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Clinical Profile and Outcome of Accidental Paraffin Ingestion in Children Under 5 Years as Seen at Kalafong Hospital

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Abstract

Background: Young children are particularly susceptible to the ingestion of poisons, especially liquids, because they are very inquisitive. Most of the admitted children after accidentally ingesting poisons are asymptomatic and remain so during the admission period. However, there are no studies indicating the admission criteria of children with accidental paraffin poisoning taking into consideration time lapse post-exposure and the population at risk for mortality. This study set out to describe those patients not at risk to reduce the number of unnecessary admissions.

Methods: This was a one-year prospective descriptive observational study at Kalafong Provincial Tertiary hospital from March 14th, 2017 to February 27th, 2018. Children with a diagnosis of accidental paraffin poisoning less than 5 years at emergency department were recruited into the study. Questionnaire was used to gather information. For discrete variables, Fisher's exact test was used to compare groups. Continuous variables, student two sample T test was employed to compare groups.

Results: A total of 52 children were recruited with accidental paraffin ingestion poisoning. The peak age group was 1 to 3 years with a mean of 22.7 months. Male predominance (61.5%) was noted with a ratio of 1.6:1. There was a seasonal preponderance to summer 18/52 (34.6%). Cough (71%), vomiting (71%), dyspnea (38.4%) were the dominant symptoms with (34/52 (65.4%) induced vomiting at home. The prevalence of chemical pneumonitis was 17.3% with no mortality. Predictors of development of chemical pneumonitis were hypoxia, respiratory distress, crepitations, wheezes, tachypnoea in the presence of significant fever of ≥ 38°C (p-value <0.05). Presence of respiratory distress, crepitations or wheezes, chemical pneumonitis and HIV status are the predictors of longer duration of hospitalization with (p-Values of 0.012, 0.001, 0.002 and 0.034 respectively). Other observed reasons for extended hospitalization were fever developing in the ward and children admitted on oxygen. Most patients 43 (82.7%) presented to hospital within 4 hours of ingestion. Over half 30 (57.7%) of patients were discharged from the hospital within first 24 h of admission.

Conclusion: There is a need for a strategic planning with parental awareness programs to reduce paraffin ingestion poisoning and complications associated with prehospital care. Presence of respiratory distress, crepitation or wheezes; hypoxia in the presence of fever ≥ 38°C were associated with the development of chemical pneumonitis therefore is mandatory to admit these children. Respiratory distress, crepitations or wheezes, chemical pneumonitis, HIV status and parental age are the predictors of longer duration of hospitalization. We recommend that all children with paraffin ingestion poisoning should be observed for 6 h post-ingestion in emergency unit and if asymptomatic can be discharged with information on warning signs. Effective triage would likely cut down healthcare expenditure, duration of hospital stays, and effect on families of children with accidental paraffin poisoning.

Abbreviation and Acronyms

RR: Respiratory Rate; HR: Heart Rate; SATS: Saturations; TEMP: Temperature; GCS: Glasgow Coma Scale; AVPU: Alert, Verbal, Pain, Unresponsive; HIV: Human Immunodeficiency Virus; HEU: HIV-Exposed Uninfected; HUU: HIV-Unexposed Uninfected; ICU: Intensive Care Unit; LOS: Length of Stay; TESS: Toxic Exposure Surveillance System: ALI: Acute Lung Injury: DAD: Diffuse Alveolar Damage: ARDS: Acute Respiratory Distress Syndrome; KPTH: Kalafong Provincial Tertiary Hospital; SAM: Severe Acute Malnutrition; RSV: Respiratory Syncytial Virus; ADMIN:

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Admission

Research Problem

Young children are particularly susceptible to the ingestion of poisons, especially liquids, because they are very inquisitive. It has been observed that in Kalafong Provincial Tertiary Hospital (KPTH) setting many children are admitted due to accidental paraffin poisoning diagnosis for observation for period of 12 h to 24 h as per essential drug list guidelines in South Africa. Most of the admitted children are asymptomatic and remain so during the admission period. However, there are no studies indicating the admission criteria of children with accidental paraffin poisoning taking into consideration time lapse post exposure and the population at risk for mortality. Admission of a child to hospital disrupts the family and exposes the child to a risk of contracting infection from the hospital. The aim for this study was to describe the clinical profile and outcome of accidental paraffin poisoning to minimize the number of admissions.

Literature Review and Motivation

Introduction

Accidental poisoning is an exposure to a toxic substance that can lead to injury or death in which the subjects (Especially children) have no intention to harm their bodies [1]. Paraffin is a refined oil obtained by distillation and purification of crude oil, petroleum or rock oil. It is a hydrocarbon, used as a source of fuel for cooking, heating and lighting [2]. Paraffin is a cheap fuel and easily accessible to children due to the use of storage containers that previously contain foods or drinks for human consumption. Exposure to paraffin can be in different routes including via oral and lungs. Children are very curious and explore everything with their taste, as result home and surroundings become a threat to children if potential toxic substances are not safely stored or packaged.

Epidemiology

Accidental poisoning is the second commonest cause of external injuries and admission to hospital in children 0 to 4 years after falls [3,4]. Paraffin poisoning is the commonest type of acute poisoning among children in developing countries accounting for more than 60% of poisoning events [5,6]. Paraffin poisoning has been largely eliminated in high income countries but is still a widely spread issue in middle- and low-income countries with rural areas most affected due to the increase use as an energy source [7]. Low- and middle-income countries have four times fatal accidental poisoning compared to high income countries [8].

Many children are victims of paraffin poisoning either through direct ingestion or through vapor inhalation, as it is mistaken for water. Paraffin poisoning is still one of the preventable health problems in children [2,4]. Toxic Exposure Surveillance System (TESS) in United States in 2008 reported 46,418 exposures to hydrocarbons, only 2,267 patients experienced moderate effects with chemical pneumonitis accounting for 20% of aspiration accidents in children [9].

The most observed cause of accidental poisoning is paraffin as observed in US, Bangladesh, Saudi Arabia, Jordan and Australia and India, however a study done in Pakistan showed a change in trend from paraffin (2010) to pharmaceuticals in 2012, this change in trend was attributed to urbanization [10-13]. Africa continent remains with a highest incident of paraffin poisoning due to the fact that paraffin remains the main source of fuel in most countries as observed in Nigeria and Kenya, [6,14] except in Egypt (2014) where Zig Zag pesticides were common in the major agricultural area [1,6,15-19]. In 2013 paraffin accounted for 2% to 37.5% case fatalities in Africa [15-19].

Paraffin is still the most common cause of acute accidental poisoning in studies on children in South Africa despite the campaign on the use of Child Resistant Containers (CRC) to store paraffin [1,15,20]. In 2006, it was estimated that there were 40,000 to 60,000 cases of paraffin poisoning per annum in SA [18]. In Mpumalanga paraffin followed by drugs remain the principal agents responsible for pediatric accidental exposures [15]. At Ga-Rankuwa Hospital in Gauteng province, paraffin poisoning accounted for 78% of all poisoning in 1992 [21]. At Red Cross Children’s Hospital and Tygerberg Hospital in the Western Cape paraffin poisoning accounted for 22% to 30% of all poisonings between 1999 and 2001 [19]. The high percentage of children with paraffin poisoning was explained by the difference in the socioeconomic status of the population. In 2008, 60% of pediatric accidental poisoning in South Africa was due to accidental paraffin ingestion [22]. The case fatality rate ranged from 0% to 1.6% in four provinces, namely Western Cape, KwaZulu Natal, Mpumalanga and Gauteng. A study done in 2013 in Tygerberg, an agricultural area like Zigzag in Egypt, pesticides are the leading cause of accidental poisoning in children [15,19].

