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Zinc phosphide (rodenticide) poisoning: A case report of deliberate self-harm in an eleven-year-old

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Abstract: Zinc Phosphide (Zn_3P_2) is a common rodenticide freely available in Nigeria for use against rodents. Occasionally human consumption occurs either accidentally or intentionally with potential consequences of multiorgan toxicity and death.

An 11-year-old boy consumed an unknown quantity of zinc phosphide marketed as Push Out® with the intention of committing suicide and killing some members of the family as his response to chastisement for a misdemeanour. Patient presented in the hospital 4 days after ingestion of zinc phosphide with a history of profound vomiting and abdominal pain. Laboratory evaluation showed evidence of hepatic dysfunction, acute kidney injury

and elevated serum amylase. He was managed conservatively and discharged home after two weeks of admission.

We report this case to emphasise the need for Paediatricians to consider ingestion of rodenticides as a differential diagnosis of hepatotoxicity and pancreatic enzyme elevation, as well as to highlight the possibility of suicide among children. There is need to control the indiscriminate use of rodenticides, strengthen public health education on poisoning as well as establish Poison Information Centres in our environment.

Key words: Poisoning, Zinc phosphide, Rodenticide, Multiorgan, Self-harm, Adolescent.

Introduction

Zinc Phosphide, marketed under various brand names including Push Out®, is one of the many commonly used rodenticides in Nigeria. Occasionally zinc phosphide is accidentally ingested or intentionally ingested for self-harm.¹⁻³ Intentional ingestion of zinc phosphide for suicide is more commonly reported in Asian countries but sporadic in Europe.⁴⁻⁶ It is estimated that the ingestion of 55-70 mg/kg body weight is potentially lethal with a high mortality rate ranging from 37-100%.^{4,7,8} Upon oral ingestion of zinc phosphide, and in the presence of gastric acid, phosphine gas is released^{1,3} which is highly toxic and may result in respiratory toxicity, myocarditis, hepatotoxicity, acute kidney injury, coma, and death.^{3,4,9}

The nature of the poisonous substance ingested is usually a reflection of its access in the environment and in Nigeria and many developing countries, kerosene is the most incriminated agent in childhood poisoning.¹¹⁻¹³ In the USA, Dine and McGovern (1982)¹⁴ documented that alcohol, glutethimide, propoxyphene hydrochloride, diazepam, insulin, lye, and pepper were intentionally administered by guardians.

Studies show very few reported cases of suicides in children, though it is likely to be under-reported.¹⁵ In addition, several people believe that children do not have the developmental maturity to act on suicidal ideation however, studies show that by 9 years of age, many children do have a thorough understanding of death and suicide^{16,17} and such children may consider death as an option to end their emotional pain.¹⁸ In this report, zinc phosphide was intentionally ingested for self-harm and potentially commit homicide of some members of the family as the patient's response to impending corporal punishment for a misdemeanour.

Therefore, this case report is to emphasise the need for Paediatricians to consider ingestion of rodenticides as a differential diagnosis of hepatotoxicity and pancreatic enzyme elevation, as well as to highlight the possibility of suicide among children.

Case Presentation

An 11-year-old boy who lives with the paternal grandmother presented at the University College Hospital, Ibadan, Nigeria, four days after ingestion of an unknown quantity of zinc phosphide marketed as Push Out®. Patient was seen at the facility with a 4-day history of

profuse vomiting, one day history of abdominal pain, jaundice, reduction in urinary volume and lethargy. Patient was said to have sprinkled Push Out® on the food of his twin sister who had reported the patient to the aunt for an infraction of the rules of the household. The aunt thereafter sought to chastise him for the misdemeanour, hence the attempted self-harm and poisoning of the food of his twin sister who made the report to the aunt who sought to act on the report. Shortly after the ingestion, patient was given home remedy made of palm oil and lime with the hope of neutralising the poison, paracetamol as well as mist magnesium trisilicate when he developed abdominal pain.

On presentation in the hospital, 4 days after ingestion of Push Out®, patient was conscious but lethargic, not pale, mildly dehydrated, moderately jaundiced and no pedal oedema. Throughout the admission, patient was haemodynamically stable with pulse ranging between 90 and 100 beats/min, capillary refill time of 2 seconds and a blood pressure between 90/60 and 100/60 mmHg. Examination findings in the other systems were essentially normal.

