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Childhood accidental poisoning among hospitalised children in a tertiary health care in North Central Nigeria - A two year prospective report

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Abstract: Background: The young child can be at risk of accidental poisoning because of its being very inquisitive to mouth objects. The prevalence and types of poisoning vary within Nigeria and different parts of the world.

Aim: To describe the sociodemographics, clinical features and outcome in childhood poisoning seen at the National hospital Abuja (NHA) Nigeria.

Subjects and Methods: A descriptive prospective study on children admitted for acute poisoning from September 2014 -August 2016. Consecutive children with a history of poisoning were recruited during the study period. The children were examined, and poison severity scores were recorded into a proforma, and followed up till discharge or demise.

Results: Twenty-two children were admitted for acute poisoning, out of the 2336 children seen during the study period, with a prevalence rate of 0.94%. Fourteen (63.6%) were of upper social class, with 12 (54. 5%) mothers having tertiary level education. The mean time (±SD) of presenta-

tion was 11.9 ± 23.9 hours, while the mean (SD) duration of hospital stay was 4.8 ± 6.2 days; hospital stay was significant with types of poisoning (Fisher exact test 22.062, p<0.0001).

The common poisoning agents were kerosene and organophosphate, 8(36.4%) each, while main clinical features were cough in 8 (36.4%), tachypnoea in 7(31.8%), fever in 6 (27.3%) and 8(36.4%) had home intervention.

Two (9.1%) and 4(18.2%) had poison severity scores (PSS) of 3 and 4 respectively, which was significant for time interval of presentation and use of harmful home intervention (Fisher exact test 3.697, p=0.024) and (Fisher exact test10.04, p=0.018) respectively. Fatality was 18.2%.

Conclusion: kerosene and organophosphate were most common poison agents, while PSS was related to time of presentation, home intervention and types of poisoning agents.

Key words: childhood, poisoning, hospitalized, outcome.

Introduction

Poisoning in children comprises a significant component of injury-related morbidity and mortality. In a WHO/UNICEF 2008 report on child injury prevention, poisoning accounted for 3.9per cent of global child injury deaths among 0-17year old. Poisoning refers to injuries that result from being exposed to an exogenous substance that causes cellular injury or death. Poisons can be inhaled, ingested, injected or absorbed. The exposure may be acute or chronic and the clinical presentation will vary accordingly. With the changing global trend of poisoning, the prevalence and types of poisoning are dependent on socioeconomic status and cultural practices, as well as on local industrial and agricultural ac-

tivities.1

The home and its immediate environment can expose the young child to risk of unintentional poisoning. Children in their innocence and curious nature tend to explore the environment imploring all their senses, including mouthing of objects for taste, which puts them at risk when poisonous substances are inadvertently ingested. The outcome depends on predisposing conditions and the quality of medical facilities available. Poisoning with specific agents produces clinical syndromes that are frequently recognizable; however, in some cases the features may be easily misdiagnosed or go unrecognized in a child. For this reason, treatment may be delayed, with serious consequences. Poisoning is therefore best prevented. Understanding the pattern of poi-

soning is helpful for reducing the risk of unintentional poisoning, as well as for preventing intentional poisoning².

Accidental childhood poisoning has been earlier reported as recognized causes of childhood morbidity and mortality in several Nigeria reports.³⁻¹¹ The developed world has accurate information on incidence and the changing trends of causative agents as a result of rigorous population census and mortality data and the development of Poisons Control Centres (PCCs). 12-14 The use of educational prevention programmes, child-resistant packaging and product reformulation have contributed to changes in incidence and causative agents in developed countries. 15, 16 In the United Kingdom, before the stringent regulatory policies, accidental childhood poisoning was a major contributor to Emergency Department presentation and hospital admission.¹⁷ In Australia, a decline in the incidence and mortality of childhood accidental poisoning has been reported with the institution of regulatory policies about 30 years ago. 18

The justification for this study was to describe our experience on childhood accidental poisoning with the changing socio-demographic of our society. Most of the earlier studies from Nigeria are over a decade ago, however none have been reported from our centre, hence this report will add to available data on the subject.

