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RESEARCH ARTICLE

KEROSENE POISONINGIN CHILDREN

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ABSTRACT

A prospective study was carried out on 64 children admitted with accidental kerosene ingestion in AL- Khansa teaching hospital for children in Mosul in the period from the first of July 1999 to the 31 1st. of December 1999. Most children (73.4%) were one to three years. And male to female ratio was 3:1. The most common presenting symptoms were cough 55 (85.9%), dyspnea 51 (79.7%), vomiting 41(64%), and drowsiness in 13 (20.3%). Fever appeared Later on (more than 6hours after ingestion of kerosene) in 32 (50%) of patients and continued for 2-8 days. Forty two patients (75%) developed kerosene aspiration pneumonitis within 6hours after ingestion of kerosene which was diagnosed mainly by radiological examination rather than by clinical findings. Right basal infiltration was the commonest radiological finding.40.6%. Vomiting seems to be the major risk factor for development of pneumonitis which is found in 85.4 % of those who developed vomiting (p=0.01). Routine. antibiotic were given to all admitted patients and have been found that it has no role in decreasing the duration of fever. (p>0.05) One patient deterioated rapidly and died within few minutes after arrival. All patients improved clinically and radiologically within 3-10 days, complete radiological recovery did not take place until 12-28 days in as inany as (60%) of patients.

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INTRODUCTION

Kerosene ingestion is the commonest cause of accidental poisoning and it's subsequent morbidity and mortality in early childhood in most developing countries as Africa, India, Iraq, and other parts of the world, where it is extensively used for lightening, cooking, and heating (Mitchell, 1984; De-Wet, 1994; AL- Azzawi, 1998).

Hydrocarbons: Hydrocarbons (petroleum distillates) represent a wide array of chemical substances contained in thousands of commercial products include: gasoline, Kerosene, benzene, lamp oil, Lighter fluid, many paint thinners and removers, and certain furniture polishes and cleaning agents (George, 2000; Bruce, 1986). All these products are fat solvents and alter the function of nerves producing depression, convulsion, and coma

Pathophysiology: Toxicity from hydrocarbons is related to one or both of the following:

Local Toxicity: which involve chemical burning of the mouth, throat ,and defatting of the skin and more seriously the lung (George, 2000; James, 1984)

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Systemic Toxicity: which involve the central nervous system andvisceral involvement with gastrointestinal symptoms. Liver toxicity, cardiomyopathy and renal toxicity."

Pulmonary involvement: Aspiration into the respiratory tract constitute the major hazards of accidental ingestion of hydrocarbons(4:8,9) And almost all deaths secondary to hydrocarbon compounds are due to respiratory complications Toxicity of hydrocarbons is related to their viscosity, generally products with high viscosity and low volatility are not toxic when ingested?). However products with low surface tension and low viscosity and high volatility have a high risk of causing pulmonary symptoms, 9,10) Halogenated hydrocarbons or those with toxic additives have the greatest systemic toxicities and should be removed by lavage As in Table 1.

Table No. I Acute Hydrocarbon Risk Assessments (10)

Systemic Toxicity Common : Trichloroethane (spot remover). Trichlorethylene, Carbon tetrachloride; inethylene chloride, benzene, hydrocarbon additives (camphor, heavy meatals, insecticides, aniline), taluene.-Local toxicity by Aspiration common, systemic toxicity uncommon :Mineral seal oil, signal oil, furniture polish, turpentine, gasoline, kerosene, charcoal ligher fluid, taluene.

Non toxic in 95% of cases : Asphalt, tar, motor oil, meniral or liquid petroleum lubricants, body oil. The pulmonary involvement after hydrocarbon ingestion is a result of aspiration and inhalation during deglutition and spread from hypopharynx to the trachea, rather than due to gastrointestinal absorption of the chemical (James, 1984; Arnold H.Einhorn, 1987). Several authors had stressed as well on vomiting after ingestion of hydrocarbon compounds as an important factor leading to aspiration into the lung (AL- Azzawi, 1998; George, 2000; Bruce, 1986) Hydrocarbons in the lungs appear to cause diffuse acute alveolitis, necrotizing bronchopneumonia, interstetial inflammation and pulmonary oedema, these pathological changes last from 3-10 days and may be followed by amore chronic inflammatory response that gradually resolved over a period of several weeks. The ventilation perfusion abnormalities seen may also be caused by surfactant damage leading to alveolar collapse and early distal airways closure (Mitchell, 1984; James G.Hughes, 1984).