Paraffin ingestion poisoning constituted on average over 20% of all poisoning cases seen at Red Cross War Memorial Children’s Hospital in Cape Town between 2003 and 2015 (1151 paraffin cases with 2 deaths) [18]. In comparison with the 1999-2001 periods there has not been reduction in admissions and mortality.

Risk factors

Children are at risk of accidental poisoning because of their developmental milestones and cognitive function. The most common affected group of children is that of less than 5 years with the mean of 2.8 years [1]. Children of this age group are more mobile and are beginning to explore the environment. Males are more affected than females with reported ratios of 1.3:1 to 1.7:1, attributed to cultural gender assigned role where boys play outside and girls stay indoors [10,15,23].

The most obvious risk factor is the presence of paraffin in the domestic environment [24]. Paraffin is the most frequently used source of energy for cooking after electricity. The absence of safe packaging legislation results in distribution of paraffin in indistinct and unlabeled containers. Paraffin is frequently stored in cool drink, milk and juice bottles, which children associate with beverages [20,23].

The unsafe storage of paraffin within reach of children has been identified as a risk factor for paraffin ingestion. The study by Krug et al. showed that 75% of children in the study who had access to the paraffin containers, was attributed to overcrowding and limited storage space [20,21,23].

The lack of or poor parental supervision is another frequently mentioned risk factor for poisoning [20,21]. The other risk factors include inexperience caregiver and educational level of the parent or caregiver [5,12,25]. It was demonstrated that children of young mothers who had only high school education were prone to have children involved in household poisoning [4]. The presence of another child or elderly >50 year, or larger family size of more or equal to four lowered the levels of supervision and predisposes the child to accidental paraffin poisoning [25]. Summer season is another risk factor for accidental poisoning in children because of increase in fluid intake due to warmer weather [15,20,21,26,27].

Clinical manifestations

The ingestion of paraffin may cause minor or no harm, however, complications due to poisoning could be lethal [28]. Most common clinical manifestations of paraffin ingestion related to respiratory system secondary to chemical damage to the lungs are chemical pneumonitis, fever, and respiratory distress [9,29,30]. Aspiration into the lungs causes pneumonitis with choking, cough, wheeze, breathlessness, cyanosis and fever. There are reports indicating that a small amount as little as 1 ml can results in chemical pneumonitis [23]. The respiratory symptoms and signs are common and have been reported in 51% to 80% of paraffin ingestion cases at two rural hospitals [23,31].

Fever is common and may be noted on presentation or may be delayed for up to 6 h. Gastrointestinal manifestations which included commonly vomiting, nausea and abdominal pains were also observed. Vomiting-whether spontaneous or induced-increases the risk of aspiration and chemical pneumonitis. Less common clinical manifestations were Central Nervous System (CNS) manifestations, which were headaches, irritability, drowsiness, seizures, ataxia and coma. CNS manifestations seen with paraffin ingestion are thought to be due to hypoxia and acidosis from damage to the lungs and not due to systemic absorption, as central nervous system complications are usually seen in association with pulmonary manifestations [32].

Complications

The clinical presentation ranges from mild to severe, with a case fatality rate of 0.72% to 2.1% [20,23]. Primarily paraffin ingestion poisoning results in pulmonary complications for which ingestion of even 1 ml is enough [12,33]. Paraffin damages type II pneumocytes compromising surfactant production and function. Paraffin aspiration leads to intra-alveolar hemorrhage, inflammation, and necrosis [5]. Chemical pneumonitis is the common manifestation following fever as the initial presentation caused by direct toxic injury to the pulmonary parenchyma and altered surfactant function by paraffin [5]. Most children remain asymptomatic and without complications following ingestion of paraffin. In approximately 15% of paraffin ingestions, aspiration pneumonitis occurs and evolves over the first 30 min up to 6 h to 8 h presenting with coughing, choking, gasping, dyspnea, fever, tachypnea, hypoxemia, and tachycardia. A symptom acme is reached within 48 h followed by progressive improvement within 2 to 5 days [34]. Up to 5% of pneumonitis cases progress rapidly to acute respiratory failure.

Severe complications like necrotizing pneumonitis and hemorrhagic pulmonary oedema occurs within 24 h to 48 h. Pneumatoceles may be uncommon complication during recovery period of chemical pneumonitis and results in prolonged hospitalization [28,34]. Acute Lung Injury (ALI) and Acute Respiratory Distress Syndrome (ARDS) are the clinical syndromes associated with chemical pneumonitis and the corresponding pathology is Diffuse Alveolar Damage (DAD). Other complications which are not commonly observed are convulsions and encephalopathy.

Investigation

Chemical pneumonitis occurs 1 h to 8 h of exposure in patients. Fever and leukocytosis also occur with chemical pneumonitis and do not indicate bacterial infections. Arterial blood gas may reveal hypoxia, hypercarbia, and metabolic acidosis. The most common radiological findings are infiltrates at the bases which may be visible as early as 30 min post-aspiration and they do not correlate with the clinical picture [1,9,27] but may be delayed. Baldachin et al. noted that the X-ray changes resembled those of non-segmental consolidation and atelectasis confined to the middle and lower zone in keeping with aspiration. Other X-ray abnormalities found include localized areas of atelectasis, consolidation, pleural effusion, empyema, pneumatocele and pneumothorax. Chest X-ray is not routinely indicated because it was found normal in 54% of cases [35].

Prevention

Paraffin ingestion poisoning occurs because of resemblance to water and improper storage [23]. In Australia, a blue dye is added to paraffin, which has dramatically reduced paraffin ingestion poisoning [13]. Prevention measures to reduce accidental paraffin poisoning include keeping poisonous agents out of reach of children in locked cupboards. A South African health economic study concluded that each home should be provided with child resistant containers [36]. Community education programs to raise the awareness of dangers of paraffin ingestion and advice on safe storage have also been suggested [37]. Other measures beyond change at home are educating caregivers on implications of paraffin poisoning and appropriate first aid techniques. Improvement of living conditions including jobs opportunities, alternative energy source for cooking and lighting will reduce paraffin ingestion poisoning.

Management

The management of accidental paraffin ingestion generally consists of observation, prevention or early treatment of complications and supportive therapy [38]. Majority of affected children were rushed by their caregivers to hospital within the first 4 h post-ingestion [9,30]. General measures include removing the child from the source of poisoning and remove all the contaminated clothing. First aid measures include maintaining the airway, breathing and refer immediately. The management of paraffin poisoning is symptomatic as there is no specific treatment or antidote available. Induced emesis or gastric lavage is contraindicated since it increases the risk of aspiration and chemical pneumonitis [21,39]. It is common for caregivers to induce vomiting after paraffin ingestion poisoning as a local belief that it will make patient better. Therefore, education on prehospital management of children who ingested paraffin is integral to all the other treatment.