This study was undertaken to describe the sociodemographics, clinical features and outcome in childhood poisoning in a tertiary hospital in North central Nigeria.

Methods and Materials

This was a descriptive prospective study on patients admitted into the Emergency Paediatric Unit of National Hospital Abuja- Nigeria for acute childhood poisoning from September 2014 - August 2016. Consecutive children with a history of acute poisoning were recruited during the study period. Cases of food poisoning and drug reactions were excluded. The children were examined by a senior resident doctor on duty with a brief patient history and findings which were recorded into a proforma. Data obtained included patients' age and sex, parental level of education and occupation, family size, type of poison and intervention prior to presentation, time of admission, clinical features at presentation, outcome (discharge, death or leaving against medical advice {LAMA}) and duration of hospital stay. The socioeconomic class of the caregivers was determined using the Oyedeji 19 criteria and were grouped into socioeconomic status (SES) upper, middle and lower, and the family sizes were categorized into three, four and six, or more than six persons per family. The children were followed up till discharge or demise or leaving against medical advice.

All the patients seen were admitted irrespective of presence or absence of symptoms. Asymptomatic patients

were placed on observation of vital signs, urine output and consciousness level. Clinical severity was recorded according to the Poisoning Severity score (PSS). 20 The PSS is a classification scheme for acute poisoning in adults and children regardless of the type and number of agents involved. It is based on occurrence of symptoms and signs identified checked against the chart and the severity grading assigned to a case determined as: None (scored 0 for zero) when there was no symptoms or signs related to poisoning; minor scored as (1), when presenting symptoms were mild, transient and spontaneously resolving; moderate scored as (2) when symptoms were pronounced or prolonged; severe scored as (3) when symptoms were severe or life-threatening; and fatal scored as (4) when death occurred. Treatments were offered according to available poisoning substance specific guidelines of the department including use of activated charcoal. Ethical approval was obtained from the institutional review board of National Hospital Abuja, Nigeria. The information was analyzed using SPSS version 20. The analysis was mainly descriptive, and results presented as prose and tables. Pearson's

Chi-square was used to test for significance at a *P* value and set at a level of less than 0.05.

Results

Twenty-two children were admitted for acute poisoning, out of the 2336 children seen at the Emergency Paediatrics Unit (EPU) during the study period, giving a prevalence rate of 0.94%. Fifteen (68.2%) of the children were less than 2 years and 20 (91%) were 5 years and below. The mean age \pm SD; range in years was 2.25 \pm 1.86; 1.08 - 8 years; with males 14(63.6%), and females 8(36.4%), male: female ratio 1.75:1. The socioeconomic status of parents were 14(63.6%) of upper class, 6(27.3%) middle class and lower class 2(9.1%); while the family sizes were 6(27.3%), 3persons/family; 15(68.2%), 4-6person/family; and >6persons/family one (4.5%). Maternal education revealed that none had no education, while those with primary, secondary and tertiary education were one (4.5%), 9(40.9%) and 12 (54.5%) respectively.

The common poisonings were kerosene and organophosphate in 8(36.4%) each, and iron tablets in 2(9.1%) subjects as shown in Fig 1. The mean time (\pm SD) of presentation of the subject following poisoning was 11.9 ± 23.9 hours; range from 1 hour -96 hours.

The differences of the interval of presentation from time of occurrence of the various poisoning was not statically significant (F= 0.487, p=0.808) as shown in table 1. However, the difference in the duration of hospital stay with the various types of poisoning was significant (F=22.062, p<0.0001) as shown in table 1. The mean (SD) duration of hospital stay was 4.8 ± 6.2 days; range from 1-31 days and hospital stay of the subject were; 16 (72.7%) stayed 5days or less on admission and 6(27.3%) stayed more than 5days.

The most common clinical features included cough 8 (36.4%); reported in six subjects with kerosene poisoning and two subjects with organophosphate poisoning. Tachypnoea was the second most common feature in 7 (31.8%) followed by fever in 6 (27.3%) subject as shown in table 2.