Central Nervous System Involvement: All hydrocarbons are central nervous system depressant (!). The amount of absorbed hydrocarbons determine the degree of central nervous system depression. Although central nervous system involvement may be due to both systemic and direct toxicity on the brain, cerebral hypoxia secondary to respiratory insufficiency seems to play the major part (Robert, 1980). Necrotizing effects on the central nervous system are more likely to be produced by hydrocarbons of low molecular weight and lower viscosity than kerosene such as: benzene, gasoline, mineral spirit (Lonard, 1996; Arnold H.Einhorn, 1987). Majeed, HA et al stressed that all patients with central nervous system complications had sever pulmonary involvement suggesting a close relationship between the severity of pulmonary involvement and the development of neurological complications.

Visceral Involvement: Aromatic and halogenated hydrocarbons are also absorbed following ingestion and are toxic to the liver, heart, and kidneys [(11)] Carbon tetrachloride can produce hepatotoxicity. A number of volatile hydrocarbons can cause myocardial sensitization with risk of dysrrhythmia and sudden death (George, 1986). Renal tubular acidosis, renal failure and toxic nephropathy can occur with hydrocarbon poisoning (Linden, 1998; AL- Azzawi, 1998). Aromatic hydrocarbons also can cause bone marrow suppression and skeletal muscle damage (1) Nitrobenzene, aniline, and related compounds produce methemoglobinemia (4) and other hydrocarbon may rarely cause intravascular hemolysis and dissiminated intravascular heinolysis (AL- Azzawi, 1998).

Clinical Features

Early on, there is burning in the mouth and throat, chocking andgagging, coughing, nausea, vomiting and hemoptysis, coma (Lonard, 1996). Tachypnea, nasal flaring, retraction, grunting, tachycardia, and sometimes manifestation of cardiac decompensation reflect the gravity of pulmonary insult (Linden, 1998). Symptoms of central nervous system depression which include "general weakness, hypotonia, dizziness, mental confusion, Lethargy“ Irritability, agitation, or convulsion may be seen (Lonard, 1996). Abdominal pain, hepatitis, renal tubular acidosis, acute hepatic or renal failure and rhabdomyolysis. Sudden deaths due to myocardial irritability and ventricular fibrillation may occurfollowing hydrocarbon sniffing (Linden, 1998).

Late sequale, might include weakness, weight loss anaemia , peripheral numbness and parasthesias, psychosis, cerebral and cerebellar atrophy and encephalopathy may occur (Robert, 1980; James, 1984; Lonard, 1996; Arnold, 1987; Steven, 1998; Linden, 1998).

Kerosene Pneumoniti : Coughing and vomiting following ingestion almost immediately. Within hours there may be temperature elevation (38-40°) (David, 2000), with less extensive aspiration the onset of pulmonary symptoms and inflammation may be delayed 12-24 hr (David, 2000; William, 1987). The child may be dyspneic, transiently cyanosed with diminished resonance on percussion, suppressed or tubular breath sounds and crackles (David, 2000; Norman, 1974).

Chest X-Rays : Pneumonic involvement is disclosed more frequently by roentgenographic examination than by physical finding, It may show minimal changes a few hours after ingestion but may progress rapidlyafter that with extensive infiltrates (David, 2000). Chest x-ray might show as well areas of punctate or mottled densities in the perihilar region which extend into midlung field. Basal pnumonitis and atelectasis are also common and may coalesce to produce a picture of consolidation (Norman, 1974). Air oesophageogram, gaseous distension of the stomach and double gastric fluid levels in the upright x-rays were also described (David, 2000; Norman, 1974). Complications as pneumothorax, subcutaneous emphysema, pleural effusion including empyema have occurred, after the first week (David, 2000; Norman, 1974), Pneumatocele may form after 2-3 week after aspiration (George, 2000; David, 2000; Norman, 1974; John B.Campbell, 1970; Bray, 1998). There is a poor correlation between physical findings and radiological abnormalities (Lonard, 1996; Norman R.Eade, 1974). In most cases radiological changes resolved completely within 10-14 days (Annobil, 1983; Annobil, 1991).