Most guidelines recommend observation of asymptomatic children, but differ on the period of and where observation should occur. Dworking et al. in 2008 recommends that asymptomatic children should be observed for 6 h to 8 h in emergency unit, discharge if remain asymptomatic and review the following day [9]. In India, all children were observed over 24 h as per protocol, and were discharged without complications in several studies done [23]. There were no guidelines for admission and no clear observation period stated in studies done in Africa [6,17,16,40]. However, the South African standard treatment guidelines and essential medicine list 2017, recommends that asymptomatic children require observation for 6 h to 8 h.

The mainstay of treatment for symptomatic children is supportive. Children in respiratory distress and hypoxia require oxygen saturations evaluation, chest X-ray, oxygen therapy, anti-pyrexials and mechanical ventilation if necessary. The development of fever 48 h post-accidental paraffin ingestion maybe suggestive of superimposed bacterial infection [28]. Poor socioeconomic circumstances, nutritional deficits and underlying respiratory illness are observed risk factors of developing secondary infection resulting in prolonged hospitalization following paraffin ingestion as observed by Balme et al. [28,31]. There are controversies regarding prophylactic use of antibiotics. Malangu et al. reported antibiotics overuse especially amoxicillin (86%) which was higher than the average of 40.5% antibiotic use reported during a provincial survey [15]. The main reason for the use of antibiotics in these patients was to prevent secondary respiratory infections. Simmank et al. reported that secondary infections due to paraffin ingestion are uncommon and was supported by Reed et al. who recommend that antibiotics should not be routinely used in the management of paraffin poisoning unless if there is high index of superimposed infection or infection that develops 48 h post-ingestion [9,30,38,41]. Corticosteroid therapy is also not recommended since it reportedly increases the risk of superimposed bacterial infection. The majority of children are admitted asymptomatic and remain so until discharge [15,18,19,38].

Motivation or justification

Accidental paraffin ingestion remains a common reason for hospital admission in children and the burden of resources for hospitalization is felt by both the hospital and the family. Literature recommends admission of all asymptomatic children for a period ranging from 6 h to 24 h with accidental paraffin ingestion and we could not identify studies looked at possible admission criteria to limit unnecessary hospital admissions. This study will allow us to elicit the most common presentation, the severity of paraffin poisoning according to presentation, prehospital treatment, need for admission and the outcome. The outcome will reduce the burden of unnecessary admission by observing the exposed children at home. Currently there is 100% management of paraffin poisoning in hospital whether observation in emergency unit or the ward [6]. and 95% to 100% of children who are asymptomatic on admissions remain so at discharge.

Aims and Objectives

Aim

The aim of the study was to describe the clinical profile and outcome of the children under 5 years old who present at Kalafong Provincial Tertiary Hospital (KPTH) with accidental paraffin ingestion.

Objectives

Primary objective:

- To determine the prevalence of paraffin ingestion complications and what factors predict development of complications.
- To model the length of stay in relation to predictor factors.

Secondary objectives:

- To determine the clinical symptoms and signs children present with after accidental paraffin ingestion.
- To determine the average time, it take for children to present at emergency department after accidentally ingestion of paraffin.
- To determine the average time, it takes for child to develop symptoms after ingestion of paraffin.

Methods

The study was conducted from March 2017 to February 2018. This was a descriptive observational prospective study conducted at Kalafong Provincial Tertiary Hospital (KPTH) in Tshwane district. KPTH is a tertiary hospital that situated facility situated in Atteridgeville 12 km west of Pretoria. It serves an estimation of 2 million populations mostly from low socioeconomic class. All children with a diagnosis of accidental paraffin poisoning fulfilling the criteria at presentation at emergency department were recruited into the study. All patients were studied according to the characteristic clinical features of paraffin ingestion, clinical severity, home or prehospital treatment, hospital treatment including investigations, admission place and clinical outcome and follow up.

Inclusion criteria

- All children under 5 years old with accidental paraffin poisoning seen in casualty and pediatric ward were recruited.
- All those with informed consent were enrolled into the study.

Exclusion criteria

- History of existing illness.
- History of multiple poisonous agents' exposure.
- Non accidental poisoning.
- No definitive history of exposure to paraffin.
- Informed consent not granted.
- A questionnaire was used to gather information about the incident of poisoning and the background: The following information was collected using the questionnaire.
- Child demographic data: Age, sex, race, socioeconomic status, HIV status, and Nutritional status.
- Parents or Guardian information: Age, parity, marital status, race, socioeconomic status, health of a parent including HIV status, literacy.
- Information to paraffin exposure: Time lapse from ingestion to presentation, referral mode, time of ingestion, manner of exposure, seasonal variation, presenting symptoms, prehospital treatment (home), hospital treatment and outcome.

Data analysis

Microsoft excel worksheet was used to capture the data and was transferred to Stata 14 for analysis. For discrete variables, Fisher's exact test was used to compare groups and for continuous variables, student two sample T test was employed to compare groups. The accepted level of significance in this study was stated at a p value of <0.05.

Outcome measures

This was part of collected information on the questionnaire.

- Fever on presentation (Temperature ≥ 38°C).
- Chemical pneumonitis (Patients who present with tachypnoea or respiratory distress or crepitations in the lung).
- Decreased level of consciousness (AVPU or GCS score will be used).
- Length of stay.
- Discharge (home or death).

- Readmissions or development of symptoms after discharge.

Ethical justifications

All parents or guardians signed informed consent before participating in the study. The ethical approval to conduct the study was granted from the University of Pretoria, Faculty of Health Sciences Research Ethics Committee. The University of Pretoria Masters of Medicine Research Committee, the KPTH Chief Executive Officer where the study took place also gave permissions to conduct the study. The parent or guardian of the participants had the right to withdraw the child from the study anytime he/she wanted. All information obtained from the patients was registered in data capturing sheets of the study, which remain confidential. The study number was used to identify the participants and patients' identity remains anonymous. The risk for the study was negligible since only questionnaires were used to collect data. Treatment was not withheld if the child developed symptoms during observation period or if parents or guardian opted to stop participating in the study.

Results

During the period spanning from March 14th, 2017 to February 27th, 2018, they were 60 patients admitted with accidental paraffin poisoning at KPTH. Out of 60 patients 52 patients were recruited at Kalafong Hospital emergency department who fulfilled the criteria. One patient opted out of the study and seven patients did not meet the criteria because of the age, co morbidities and or consent not signed. Children's age ranged from 12 to 40 months with a mean of 22.7 months. Among 52 cases, (96.2%) of children were between ages 12 and 36 months with decreasing numbers after 36 months. Boys outnumbered girls (61.5% *vs.* 38.5%) with a ratio of 1.6:1. The age of carers ranged from 17 to 47 years with mean age of 27.6 years and rest of epidemiological characteristics are shown in Table 1.