Fig 1: Distribution of the Various Poisoning Agents

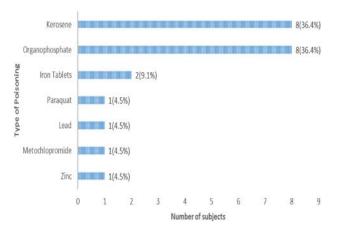


Table 1: The Interval of Presentation from Time of Poisoning and Duration of Admission of the Various Types of Poisoning

	Presentation interval	Duration of admission
	Mean (SD)	Mean (SD)
	(hours)	(days)
Kerosene	24.2(37.5)	4.0(2.2)
Organophosphate	5.1(5.4)	4.3(2.7)
Iron tablets	2.5(0.7)	1.5(0.7)
Paraquat	8	31
Lead	2	1
Metoclopramide	10	3
Zinc	2	2
F	0.487	22.06
P value	0.808	< 0.0001
P value	0.808	<0.0001

Table 2: Clinical Features of the Subjects with Poisoning								
Clinical features	Kero sene	Or- gano phos phate	Iron tab- lets	Paraq uet	Lea d	Me- toclo pram ide	Zin c	Tot al
	N	N	N	N	N	N	n	
Cough Tachyp-	6	2	0	0	0	0	0	8
noea	5	1	0	1	0	0	0	7
Fever Tachycar-	4	2	0	0	0	0	0	6
dia	3	1	0	1	0	0	0	5
Vomiting Abdominal	1	2	1	1	0	0	0	5
Pains	1	1	1	0	1	1	0	5
Weakness	2	3	0	0	0	0	0	5
Diarrhoea	0	3	1	0		0	0	4
Difficult								
Breathing	3	1	0	0	0	0	0	4
Seizures	1	2	0	0	0	0	0	3
Drowsiness	3	0	0	0	0	0	0	3

Harmful home intervention after the poisoning were reported in 8 (36.4%) of the subjects using the following; 5(22.8%) used palm oil and 3(13.6%) used milk drink. All the eight had induction of gag reflex by insertion of a finger into the throat to induce forceful vomit-

ing. The subject who had harmful home intervention stayed 7.2 (\pm 9.9) days compared with 3.4(\pm 2.3) days for those who did not (T test=1.407, df =20, p=0.175).

The Poison severity score were; 5 (22.7%) subjects with no symptoms or signs had PSS of none (zero), 7(31.8%) had PSS of mild (score 1), with transient or spontaneously resolving symptoms/signs; and severe/life threatening symptoms/signs (score 3) and death (score 4) were recorded in 2(9.1%) and 4(18.2%) respectively. The mean (SD) time interval between poisoning and presentation increase significantly with worsening PSS score (F= 3.697, p=0.024) as shown in table 3. The duration of hospital stay was not statistically association with the PSS (F2.243, p=0.107). There was a significant association between a worsening PSS and harmful home intervention as four of the subjects that died had harmful home intervention (Fisher exact test=10.04, p=0.018) as shown in table 4.

Seventeen (77.3%) were discharged, 4(18.2%) died and one (4.5%) left against medical advice. The mean duration (SD) of admission of those who died was 12.0(12.8) days and 3.2(2.1) days for those who survived (t test 2.966, df= 20, p=0.008).

Of the four deaths, two (50%) were males, both aged one year 3months old, following complications of kerosene poisoning (pneumonitis); one (25%), was a 4year old male who ingested paraquat (dipyridylium) a herbicide plant killer; and one (25%), was a one year 2months old female, who ingested an organophosphate insecticide.

Table 3: Distribution of poison severity score (PSS) among the children and the association of PSS with interval of presentation after poisoning and duration of admission

		C		
PSS	Sub- jects n(%)	Interval of Presentation mean (SD)	Duration of hospital stay mean (SD)	Poisoning Agent
0	5(22.7)	2.0(0.7)	1.4(0.5)	Zinc, Kerosene Metoclopramide tabs, Lead bat- tery, Kerosene,
1	7(31.8)	4.3(3.4)	3.4(2.5)	OP
2	4(18.2)	7.5(6.6)	3.8(1.3)	OP
3	2(9.1)	4.5(3.5)	6.0(1.4)	OP
				Kerosene, OP,
4	4(18.2)	45.5(45.5)	12.0(12.8)	Paraquat
F		3.697	2.249	•
P value		0.024	0.107	
		-		