Laboratory Investigations: Accompanying leukocytosis may be misleading because in most cases of aspiration pneumonitis no bacteria are present in the lungs (George, 2000). Pulmonary function test in a symptomatic patient a few years after the pulmonary insult show that small airways (<2mm) abnormalities with or without loss of elastic recoil had occurred, and these abnormalities seem to be related to the severity of the acute insult. These patients may be at risk for the development of chronic lung disease as adults when they are exposed to exogenous factors such as air pollution or smoking (Norman R.Eade, 1974; Dennis Gurwitz, 1978; Tal, 1974).

Treatment Hospitalization: Patients who are symptomatic when they are first examined or during 6 hours of observation, those with abnormal chest x-ray, and all patients who ingested a particularly toxic agent (furniture polish) should be admitted to hospital for an average period of (3-5) days (David, 2000; William, 1987; Norman, 1974). If the child is symptomelessinitialy and with normal chest x-ray 6 hours after ingestion the child can be observed at home but parents should be instructed to bring him if any respiratory symptoms appear (Norman, 1974). Treatment is only supportive. Fever and dyspnea may last up to 2 weeks in sever cases but with oxygen, respiratory physiotherapy and caloric intake most children will recover completely. Antibiotic: are indicated only if secondary bacterial infection is confirmed (David, 2000; William, 1987).

Elimination of Hydrocarbons

After ingestion of small to moderate amounts of hydrocarbons induction of vomiting or gastric lavage is contraindicated because of the risk of aspiration especially if several hours have elapsed (Lonard, 1996; Linden, 1998; David M.Orenstein, 2000; Norman R.Eade, 1974; Richard, 1997). If large volume of hydrocarbons is thought to be in the stomach and if an endotracheal tube with balloon cuff can be inserted without inducing vomiting nasogastric suction performed with great care to avoid aspiration is indicated (David, 2000).

Antimicrobial Therapy; The routine use of antibiotic is not recommended (David, 2000; Reed, 1997). The occurrence of secondary bacterial infection can be detected by the reappearance of fever on the 3rd, 5th. day after ingestion while penicillin and tobramycin might prove adequate therapy for most cases (David, 2000). In certain situations the addition of antimicrobials effective against anaerobes like metronidazole is indicated (Singh, 1992).

Adrenocorticosteroids :

Corticosteroid have no benificial effect on the course of the illness and may be harmful [11,14,15,15,25,28].

Venilation and Oxygen Therapy : Continuous positive airway pressure and other forms of ventilatory support are important parts of therapy especially in complicatedcases [14,16,29].

Extracorporial membrane oxygenation might also be life saving [25,30].

Prognosis: Most children survive without complications or bad sequale [15,14]. Some progress rapidly to respiratory failure and death. The prognosis depend on the volume of ingestion or aspiration and adequacy of medical care[14].

Prevention: Measures necessary to prevent kerosene should be stressed repeatedly by education personale to medical students and the public[31]. Kerosene should be kept in a safe place and containers out of reach of young children [1,16,31].

Aim of the Study

The aim of this study is to evaluate children presented after kerosene ingestion clinically and radiologicaly according to. time of presentation

PATIENTS AND METHODS

Sixty four children admitted to AL-Klransa Paediatric teaching Hospital in Mosul after kerosene ingestion during the period from the 1st. of July 1999 to the 31 St of December 1999 were studied prospectively .All children were admitted to the casualty unit at least six hours for observation. History was taken from the parents on a specially designed questionnaire appendix .Family size, maternal occupation and kerosene storage were evaluated .Complete physical examination was performed in every patient concentrating on symptoms of cough, dyspnea, cyanosis, dowsiness, vomiting, and signs of consolidation .CXR was taken for every patient six hours after arrival .Total white blood cell counts were done for 24 patient within the 1st (24 hours) of admission

Forty eight patients necessitated hospital admission because of their clinical and /or radiological abnormalities. Daily follow up for these patients was carried on and their temperature, clinical course, complications during hospitalization and the time needed for recovery were evaluated.