According to social status, all patients belonged to middle and lower socioeconomic status as seen in Figure 1, but also representing the referral routes for KPTH.

Paraffin ingestion occurred mostly at home (80.8%) because it was routinely used for cooking, heating and lighting the house. This study revealed that only 57% of cases kept paraffin out of reach of children. This has important implication for health education. Regarding the storage of paraffin, in our study (90.4%) of cases used empty soft drink bottles. Table 2 shows that, 43(82.7%) presented within the first 4 h post-accidental paraffin ingestion. More than half (67.4%) of these patients presented within the first 2 h post-paraffin ingestion. Table 2 also shows that most children (89.1%) developed the symptoms within the first 30 min and only 4.3% who developed symptoms after 8 h presented with chemical pneumonitis.

Paraffin poisoning seen at KPTH occurs mainly during the summer months. The monthly distribution for the period of 1 year is shown in Figure 2, it shows the months where it is predominantly hot and children became thirsty increasing the risk for poisoning. Figure 2 Shows marked peaks during January, February, March, and September.

The peak hours of paraffin ingestion were at 09:00 am and 14:00 pm but took place throughout the day time as shown in Figure 3.

Prehospital care was associated with carers level of education predominantly with basic education as seen in Figure 4. The home

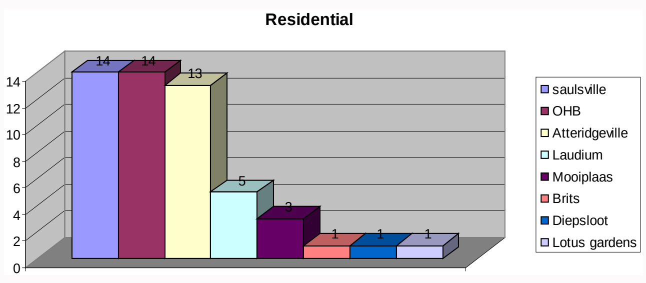

Figure 1: Residential address of recruited patients with paraffin poisoning.

Table 1: Sociodemographic characteristics of children and parents (n=52).

Sociodemographic characteristics	Frequency	Percentage %
Child gender		
Male	32	61.5
Female	20	38.5
Child age		
Infant (<12 months)	0	0
Toddler (12-36 months)	50	96.2
Preschool (37-59 months)	2	3.8
Race		
African	51	98.1
Coloured	1	1.9
Parent or Guardian age		
≤ 25 years	22	42.3
>25 years	30	57.7
Employment of mother/guardian		
Employed	21	40.4
Unemployed	31	59.6
Education level of mother/guardian		
No Schooling	1	1.9
Primary education	8	15.4
Secondary education	35	67.3
FET	5	9.6
University	3	5.8
Marital status of mother		
Single	45	86.5
Married	6	11.5
Divorced	1	1.9
Occupants in the house		
<4 occupants	19	36.5
≥ 4 occupants	33	63.5
Residence		
Sausville	14	30
OHB	14	30
Atteridgeville	13	25
Laudium	5	9.6
Mooiplaas	3	5.8
Brits	1	1.9
Diepsloot	1	1.9
Lotus gardens	1	1.9

Table 2: Epidemiological characteristics of paraffin.

Characteristics	Frequency	Percentage
Season		
Summer	18	34.6
Autumn	12	23.1
Spring	12	23.1
Winter	10	19.2
Place of poisoning		
Home	42	80.8
Neighbor	9	17.3
Others (nanny's house)	1	1.9
Site of storage		
Locked or unlocked cupboards	15	28.8
Top of cupboards	15	28.8
Reachable places	19	36.5
None	3	5.8
Type of container		
Empty soft drinks bottles	47	90.4
Paraffin jar or lamp	2	3.8
No paraffin at home	3	5.8
Paraffin use		
Cooking, heating, lighting	44	80.8
Cleaning	5	9.6
No use	3	5.8
Incident observed		
Observed	23	44.2
Not observed	29	55.8
Pre-hospital care	34/52	65.4
Mechanical Induced vomiting	10	29.4
Milk	28	82.4
Cooking oil	5	14.7
Referrals		
Clinic	17	32.7
Self-referral	35	67.3
Hours lapse prior presentation		
≤ 4 hours (≤ 2 hours)	43(29)	82.7(67.4)
> 4 hours	9	17.3
Minutes lapse prior development of symptoms (n=46)		
≤ 30 minutes	41	89.1
>30 minutes ≤ 8 hours	3	6.5
>8 hours	2	4.3
Asymptomatic	6	11.5

ailments used for prehospital care in this study was milk ingestion (82.4%), mechanical induced vomiting (29.4%), and cooking oil ingestion (14.7%) as seen in Table 2.

In our study, there were no patients with HIV infection or SAM. Six patients (11.5%) remained asymptomatic for 12 h to 24 h of observation. In the symptom analysis of paraffin poisoning; the respiratory symptoms dominated the picture. The observed symptoms were cough (71%), vomiting (71%), difficulty breathing (38.4%), fever (28.9%) and drowsiness (21.2%) as seen in Table 3 and Figure 4, 5. Among 37 cases of vomiting, 70.1% were induced by carers and the remaining 29.9% were spontaneous.

Fever (62%) was the most common clinical sign followed by signs of respiratory distress (21.1%) and presence of crepitations

Table 3: Clinical profile of paraffin ingestion poisoning.

Symptoms/signs/complications	Frequency	Percentage %
HIV status		
Negative	16	30.8
HEU	5	9.6
HUU	27	52
Unknown	4	7.7
Growth		
Normal	47	90.4
Overweight	1	1.9
Underweight	1	1.9
Stunted and underweight	3	5.8
Asymptomatic	6	11.5
Coughing	37	71
Vomiting	37	71
Fever (on history)	15	28.9
Difficulty breathing	20	38.4
Drowsiness	11	21.2
Wheezing/crepitations	7	13.5
Fever ≥ 37.6°C	32	62
Respiratory distress	11	21.1
Hypoxia (Saturations <92%)	6	11.5
Chemical pneumonitis	9	17.3
Decreased level of consciousness & ataxia	1	1.9

Table 4: Treatment received by children with paraffin poisoning n=52.

Treatment	Frequency	Percentage %
No treatment (observation)	6	11.5
Oxygen	16	30.8
Nebulisation	1	1.9
Paracetamol	40	77
Antibiotics	2	3.9
Ventilation	0	0
Anticonvulsants	0	0

Table 5: Outcomes following paraffin ingestion poisoning.

Outcome	Frequency	Percentage
Significant fever ≥ 38°C on triage	5	9.6
Length of stay		
≤ 24 hours	30	57.7
>24 hours	22	42.3
Discharge		
Home	52	100
Death	0	0

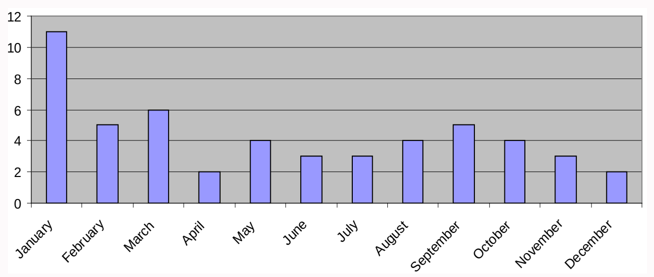

Figure 2: Distribution of monthly occurrence.