Laboratory investigations showed haemoglobin on admission of 13.4 g/dl, total WBC $4.12 \times 10^3/\mu\text{L}$, Neutrophil $2.0 \times 10^3/\mu\text{L}$, Lymphocytes $1.08 \times 10^3/\mu\text{L}$, Monocyte $0.30 \times 10^3/\mu\text{L}$, Eosinophil $0.71 \times 10^3/\mu\text{L}$, Basophil $0.03 \times 10^3/\mu\text{L}$ and Platelets $225 \times 10^3/\mu\text{L}$. Tables 1&2 show other results of investigations at admission and subsequently. On admission, the renal function tests showed urea 88mg/dl (Normal range: 15-45 mg/dl) and a creatinine of 1.4 mg/dl (Normal Range: 0.5-1.2 mg/dl) (Table 1). Table 2 showed total bilirubin 8.4 mg/dl (Normal Range: 0.2 – 1.0mg/dl), direct bilirubin 5.1 mg/dl (Normal Range: 0 – 0.4mg/dl) and elevated liver enzymes on admission and days 3 and 9 (Table 2; Figure 1). The International Normalised Ratio was 1.12 on day 2 of admission. Serum amylase on days 6 and 10 of admission was 157 U/L and 207 U/L respectively (Normal Range 28 - 100 U/L). Patient was negative for Hepatitis C antibody, Hepatitis B surface antigen and negative for HIV 1 & 2. The Electrocardiograph on day 3 of admission showed right axis deviation, normal T wave and voltage criteria for left ventricular hypertrophy for which he is being investigated. Abdominal USS revealed a normal sized liver with normal echotexture and features in keeping with acute acalculus cholecystitis.

Patient was managed conservatively with adequate hydration with urine output returning to normal within 72 hours of admission. Serum Creatinine and Urea returned to normal between days 3 and day 9 of admission (Table 1). On day 9 (Table 2), total serum bilirubin was 1.3mg/dl (Normal Range: 0.2 – 1.0mg/dl) with direct bilirubin 1.2 mg/dl (Normal Range: 0 – 0.4mg/dl).

Patient had psychiatric evaluation and non-pharmacological management as no definite mental illness was identified. Patient and family had psychosocial counselling and support from the medical social

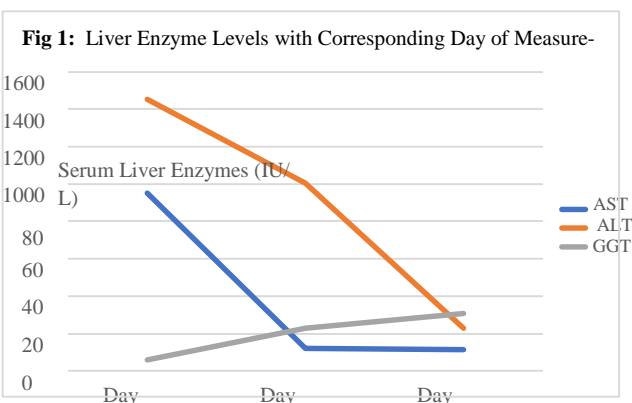
workers of the hospital and was subsequently discharged home after about 2 weeks of hospital admission.

Table 1:

Normal Range	Day 1	Day 3	Day 9
Na (130-145 mmol/l)	133	132	134
K (3.5-5 mmol/l)	3.7	3.2	3.5
Cl (95-110 mmol/l)	95	94	100
HCO ₃ (20-30 mmol/l)	25	25	20
Urea (15-45 mg/dl)	88	53	29
Creatinine (0.5-1.5 mg/dl)	1.4	1.2	0.9

Table 2:

Normal Range	Day 1	Day 3	Day 9
Total bilirubin (0.2 – 1.0) mg/dl	8.4	2.8	1.3
Direct bilirubin (0 – 0.4) mg/dl	5.1	2.4	1.2
ALP (187 - 400) IU/L	386	370	422
AST (0 – 37) IU/L	952	120	115
ALT (0 - 40) IU/L	1452	1005	230
GGT (7 - 50) IU/L	60	180	308
Total protein (6.0 – 8.0) g/dl	8.2	7.4	7.5
Albumin (3.5 – 5.0) g/dl	4.2	3.5	4.0



Discussion

Intentional poisoning either instigated through child abuse¹⁴ or suicidal ideations are uncommon in children. Dayasiri *et al*¹⁹ in a 7-year multi-hospital observational review of 1, 621 children aged between 1 and 12 years who presented with acute poisoning in Sri Lanka, reported 71 children (4.4%) presented with non-accidental poisoning comprising 32 cases of intentional poisonings with pesticides and 27 children with plant poisons. Although suicide remains a public health concern, there is extreme paucity of information on child and adolescent suicide from developing countries including Nigeria. Omigbodun *et al* reported a 20% prevalence of suicidal ideation and 12% of attempted suicide among Nigerian adolescents aged 10 – 17 years.²⁰

The root cause of this patient's risk for suicide probably emanated from family conflicts as the child lives with the paternal grandmother while his parents are divorced.