RESULTS

Epidemiological aspect: Age of the sample ranged between nine months and ten years with clustering of cases from one to three years (mean 2.5 years), Fig 1 .Male to female ratio was 3:1. Most cases were from urban areas 35 (54.7%). 48(75%) patients were considered from poor families and 44(68.8%) children from crowded families (live with six and more person in the family) .In 60 (93.7%) patients the mothers were full time housewives. Kerosene stored in barrels in 18(28.1%) cases, and in the rest in small containers which include small cans milk or soft drink bottles, kettle and drinking glasses as in table II .No family gave an accurate clue about the volume ingested but most of them claimed small amounts .

Clinical Features: Cough was the leading symptom in 55 (85.9%)patients Dyspnea occurred in 51(79.7%) patients . Other clinical features which appeared within 6 hours of ingestion included vomiting, kerosene odour, drowsiness, skin changes, as show in Table III .Diminished air entry, ronchi and crepitation were seen in minority of patients (17.2%) and mainly on the right side .One patient had progress to coma and death within few minutes from admission .The clinical course in admitted patients (48) was dominated by fever ranging from 38-39.5C° and continue for few days, constipation, abdominal pain and chest pain as shown in table. IV .Vomiting was found to have a significant association with the development of pneumonitis OR: 4.48 (P=0,01) table.V .White blood cell counts were done for 24(37.5%) patients. In 150f them the counts ranged from 12000-15000/ mm' and in the rest they were more than 15000 /mm).

Radiological abnormalities that appeared six hours after ingestion included basal infiltrates mostly on the right side in 40.6%, and bilateral perihilar infiltrate with clear bases in 21.9% (Table no.VI). All patients received oxygen on admission Antibiotics were were standard therapy in all admitted patients 48(75%) as ordered by the consultant in charge and these included procaine penicillin, ampicillin, and ampiclox .When a correlation was made between types of antibiotics used and duration of fever in the admitted patients no significant differences were shown as demonstrated in tab. VII.

DISCUSSION

Kerosene poisoning is still considered a significant cause of accidental poisoning in young children in Iraq and other developing countries with subsequent morbidity and mortality (1,3,32,33). About 47 (73.4%) of our patients were between one to three years of age which agree with other studies (3,26.32,34)The preponderance of males that is seen with kerosene and other accidents and poisonings is mostly because boys are more curious and active than girls of the same age [3,92,93]. In our study 44(68.8%) and 48(75%) patients were from crowded and poor families respectively which agree with other studies [32,33]

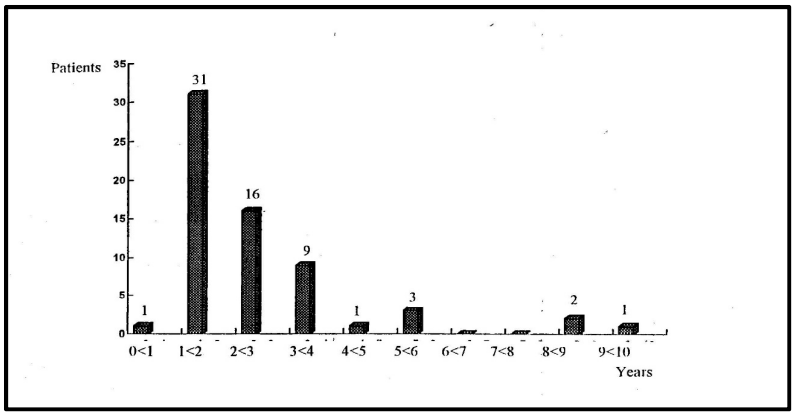

Fig. 1. Age distribution of 64 children with kerosene poisoning admitted to casuality unit

Table 2. 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	House wife	60	93.75
	Worker	4	6.25
Kerosene storage	Small container	46	71.9
	Barrels	8	28.1

Table 3. 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 4. Symptoms and signs that appeared after 6 hours No of affected patients

Symptoms and signs	Noof affected patients	%
Fever	.32	50
Constipation	18	28.1
Abdominal pain	10	15.6
Cost pain	1	1.6

Table. 5. 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)

Table 6. 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 7. 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]