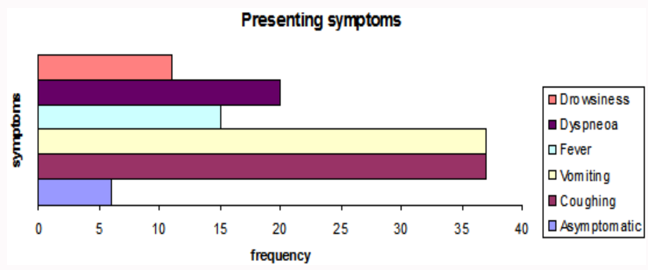

Figure 5: History-presenting symptoms of paraffin poisoning.

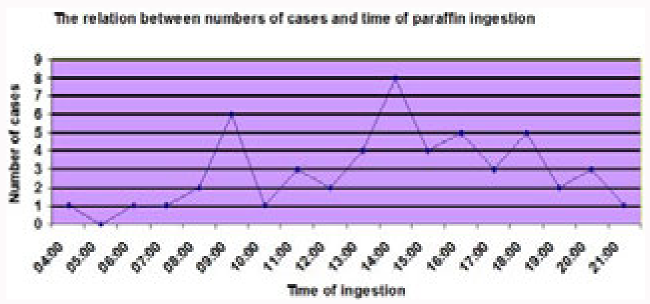

Figure 3: Number of cases and number of paraffin ingestion.

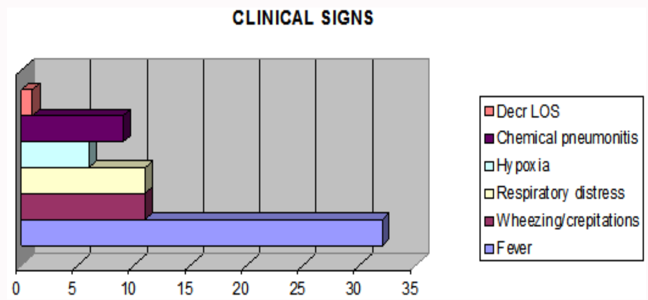

Figure 6: Clinical signs of paraffin poisoning.

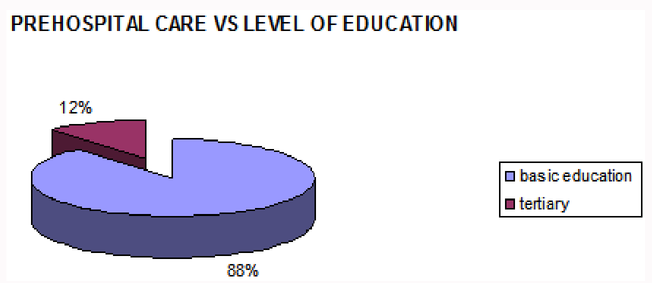

Figure 4: Relationship between level of education and prehospital care.

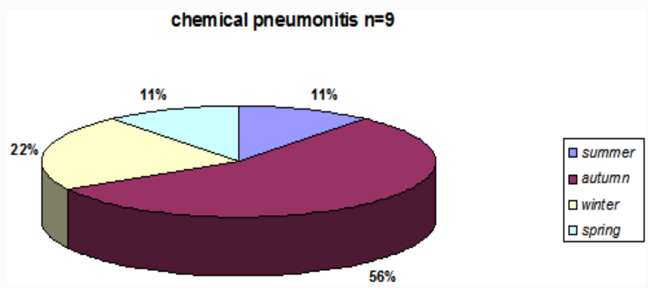

Figure 7: Seasonal occurrence of chemical pneumonitis.

or wheezes (13.5%) as shown in Figure 6. Chemical pneumonitis occurred in 9 (17.3%) patients and 6 (11.5%) had hypoxia. Regarding children with chemical pneumonitis (77.8%) of these patients required to be transferred from short stay ward to a general pediatric ward. There was only one patient with documented decreased level of consciousness and ataxia and recovered without complications. This patient also had hypoxia and chemical pneumonitis.

As seen in Figure 7, Chemical pneumonitis was most common in children who had paraffin ingestion poisoning in autumn in contrast with the seasonal peak of paraffin poisoning as noted in Figure 2.

In this study no patients required ventilation or intensive care. The majority of patients received paracetamol (77%) however 32 (62%) patients had documented fever. Oxygen was administered to 16 (30.8%) of patients and only 6 (11.5%) patients had documented hypoxia. Two patients received the course of oral antibiotics, amoxicillin 90 mg/kg/day was prescribed for both patients with suspected secondary bacterial infection. Other treatment modalities also included nebulization in one patient that was given in casualty see Table 4.

Complications of paraffin ingestion poisoning were analyzed in terms of development of chemical pneumonitis, ICU admission or need for ventilator support, readmissions and death. There were 5 (9.6%) patients who presented with significant fever ≥ 38°C at triage and 4/5(80%) of those developed chemical pneumonitis. Twenty-two (42.3%) patients had hospital stay of more than twenty-four hours. The case fatality rate was zero.

Children who presented with Significant fever of ≥ 38°C were associated with younger parental age and diagnosis of chemical pneumonitis with P-value of 0.036 and 0.026 respectively, as shown in Table 6.

Chemical pneumonitis

Table 7, shows the presence of fever of >38°C and crepitations or wheezes on chest examination at admission were significantly associated with chemical pneumonitis with p values of 0.001 and 0.002 respectively.

Length of stay

In this study, 57.7 % of patients had a length of stay of ≤ 24 h and 42.3 % stayed longer than 24 h with a mean of 32 h. The hospital stays for more than 24 h was associated presence of respiratory distress, crepitations or wheezes, chemical pneumonitis and HIV unexposed. Respiratory distress and presence of wheeze/crepitation was more significant during the stay in the ward with P-values of 0.005 and 0.001 respectively. Age (both parents and child) gender, fever, hypoxia and prehospital care and time lapse prior to presentation was

Table 6: Significant fever as a predictor of respiratory complications.

Variable	T<38°C	T ≥ 38°C	p-Value
Child's age in months (mean)	22.7	22.2	0.554
Parents' age in years (mean)	28.2	21.8	0.036
No. of occupants in the house(mean)	4.7	3.8	0.277
Hours prior presentation (mean)	3.3	7.2	0.067
LOS in hours(mean)	32	33.5	0.359
Chemical pneumonitis			
No	41	2	
Yes	6	3	0.029

Table 7: Predictors of chemical pneumonitis.