As part of the conditions of living with the grandmother, the father had cautioned that his twin children must be of good conduct and deviation from appropriate conduct will result in significant corporal punishment to be carried out by the father. In the realisation of his misconduct, child acted pre-emptively for fear of the anticipated punishment. The other members of the family whose food were poisoned did not take the food as the colour of the food appeared strange and therefore spared of toxicity.

The burden of rodenticide poisoning in Africa is unknown, however, there are occasional reports in literature where ingestion by children is documented.^{6,7,21} In the United States, the American Association of Poison Control Centers documented that rodenticide accounted for 0.3% of 2.3 million human exposures^[22] Rats have been known to be responsible for several diseases in Africa, therefore, rodenticides are commonly utilised to eliminate rats.²³

Zinc phosphide poisoning is known to cause significant morbidity and mortality among both socioeconomically low but economically active age demographics, especially in developing countries.³ Although, the quantity of zinc phosphide ingested in this patient could not be ascertained, a dosage of 4 to 5 g of zinc phosphide (55–70 mg/kg) had resulted in human deaths in acute toxicity,⁹ as well as a mortality of up to 37–100%.¹ It is recognised to cause both metabolic and non-metabolic toxic effects.

Following oral ingestion of zinc phosphide and its interaction with gastric acid, phosphine gas is released and rapidly absorbed, leading to circulatory collapse.^{1,3} However, in this patient, circulatory collapse did not manifest, rather multisystem toxicity occurred.²² It is noteworthy that this patient exhibited the more common gastrointestinal toxicity of nausea, abdominal pain and vomiting.^{1-4,7} Multiorgan impairment expressed as hepatotoxicity,^{10,24-27} pancreatitis³ which is not commonly reported³ and acute kidney injury demonstrated by oliguria and azotaemia³ were all documented in this patient. These symptoms are similar but not limited to the symptoms documented in the literature as recorded in reports of zinc poisoning cases from across the world. It is possible that the full manifestations of its toxicity were not demonstrated in this patient because of the repeated vomiting, administration of local remedy, antacid and ingestion of sub-lethal dose since only one sachet was sprinkled on the meals of two other members of the family.

Various studies have explained the mechanism of phosphide poisoning as follows

1. Phosphine inhibits the oxygen uptake in the rat liver mitochondria.^[28]
2. It inhibits ADP uncoupled site and ion stimulated respiration thus affecting pyruvate malate, succinate, glycerophosphate and ascorbate cytochrome biomolecules in liver mitochondria^[29]. However,

the exact site is a contentious issue.

3. It alters mitochondrial morphology, inhibits oxidative respiration by 70% and causes a large drop in mitochondrial membrane potential within 5 h of exposure.³⁰
4. Phosphine and hydrogen peroxide can interact to form the highly reactive hydroxyl radical that causes lipid peroxidation which is the main mechanism of oxidative damage to cell structures that lead to cell death.¹
5. Cytochrome C oxidase system is inhibited.^{31,32}
6. There is decreased activity of cytochrome oxidase along with altered NADH and succinic dehydrogenase activities.³³
7. It increases the lipid peroxidation in the central nervous system while reducing the antioxidant defence system such as superoxide dismutase, catalase and glutathione reductase.³⁴
8. It inhibits protein synthesis and enzymatic activity
9. It has anti-choline esterase effects and causes denaturation of oxy-haemoglobin molecules.²⁷

Phosphide poisoning is also known to cause glycaemic derangement. Severe hypoglycaemia is more commonly seen due to the reduced hepatic glycogenolysis and gluconeogenesis. The other differential diagnoses of phosphide poisoning include respiratory toxicity, myocarditis, hepatotoxicity, acute kidney injury and coma.

Though our patient did not present early or with arrhythmias or hypotension which are poor prognostic factors, the toxic effects of zinc phosphide and its related aluminium sulphide should not be downplayed.

To mitigate such recurrence, there is need for public education on the dangers of ingestion of zinc phosphide, poison control and establishment and strengthening of poisons control centres in Nigeria and West Africa. Health practitioners need to have heightened suspicion of poisoning in children and adolescents who present with features of multiple organ damage. As the victims of poisoning often present in emergencies, various antidotes and essential treatment for the management of such presentations should be made readily available by health administrators and government, and possibly provided at no cost to the victims.

Conclusion

In an environment where rats are uncontrolled and regulation of rodenticides is suboptimal, the use of zinc phosphide may continue to be available for accidental and intentional use in humans. There is therefore the need for control of both the rodents and rodenticides in order to prevent morbidity and mortality associated with the uncontrolled availability of rodenticides. Establishment of Poison Information Centres and strengthening of public health education on poisoning in our environment are advocated. Management in this case was largely supportive as there is no antidote.

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