Most of our patients 35(54.7%) were urban in parallel with study done by Singi-S.et al [35]. Kerosene was kept in a small unsuitable containers for storage in 46(71.9%) case which agree with other studies [26,33,36]

The most common presenting symptoms in our patients were cough in 55 (85.9%) patients, shortness of breath in 51 (79.7%) patients and vomiting in 41(64.1%) patients which agree with other studies (8,11,32)Drowsiness 13(20.3%) patients, 9(14%) of them were cyanosed at presentation and subsequently proved to have sever pulmonary involvement which indicate that probably hypoxia resulting from pulmonary involvement is responsible for drowsiness rather than the direct toxic effect of kerosene on the central nervous system, a result similar to that stated by other studies.(3,12,92) Fever in 50% of patients and leukocytosis were also evident which agree with other studies[3,4,32]. Although majority of cases (85.4%) who developed pneumointis in this study had vomiting shortly after ingestion of kerosene this was not the case in other 13 patient who also developed pneuinonia without history of vomiting · Kerosene aspiration therefore can occur as well during ingestion of the material which has been reported also by others [7,9,34], although the risk is much increased by vomiting [X2 = 6.520 95% C.I. =1.43 ~ 14) [3,4,9,32]

Constipation in 18(28.1%) patient and abdominal pain in 10 (15.6%) appeared later similar to other studies [3,4,32] Diarrhoea didn't develope in any patient compared to other studies [12,32] Non of our cases developed convulsions although it has been reported as complication in literatures [32,34], consolidation and were diagnosed by radiological examination this demonstrates the superiority of X.R. examination over the clinical diagnosis in kerosene pneumonia and this was supported by other studies. [8,16]The radiological findings agree with other studies [3,32,37], and composed mainly of right basal involvement, bilateral perihilar infiltration and left basal in 26 (40.6%), 14 (21.9%), 8 (12.5%) patients respectively. The involvement of the right lung base in majority of cases may be due to direct communication with trachea .The most frequent complication to aspiration pneumonia found in the study group was pleural effusion which developed in 3(4.7%) patients two of them were in the right and the other was left sided. Pleural effusion is the most common complication also in the studies of ALAzzawi and Fagbule, et al, [3, 36]. Chest tube drainage was not required for any patient .Pneumatocele and pneumothorax were not seen in the study group. All patients were treated by supportive measures which include oxygen, intravenous fluids and antipyretics .Although the role of antibiotics in kerosene pneumonitis is denied by the majority of authors [3,5,7,14,26,52], the practice in hospitals may be different. Our patients had recieved different antibiotics consisting mainly of procaine penicillin, ampicillin, and ampiclox and the statistical analysis revealed the role of antibiotics in decreasing the duration of fever is not significant (p> 0.05) and therefore these data support the generally accepted fact that antibiotics are not necessary in routine care of patients with kerosene pneumonitis unless secondary bacterial infection has been proven. All patients improved clinically and radiologicaly within 3-10 days although complete radiological recovery did not occur until 12, 28 days in (60%) of patients.

Conclusion

- One should not depend on clinical finding only for exclusion ofkerosene pneumonitis; radiological examination is necessary.
- No patient should be discharged before six hours after ingestion ofkerosene because developement of pneumonitis might be delayedseveral hours.
- Vomiting is a risk factor for development of pneumonitis; therefore,health workers should stress this point on people.
- Antibiotics should not be used unless secondary bacterial infection issuspected

Recommendation

Public health education through T.V., radio, newspapers, and other measures should concentrates on dangers of storing kerosene in small containers particularly those which might be used for storage of water or soft drinks. Through these measures kerosene ingestion as an accident meight be largely minimized or prevented.

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Appendix

questionnaire used for collecting data from kerosene poisoning patients：

Name----------

ANT

Age ---------- Sex----------

Date of admission：

Residence：

rural urban

Family Size:

Small < 6 · Large 6 or more

Mother- :House Wife Worker

Kerosene Storage <Special tanks Small containers

Amount ingested if known: Presentation: cough dyspnea cyanosis drowsiness grunting coma & others Vomiting spontaneous induced

Physical examination: Chest X-ray after 6 hours of admission: W.B.C. count (white blood cell count): Symptoms and signs that appear later on (more than 6hr): Management: hospitalization not oxygen, antibiotics, others

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