Variable	Absence of Hypoxia, respiratory distress, tachypnoea	Presence of Hypoxia, respiratory distress, tachypnoea	Total	p-Value
Fever				
No	18	2	20	
Yes	14	18	32	0.001
Creps/Wheezes (Triage)				
No	32	17	49	
Yes	0	3	3	0.052
Creps/Wheezes (Adm)				
No	32	14	46	
Yes	0	6	6	0.002
Creps/Wheezes (Ward)				
No	30	15	45	
Yes	2	5	7	0.092
Gender				
Male	20	12	32	
Female	12	8	20	1
Prehospital care				
No	11	7	18	
Yes	21	13	34	1
Employment				
No	18	13	31	
Yes	14	7	21	0.575
Electricity				
No	10	9	19	
Yes	22	11	33	0.382
Grant				
No	6	5	11	
Yes	26	15	41	0.73
Referral				
No	23	12	35	
Yes	9	8	17	0.544
HIV status				
Exposed uninfected	1	4	5	
Negative	12	4	16	
Unexposed	16	11	27	
Unknown	3	1	4	0.166

Table 8: Predictors for duration of stay (LOS).

Variable	Length of stay (Hospitalization)			
	≤ 24 hrs	>24 hrs	Total	p-Value
RD (Triage) Vs LOS				
No	27	15	42	
Yes	3	7	10	0.075
RD (Adm)				
No	28	14	42	
Yes	2	8	10	0.012
RD(Ward)				
No	28	13	41	
Yes	2	9	11	0.005
Fever				
No	14	6	20	
Yes	16	16	32	0.249
Hypoxia (Triage)				
No	28	18	46	
Yes	2	4	6	0.382
Hypoxia (Adm)				
No	30	19	49	
Yes	0	3	3	0.07
Hypoxia (Ward)				
No	29	21	50	
Yes	1	1	2	1
Creps/Wheezes (Triage)				
No	30	19	49	
Yes	0	3	3	0.07
Creps/Wheezes (Adm)				
No	29	17	46	
Yes	1	5	6	0.072
Creps/Wheezes (Ward)				
No	30	15	45	
Yes	0	7	7	0.001
Chemical pneumonitis				
No	29	14	43	
Yes	1	8	9	0.002
Gender				
Male	16	16	32	
Female	14	6	20	0.249
Prehospital care				
No	10	8	18	
Yes	20	14	34	1
Employment				
No	20	11	31	
Yes	10	11	21	0.263
Electricity				
No	12	7	19	
Yes	18	15	33	0.576

Grant				
No	5	6	11	
Yes	25	16	41	0.495
Referral				
No	21	14	35	
Yes	9	8	17	0.767
HIV status				
Exposed uninfected	4	1	5	
Negative	13	3	16	
Unexposed	11	16	27	
Unknown	2	2	4	0.034
Hours prior presentation	3.2	4.3		0.802
Child age	22.7	22.6		1
Parents' age	27.9	27.3		0.343
Occupants in the house	4.5	4.7		0.811

not significantly association with a length of stay (Table 8).

Discussion

Paraffin ingestion poisoning is rarely associated with mortality but associated with morbidity. It remains a serious contributor to childhood poisoning in developing countries. This study shows that among the total of 52 cases of paraffin ingestion poisoning, 50/52 (96.2%) of children were between ages 12 and 36 months with decreasing numbers after 36 months (Table 1). Children between 1 and 5 years of age are more vulnerable to accidents because of their inquisitiveness and inability to differentiate harmful and harmless things. This trend was also confirmed by data from other sources [11,12,21,39,41,42]. The male predominance was observed and is the feature of most accidental paraffin poisoning in children [15,23]. The majority of recruited patients were black with one (1.9%) colored patient and was comparable to other data, and level of socioeconomic status and use of paraffin as a source of fuel amongst blacks [23]. The monthly distribution with predominance of hot season when children became thirsty is consistent with other studies [12,20,23,32,33,39,43].

Most paraffin ingestion poisoning was due to poor supervision which was comparable to previous data [20,44]. The observed risks for paraffin poisoning in this study were unemployment, low level of education and improper storage and packaging of paraffin (Table 2), which are comparable to other studies [23,20,33]. Poor socioeconomic status and inadequate house space were also reported as risk factors for paraffin poisoning in studies from Africa and Middle East [2,21]. We observed that caregivers had minimal support evidenced by majority being single, unemployed and with basic education that contributed to the risk for poisoning. Overcrowded families of more than 4 occupants with a mean of 4.6 was observed as a risk factor which was observed by Ellis et al. Incidence of paraffin poisoning is lower in children of working mothers at 40%. The most striking feature of this study was the use of empty soft drink or juice bottle as the container of paraffin which is the same trend with other studies reported even after campaigns on paraffin safety storage and provision of safety caps [21,23,21,44].

The highest rate (80.8%) of paraffin ingestion poisoning occurred at home (Table 2), this is consistent with findings in United States of America (USA) where 90% of poisoning occurs at home (CDC, 2002). The majority of patients presented in the afternoon (Figure 3) as observed in previous studies [2,45]. This was probably because of the temperature severity. The harmful prehospital care practices of using home remedies to induce vomiting are prevalent and are detrimental to health, occurred in 65.4% of children (Table 2), this was the common practice as noted by Reed et al. [21]. The prehospital care or induced vomiting was related to level of education (Figure 4), observed in 88.2% of caregivers with basic education and 11.8% cases of higher education. Parents should be warned about effects of induced vomiting because it precipitates aspiration and predispose children to pneumonitis. This study revealed that most patients (82.7%) presented to the hospital within 4 h of ingestion of paraffin (Table 2), indicating a fair level of awareness amongst the general population to seek medical care following such an incident at home and was observed by Matzopoulos in South Africa [44]. Two out of nine patients (22.2%) that presented after 4 h had chemical pneumonitis.

There was no association between HIV and Severe Acute Malnutrition (SAM) as there was not a single patient with such condition (Table 3), in a country where the prevalence of SAM and HIV is high. Majority of children in this study developed symptoms within first 30 min post-ingestion (Table 2) as observed by Gupta et al. [46] but may be delayed for up to 8 h as Matzopoulos observed [44]. Cough and vomiting were two major symptoms present in 37/52 (71%) as seen in Table 3 comparable to previous data [23,20,33]. Vomiting was previously attributed to mucosal irritation; but in our study majority were induced not spontaneous [47].

Vomiting whether spontaneous or induced, increases the severity of pneumonitis. It has been established that pneumonitis may follow gastrointestinal symptoms of paraffin [32]. Previous researcher had reported poor systemic absorption of paraffin from the gastrointestinal tract; therefore, respiratory manifestations are due to hypoxia and acidosis from injured lungs and not due to systemic absorption [32]. But an unexpected finding in our study was that vomiting was not significantly associated with chemical pneumonitis. Among nine cases of chemical pneumonitis seven patients (77.8%) correlated with induced vomiting but was statistically not significant (P=1.000) as seen in Table 7 in contrast to most studies which observed induced vomiting as a risk factor for aspiration [47,48].

In the current study twenty-one children (65.6%) developed fever during hospital stay. This finding shows that fever manifestation can be delayed as supported by previous data [44]. Only 5 (9.6%) children had significant fever of ≥ 38°C at presentation and 2/5 (40%) children progressed to chemical pneumonitis. All these manifestations were observed in previous studies [13,20,21,33]. In this study, six patients (11.5%) remained asymptomatic for 12 h to 24 h and was observed in previous studies [12,23].