Legend

PSS – Poison severity Score

- 0- No symptom/sign
- 1- Mild, transient or spontaneously resolving Symptom/sign
- 2- Pronounced/prolonged symptoms/sign
- 3- Severe/life threatening symptoms/sign
- 4- Death

OP- organophosphate

Table 4: Harmful Practice and Interval of Presentation, Duration of Hospital Stay and PSS								
Poison severity Score								
Harmful practice	Subject	0	1	2	3	4	Fisher exact	P value
Yes	8	2(40)	1(14.3)	0	1(50)	4(100)	10.04	0.018
No	14	3(60)	6(85.7)	4(100)	1(50)	0		

Discussion

The prevalence of childhood poisoning from the present report is 0.9percent. Compared to recent reports from the same environment, is higher than the 0.442% by Edelu et al ⁶ in a 10 years retrospective 2016 report, but lower than the 1.54% by Olatunga et al ⁹ in a 2 years retrospective 2015 report, and 3.3% by Dominic et al ⁷ in a 10years retrospective 2013 report. However, earlier reports by Adejuyighe et al (2002), ³ Ochiagho et al (2004),⁴ and Oguche et al (2007) ⁵ on childhood poisoning among Nigerian children were 0.52%, 2.2%, and 0.74% respectively. A study from an urban population in India, a similar developing country reports a prevalence rate of 0.5%. ²¹ These finding may be attributable to the changing trends of acute childhood poisoning with age groups, types of exposure, nature and dose of poison and regulatory polices over time. 12-18, 22

Children aged two years and below and males were more at risk for childhood poisoning, which has been earlier reported. 3,5-7 Age has been strongly associated with poisoning as it determines the behaviour, size and physiology of the child, thus influencing types of exposure and outcome. 2 Infants and small children tend to put their hands and small objects into their mouth; hence the young child has increased mobility and increased susceptibility to the unintentional poisoning. ² More male involvement is unintentional childhood poisoning have been commonly reported 4, 6-8, 22 which may be associated with greater attitude of socialisation and curious exploration. However, the report by Annu et al ²¹ on accidental poisoning did not show any sex predilection. This report showed that 54. 5percent of the mothers had tertiary level education and 63.3 percent of parents were from upper socioeconomic class. Earlier reports from Nigeria²⁻¹¹ showed unintentional childhood poisoning to be associated with a lower social class and low parental education. However, over 70percent of the parents in our report had four or more persons living in a room, a reflection of overcrowding in urban cities with limited housing facilities and the global recession that places more financial hardship on families even in the presence of higher social class. Poisoning profile is known to be influenced by the social, economic and cultural practices of the region.^{1,2} Other social factors associated with childhood poisoning that have been reported include inadequate and unsafe storage facilities, the presence of young parents and limited adult supervision. 1,2 18, 23 Some report showed that childhood poisoning occurred when a child was left in the care of another child or with a grandparent.^{23, 24} The report among educationally and economically advanced south India population with

high health indicators and advanced health care facilities, showed that accidental poisoning in children was common. ²¹

The main poison agents were kerosene and organophosphates which are common household agents for cooking and poison for rodents and insects in the home. Kerosene is a significant source of home energy and is frequently storage in bottles or other containers, such as empty bottle water and cups, which children associate with drinking water and beverages like milk and juices. 22-24 In a study report from South Africa on poisoning presenting to hospital in 1987 and 2008, kerosene remained the biggest single cause of poisoning agent responsible for almost 60% of new cases.²²In an earlier South African report,²⁴ the government launched a program to tackle the dangerous practice of kerosene used for cooking storage in bottles previously used for storing beverages. This involved the free distribution of containers with child-resistant closures. The result was a fall of the annual incidence of kerosene poisoning over a period of 14 months, from 104 per 100 000 to 54 per 100 000.²⁴ Organophosphates poisoning was accidental among the children in this study report, as it was a common household agent used to kill rodents and insects in the home. In some agricultural communities, organophosphate contributes significantly to poisoning especially among adult population.²⁵ The use of drugs in the present report was limited to pharmaceutical that can easily be purchased from over the counter for minor ailments to which the families have easier access. It is known that mothers brought their iron tablets from antenatal clinics in unsealed envelopes, or zip lock bags, and liquids in non-distinctive, poorly labelled containers.^{1, 26} However, we did not report the use of agents such as paracetamol and household cleaning solutions such as bleach and detergents, which are the common agents of accidental poisoning in advanced countries like the United Kingdom¹² and other developed countries. 23, 27 The physical appearance and attractiveness to children of these agents, especially clear liquids rather than solids and small solids appeal to young children and are therefore more likely to be ingested by them.²⁶,

