blood cell counts were measured in 24 (37.5%) patients; among these, 15 had counts ranging from 12,000 to 15,000/mm³, while the remainder exceeded 15,000/mm³.

Table 2: Symptoms and signs that appeared within 6 hours of ingestion.

Symptoms and signs	No. of affected patients	%
Cough	55	85.9
Dyspnea	51	79.7
Vomiting	41	64
Grunting	40	62.5
Kerosene odour	30	46.9
Drowsiness	13	20.3
Cyanosis.	9	14
Skin changes	3	4.7
Convulsions	0	0
Coma	1	1.6

Table 3: Symptoms and signs that appear after 6 hours.

Symptoms and signs	No. of affected patients	%
Fever	32	50
Constipation	18	28.1
Abdominal pain	10	15.6
Cost pain	1	1.6

Table 4: Relation of vomiting to radiological‹ pneumonitis.

Case	Pneumonitis	No. pneumonitis	Total
Vomiting	(85.4°/°)35	6(14.6%)	41(100%)
No vomiting	13(56.5°/o)	(43.4°/°)10	23(100%)

0. R=4.48 P=O.O1, x2 =6.52 (95% C.I. 1.43- 14)

Radiological abnormalities observed six hours after ingestion included basal infiltrates, primarily on the right side, in 40.6% of cases, and bilateral perihilar infiltrates with clear bases in 21.9% (Table 5). Oxygen therapy was administered to all patients upon admission. Antibiotics were part of the standard treatment for all 48 admitted patients (75%), as prescribed by the attending consultant. The antibiotics used included procaine penicillin, ampicillin, and ampiclox. However, no significant correlation was found between the type of antibiotic administered and the duration of fever in admitted patients, as shown in Table 6.

Table 5: Chest radiological findings in the study group.

Radiological Findings	No. of patients	%
Normal	16	25
Right basal infiltration	26	40.6
Left basal infiltration	8	12.5
Bilateral perihilar infiltration with clear bases	14	21.9
Pleural effusion	3	4.7
Total	64	100

Table 6: Correlation between duration of fever and antibiotic usage.

Duration Of fever in days	No. of patients		
	Procaine Penicillin	Ampicillin	Ampiclox
2	1	2	2
3	1	4	3
4	3	1	1
5	2	3	3
6	1	2	2
7	0	1	0
	n1=8	n=13	113=11

* P > 0.05, p: calculated by usage of ANOVA [F .test]

DISCUSSION

Kerosene ingestion remains a significant cause of accidental poisoning and subsequent morbidity and mortality in young children, particularly in Iraq and other developing countries.[1, 3] In this study, 47 (73.4%) of the patients were aged one to three years. The higher incidence in males (consistent with findings on kerosene and other accidents[3] is likely attributable to boys' increased curiosity and activity levels compared to girls of the same age. The majority of patients, 44 (68.8%) and 48 (75%), were from crowded and impoverished families, respectively. Most patients (35, 54.7%) resided in urban areas, mirroring the results of Singi-S. et al.[23] Kerosene was stored in inappropriately sized containers in 46 (71.9%) of the cases.

The most common presenting symptoms were cough (55, 85.9%), shortness of breath (51, 79.7%), and vomiting (41, 64.1%). Drowsiness was reported in 13 (20.3%) patients; of these, 9 (14%) were cyanotic upon presentation and subsequently diagnosed with severe pulmonary involvement. This suggests that hypoxia secondary to pulmonary involvement, rather than a direct central nervous system effect of kerosene, is the likely cause of drowsiness, consistent with prior studies.[3, 12] Fever (in 50% of patients) and leukocytosis were also observed, aligning with previous findings.[3, 4] While the majority (85.4%) of patients who developed pneumonitis experienced vomiting after ingestion, 13 patients developed pneumonia without a history of vomiting. Kerosene aspiration can thus occur during ingestion, although vomiting significantly increases the risk (χ^2 = 6.520, 95% CI = 1.43–14).[3, 4, 9] Constipation (18, 28.1%) and abdominal pain (10, 15.6%) appeared later, mirroring previous studies.[3, 4] Diarrhea was not observed. No patients experienced convulsions or consolidation, highlighting the superiority of radiological examination over clinical diagnosis in kerosene pneumonia. Radiological findings, consistent with previous studies[3], most commonly revealed right basal involvement (26, 40.6%), bilateral perihilar infiltration (14, 21.9%), and left basal involvement (8, 12.5%). The frequent right basal involvement may be due to the proximity to the trachea.

Pleural effusion, the most frequent complication of aspiration pneumonia in this study (3, 4.7%), was observed in two right-sided and one left-sided case, aligning with the findings of Al-Azzawi and Fagbule et al.[3, 24] Chest tube drainage was not necessary. Pneumatocele and pneumothorax were not observed. All patients received supportive care (oxygen, intravenous fluids, and antipyretics). While the role of antibiotics in kerosene pneumonitis remains debated, the use of procaine penicillin, ampicillin, and ampiclox in our patients did not significantly reduce fever duration ($p > 0.05$), supporting the consensus that antibiotics are generally unnecessary unless secondary bacterial infection is confirmed. All patients showed clinical and radiological improvement within 3–10 days, though complete radiological resolution took 12–28 days in 60% of cases.

CONCLUSIONS

Clinical findings alone are insufficient to exclude kerosene pneumonitis; radiological examination is essential. Patients should not be discharged within six hours of kerosene ingestion, as pneumonitis development may be delayed. Because vomiting is a significant risk factor for pneumonitis, healthcare providers should emphasize this point to the public. Antibiotics are contraindicated unless secondary bacterial infection is suspected. Public health campaigns utilizing television, radio, newspapers, and other media should focus on the dangers of storing kerosene in small, easily mistaken containers (e.g., those used for water or soft drinks). Such preventative measures can substantially reduce or eliminate kerosene ingestion accidents.

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