Children in current study had predominant respiratory tract related clinical manifestations and it was similarly seen in other studies [13,20,21,33]. Chemical pneumonitis occurred in (17.3%) as seen in Table 3, and different studies reported 12% to 40% cases of chemical pneumonitis [12,42,39]. The unexpected finding of this study was the occurrence of chemical pneumonitis in autumn (56%) followed by winter (22%) as outlined in Figure 7, in contrary to the highest number of paraffin poisoning seen in summer. This period however is associated with an epidemic of Respiratory Syncytial Virus (RSV) in Gauteng which begins in late February and ends in August as stated by Green et al. [48]. No studies found describing the association between chemical pneumonitis and RSV infection. It is possible that some of the children diagnosed as chemical pneumonitis may have had RSV infection.

The supportive care given was oxygen, anti-pyrexial and antibiotics which are the most common supportive treatment (Table 4) [15,32,45,46]. The only 2 patients who received antibiotics in this study were suspected of secondary bacterial infection, as recommended in SA guideline 2017 and Kate Balme SA guideline [49]. This is a good antibiotic stewardship in KPTH with regard to patients with paraffin poisoning. There was no mortality reported in our study, as also reported by Hatti et al. [46] Gupta et al. reported (4.3%) case fatalities in India however other authors in South Africa reported case fatalities of 0.74% to 1.6% in KZN, Cape Town, Gauteng and Mpumalanga [15,18,19,23,20,47]. The zero mortality in our study may attribute to the early health seeking behavior by these communities after ingestion of paraffin.

In this study 42.3% patients stayed longer than 24 h with a mean of 51.2 h (Table 5). The reasons for extended stay were presence of chemical pneumonitis, fever developing in the ward or were admitted on oxygen without evidence of hypoxia. Presence of respiratory distress, crepitations or wheezes, chemical pneumonitis, HIV status and parental age are the predictors of longer duration of hospitalization with a significant ($P<0.05$), refer to Table 8. Majority of patients with respiratory distress had the duration of stay of ≥ 24 h with ($P=0.01$) in agreement with Dayasiri et al. [39]. The children with crepitations or wheezes stayed longer with ($P=0.001$), in agreement with Hatti et al. [46]. The HIV exposure was not associated with hospitalization for >24 h as most patients that stayed longer were unexposed ($P=0.03$). The duration of hospital stay and severity of complications have been shown to have a direct association with lag time in reaching the hospital following the paraffin poisoning event as observed by Basu et al. [5]. There was no association in this current study as majority of our patients arrived early. The presence of hypoxia, fever, gender, prehospital care, referral system or significant fever was not associated with prolonged length of stay.

Fever as predictor for development of respiratory problem was significant with young caregiver's age, mean age 21.8 years in relation to significant temperature of ≥ 38°C ($P=0.003$) as seen in Table 6. Chest signs as classified by the presence of hypoxia, respiratory distress, and tachypnea was observed to be predictors of chemical pneumonitis in the presence of fever ($P=0.001$), refer to Table 6. Jayashree et al. also stated hypoxia as a predictor of chemical pneumonitis [47]. The presence of crepitations or wheezes were significant signs to predict development of chemical pneumonitis with ($P=0.02$) as seen in Table 7, also supported by Hatti et al. [46]. The child's age, gender, growth did not have any effect on developing chemical pneumonitis or longer duration of stay.

Majeed et al. reported a close relationship between the severity of pulmonary involvement and the development of neurological complications [21,43,46]. There was only one patient who had depressed level of consciousness; this patient also associated chemical pneumonitis and hypoxia. We did not have enough number to confirm or refute this association. Other factors (e.g., a direct CNS paraffin toxic effect) may also play a role in the pathogenesis of CNS involvement [21,50].

Conclusion

Accidental paraffin ingestion poisoning was common among male toddlers in the ages between one and three years. Common presentation with paraffin ingestion is respiratory disease followed by gastrointestinal manifestations. Presence of respiratory distress, crepitation or wheezes; hypoxia in the presence of fever ≥ 38°C were associated with the development of chemical pneumonitis therefore is mandatory to admit these children. Presence of respiratory distress, crepitations or wheezes, chemical pneumonitis, HIV unexposed status and parental younger ages are the predictors of longer duration of hospitalization. Majority of children 44/46 (96%) who developed symptoms did so within 6/8 hour of ingestion of paraffin.

We recommend that children who present asymptomatic after 8 h of exposure do not need hospital admission, and those who present less than 8 h to be observed at least 8 h since ingestion has elapsed without developing symptoms. All children without symptoms should be discharged home with information on warning signs if develop fever, tachypnoea or difficulty in breathing to return immediately.

While use of locked cabinets, allocation of high storage spaces out of reach of children and childproof containers are an economic impossibility to these families, there are some public health measures that should be evaluated in these settings. There is a need for a strategic planning with parental awareness programs to reduce paraffin ingestion poisoning, practice of inducing vomiting and complications associated with prehospital care. Effective triage would likely cut down healthcare expenditure, duration of hospital stays, and effect on families of children with accidental paraffin poisoning.

References

1. Abbas SK, Tikmani SS, Siddiqui NT. Accidental poisoning in children. J Pak Med Assoc. 2012;62(4):331-4.
2. Siddiqui E, Razzak J, Naz F, Khan SJ. Factors associated with hydrocarbon ingestion in children. J Pak Med Assoc. 2008;58(11):608-12.
3. Nixon J, Spinks A, Turner C, McClure R. Community based programs to prevent poisoning in children 0-15 years. Inj Prev. 2004;10(1):43-6.
4. Manzar N, Saad SMA, Manzar B, Fatima SS. The study of etiological and demographic characteristics of acute household accidental poisoning in children-a consecutive case series study from Pakistan. BMC Pediatr. 2010;10(1):28.
5. Basu M. Sociodemographic factors, symptoms, complications and treatment of kerosene oil poisoning in children: A hospital based cross-sectional study. Sri Lanka J Child Health 2016;45(2):72-5.
6. Lang T, Thuo N, Akech S. Accidental paraffin poisoning in Kenyan children. Trop Med Int Health. 2008;13(6):845-7.
7. Peden M, Oyegbite K, Ozanne-Smith J, Hyder A, Branche C, Rahman A. WHO world report on child injury prevention. Geneva: World Health Organization, UNICEF, 2008.
8. Baqir H, Baig MA, Brown N, Mian AI. Accidental poisoning in young children: An emergency medicine perspective for Pakistan and other low- and middle-income countries and a call for action. Eurasian J Emerg Med. 2017;16(4):140-3.
9. Wax PM, Wong SC. Hydrocarbons and Volatile Substances: Introduction. Chemical intermediate. 2015;2:16.
10. Hassan BA, Siam MG. Patterns of acute poisoning in childhood in Zagazig, Egypt: An Epidemiological Study. Int Sch Res Notices. 2014:2014:245279.
11. Vasavada H, Desai P. Clinical profile and outcome of children presenting with poisoning (A Hospital Based Study). Alcohol. 2013;4(4):1-7.
12. Anwar S, Rahman A, Houqe SA, Moshed A, Yasmin L, Saleh A, et al. Clinical profile of kerosene poisoning in a tertiary level hospital in Bangladesh. Bangladesh J Child Health. 2014;38(1):11-4.