Symptoms were mainly cough, tacypnoea, and fever, which could be related to the poisoning agents and worsens with harmful intervention use of palm oil and milk drink to induce vomiting, a finding reported in several Nigerian reports. ³⁻⁸ These actions provoke aspiration and results in complications such as chemical pnuemonitis. Majority of the subjects in the present study had poisoning severity scores (PSS) of zero and one, signifying non- effects of the poisoning or mild disease with hospital stay of five days and less. The mean time of presentation of the subject following poisoning was 11.9 ± 23.9 hours; range from 1 hour -96

hours. Annu et al similarly reported a PSS of zero and mild in 91(75.20percent) in their study population. ²² The outcome for poisoning depends on the poisoning agents and quality of medical facilities available. 1, 2, 17, 18 Although WHO reported that mortality rates are generally low for childhood poisoning, giving an estimate of 4 per 100 000 population. The rate of 18.2 percent in the present report is quite high, compared to the lower rates of 3.1 percent by Edule et al⁶ and 8.6 percent by Olatunya et al, but similar to the 20 percent by Ochigbo et al. The high fatality in this report was mostly related to the nature of poisoning agents, some with no known antidotes and complications of home interventions. Of the four case fatalities in the study, two were from kerosene poisoning with home interventions and its attending systemic complications. The other deaths were from parquet, an herbicide poison and an organophosphate (locally called ota- pia-pia) which are known to be deathly poisons with little chances of survival. Some countries have employed the blue colouring of kerosene to reduce its attraction as a drink.¹⁷ Standards and policies for child-resistant packaging currently exist in some of the high-income countries. 15- 18

Conclusion

Childhood poisoning still contributor to preventable child- related injury. The subjects were among urban settlers of higher maternal education and social class. Kerosene and organophosphate were most prevalent poisoning agents and severity and mortality was associated with type of poisoning agent and home intervention.

Recommendations and Limitation

The use of sustained health education and awareness campaigns to discourage the storage of kerosene in commonly used house- hold containers such as soft drink and bottled water containers should be intensified and also to discourage the use of dangerous poison insecticides and rodenticides agent at home. Diagnosis was limited to historical and clinical findings, as facilities for toxicological evaluation was not available.

Conflict of Interest: None

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References

- World Health Organisation/ UNICEF. World report on child injury prevention 2008. Available at http:// whqlibdoc.who.int/ publications/2008/9789241563574_eng.pdf (accessed February 2018).
- 2. Bateman DN. The epidemiology of poisoning. *Medicine*, 2007, 35:537–539.
- Adejuyigbe EA, Onayade AA, Senbanjo IO, Oseni SE. Childhood poisoning at the Obafemi Awolowo University Teaching Hospital, Ile-Ife, Nigeria. Niger J Med 2002; 11:183-6.
- Ochigbo SO, Udoh JJ, Antia-Obong OE. Accidental childhood poisoning in Calabar at the turn of the 20th century. Niger J Paediatr 2004; 31:67-70.
- Oguche S, Bukbuk DN, Watila IM. Pattern of hospital admissions of children with poisoning in the Sudano-Sahelian North Eastern Nigeria. Niger J Clin Pract 2007; 10:111-5.
- Edelu BO, Odetunde OI, Eke CB, Uwaezuoke NA, and Oguonu T. Accidental Childhood Poisoning in Enugu, South-East, Nigeria. Ann Med Health Sci Res 2016; 6(3)168.