13. Sibert JR, Routledge PA. Accidental poisoning in children: can we admit fewer children with safety? Arch Dis Child. 1991;66(2):263-6.

14. Tshiamo W. Paraffin (kerosene)* poisoning in under-five children: A problem of developing countries. Int J Nurs Pract. 2009;15(3):140-4.

15. Malangu N, Du Plooy W, Ogunbanjo G. Paraffin poisoning in children: What can we do differently? S Afr Fam Pract. 2005;47(2):54-6.

16. Ugwu GI, Okperi BO, Ugwu EN, Okolugbo NE. Childhood poisoning in Warri, Niger Delta, Nigeria: A ten-year retrospective study. Afr J Prim Health Care Fam Med. 2012;4(1):5.

17. Al Hazmi AM. Patterns of accidental poisoning in children in Jeddah, Saudi Arabia. Ann Saudi Med. 1998;18(5):457-9.

18. Balme K, Roberts JC, Glasstone M, Curling L, Mann MD. The changing trends of childhood poisoning at a tertiary children's hospital in South Africa. S Afr Med J. 2012;102(3 Pt 1):142-6.

19. Veale DJH, Wium CA, Müller GJ. Toxicovigilance I: A survey of acute poisonings in South Africa based on Tygerberg Poison Information Centre data. S Afr Med J. 2013;103(5):293-7.

20. Krug A, Ellis J, Hay I, Mokgabudi N, Robertson J. The impact of child-resistant containers on the incidence of paraffin (kerosene) ingestion in children. S Afr Med J. 1994;84(11):730-4.

21. Shotar AM. Kerosene poisoning in childhood: A 6-year prospective study at the Princes Rahmat Teaching Hospital. Neuro Endocrinol Lett. 2005;26(6):835-8.

22. Kimemia D, Vermaak C, Pachauri S, Rhodes B. Burns, scalds and poisonings from household energy use in South Africa: Are the energy poor at greater risk? Energy Sustain Dev. 2014;18:1-8.

23. Ellis JB, Krug A, Robertson J, Hay I, MacIntyre U. Paraffin ingestion--the problem. S Afr Med J. 1994;84(11):727-30.

24. Arroyo A, Rodrigo C, Marron MT. [Toxicological evaluation in the childhood]. Med Clin (Barc). 2014;142 Suppl 2:43-6.

25. Ahmed B, Fatmi Z, Siddiqui AR. Population attributable risk of unintentional childhood poisoning in Karachi Pakistan. PloS One. 2011;6(10):e26881.

26. Oguche S, Bukbuk D, Watila I. Pattern of hospital admissions of children with poisoning in the Sudano-Sahelian North eastern Nigeria. Niger J Clin Pract. 2007;10(2):111-5.

27. Manikyamba D, Madhavi N. Clinical profile of poisoning in children admitted in a tertiary care centre. Mortality. Int J Sci Res. 2015;4:3.

28. Simmank K, Wagstaff L, Sullivan K, Filteau S, Tomkins A. Prediction of illness severity and outcome of children symptomatic following kerosene ingestion. Ann Trop Paediatr. 1998;18(4):309-314.

29. Paulsen R. The dynamics of paraffin use in a low-income South African community.

30. Wax P, Beuhler M. Hydrocarbons and volatile substances. 2004.

31. Reed RP, Conradie FM. The epidemiology and clinical features of paraffin (kerosene) poisoning in rural African children. Ann Trop Paediatr. 1997;17(1):49-55.

32. Abu-Ekteish F. Kerosene poisoning in children: a report from northern Jordan. Trop Doct. 2002;32(1):27-9.

33. Paswan W, Singh BB. Analysis the clinical profile of children admitted with kerosene poisoning in a tertiary care medical college hospital. Cough. 2017;86:82.

34. Bond GR, Pieche S, Sonicki Z, Gamaluddin H, Guindi ME, Sakr M, et al. A clinical decision rule for triage of children under 5 years of age with hydrocarbon (kerosene) aspiration in developing countries. Clin Toxicol (Phila). 2008;46(3):222-9.

35. Abdel Ghaffar N. Accidental poisoning of children presenting to emergency departments in Khartoum State. 2015.

36. Tormoehlen LM, Tekulve KJ, Nanagas KA. Hydrocarbon toxicity: A review. Clin Toxicol (Phila). 2014;52(5):479-89.

37. Ruas F, Mendonca MC, Real FC, Vieira DN, Teixeira HM. Carbon monoxide poisoning as a cause of death and differential diagnosis in the forensic practice: A retrospective study, 2000-2010. J Forensic Leg Med. 2014;24:1-6.

38. Madboly AG, Elgendy FS. Epidemiology, clinical characteristics, and management of acute hydrocarbons poisoning at Benha Poisoning Control Unit: A One-Year Prospective Clinical Study. 2011.

39. Dayasiri MBKC, Jayamanne SF, Jayasinghe CY. Kerosene oil poisoning among children in rural Sri Lanka. Int J Pediatr. 2017:2017:8798610.

40. Osaghae D, Sule G. Accidental childhood poisoning in Benin City: Still a problem? J Med Biomed Res. 2014;12(2):19-26.

41. Kondle Raghu PS, Kumar SS, Gopal S, Shaik MV, Ahammed B. Incidence and outcome of poisoning patients in a tertiary care teaching hospital. Asian J Pharmacol Toxicol. 2015;3(7):23-6.

42. Belonwu R, Adeleke S. A seven-year review of accidental kerosene poisoning in children at Aminu Kano Teaching Hospital, Kano. Niger J Med. 2008;17(4):380-2.

43. de Wet B, van Schalkwyk D, van der Spuy J, du Plessis J, du Toit N, Burns D. Paraffin (kerosene) poisoning in childhood is prevention affordable in South Africa? S Afr Med J. 1994;84(11):735-8.

44. Matzopoulos R, Carolissen G. Estimating the incidence of paraffin ingestion: Original contribution. African Safety Promotion. 2006;4(3):4-14.

45. Jayashree M, Singhi S, Gupta A. Predictors of outcome in children with hydrocarbon poisoning receiving intensive care. Indian Pediatr. 2006;43(8):715-9.

46. Hatti SS, Uplaonkar V, Honnalli C. Hydrocarbon poisoning in children: 1-year retrospective study. Indian J Child Health. 2018;5(3):193-5.

47. Jayashree M, Singhi S. Changing trends and predictors of outcome in patients with acute poisoning admitted to the intensive care. J Trop Pediatr. 2010;57(5):340-6.

48. Green RJ, Zar HJ, Jeena PM, Madhi SA, Lewis H. South African guideline for the diagnosis, management and prevention of acute viral bronchiolitis in children. S Afr Med J. 2010;100(5):320, 322-5.

49. Balme K, Stephen C. Paraffin ingestion in children: Rationalising antibiotic treatment. S Afr Med J. 2017;107(8):646.

50. Lifshitz M, Sofer S, Gorodischer R. Hydrocarbon poisoning in children: A 5-year retrospective study. Wilderness Environ Med. 2003;14(2):78-82.