- Dominc OO and Godwin S. Socio-demographic factors in accidental poisoning in Children. J Medicine and Medical Sciences 2013; 4(1): 13-16.
- 8. Ugwu GIM, Okperi BO, Ugwu EN, Okolugbo NE. Childhood poisoning in Warri, Niger Delta, Nigeria: A ten-year retrospective study. *Afr J Prm Health Care Fam Med.* 2012; 4 (1): 321-25.
- 9. Olatunya OS, Isinkaye AO, Ogundare EO, Oluwayemi IO, Akinola FJ. Childhood Poisoning at a Tertiary Hospital in South West Nigeria. *J Nepal Paediatr Soc* 2015; 35(2):103-110.
- Ibekwe RC, Amadife MU, Muoneke VU, Onyire BN. Accidental childhood poisoning in Ebonyi State University Teaching Hospital (EBSUTH). Abakaliki, South Eastern Nigeria. Ebonyi Med J 2007; 6:26-9.
- Fagbule DO, Joiner KT. Kerosene poisoning in childhood: A
 6-year prospective study at the
 University of Ilorin Teaching
 Hospital. West Afr J Med 1992;
 11:116-21.

- 12. Kivisto JE, Mattila V, Arvola T, Paavola M, Parkkari J. Secular trends in poisonings leading to hospital admission among Finnish children and adolescents between 1971 and 2005. *J Pediatr* 2008; 153:820-824.
- 13. Liebelt E, DeAngelis C. Evolving trends and treatment advances in pediatric poisoning. *JAMA 1999*; 282:1113-115.
- 14. Litovitz T, Manoguerra A. Comparison of pediatric poisoning hazards: an analysis of 3.8 million exposure incidents. A report from the American Association of Poison Control Centres. *Pediatrics* 1992; 89(6):999-1006.
- Meyer S, Eddleston M, Bailey B, Desel H, Gottschling S, Gortner L. Unintentional household poisoning in children. Klin Padiatr 2007; 219:254-270.
- McKenzie L, Ahir N, Stolz U, Nelson N. Household cleaning product-related injuries treated in US Emergency Departments in 1990-2006. *Pedi*atrics 2010; 126(3):509-516.

- 17. Pearn, J., Nixon, J., Ansford, A., Corcoran, A. Accidental poisoning in childhood: five-year urban population study with 15-year analysis of fatality. *BMJ*. 1984; 288:44 46
- 18. Jolly DL, Moller JN, Volkmer RE. The socio-economic context of child injury in Australia. *J Paediatr Child Health*, 1993, 29:438–444.
- 19. Oyedeji GA. Socio-economic and cultural background of hospitalized children in llesa. *Niger J Paediatr.* 1985; 12:111–7.
- Persson HE, Sjoberg GK, Haines JA, Pronczuk de Garbino J. Poisoning severity score. Grading of acute poisoning. J Toxicol ClinToxicol 1998; 36(3):205-213.

- 21. Annu J, Sivanandam S and Matthai J. Poisoning in Children from an Educationally and Economically Advanced Urban Area of South India. *Asian J Epidemiology 2012; 5: 123-129.*
- 22. Balme, K., Roberts, J.C., Glasstone, M., Curling, L., Mann, M.D. The changing trends of childhood poisoning at a tertiary children's hospital in South Africa. *S Afr Med J 2012*; 102:142-146.
- 23. Azizi BH, Zulkifl i HI, Kasim MS. Risk factors for accidental poisoning in urban Malaysian children. *Ann Trop Paediatr*. 1993; 13:183–188.
- 24. Reed RP, Conradie FM. The epidemiology and clinical features of paraffin (kerosene) poisoning in rural African children. *Ann Trop Paediatr. 1997*; 17:49–55.

- 25. Ferrer A, Cabral R. Recent epidemics of poisoning by pesticides. *Toxicology Letters* 1995; 82–83:55–63.
- 26. Abrahams N. Safe closures for paraffin. *Trauma Review* 1994; 2:5–6.
- Maharani B Vijayakumari N. Profile of poisoning cases in a Tertiary care Hospital, Tamil Nadu, India. J Applied Pharmaceutical Sci 2013; 3 (01), 091-094.
- 28. Gee P, Ardagh M. Paediatric exploratory ingestions of paracetamol. *New Zealand Med J 1998*; 111:186–188.