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Clinical-Profile of Rodenticide Poisoning Cases Treated at BPKIHS (A Tertiary Care Hospital in Eastern Nepal): A Retrospective Study

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Abstract: ***Background**: Rodenticides are commonly known as rat poison, are typically non-specific pest control chemicals which are made and sold for the purpose of killing rodents [1]. It is directly toxic to mammals and risk of poisoning depends on its toxicity. These can be high, moderate or low toxicity [2]. Depending upon compound used it is broadly classified into metal phosphide and anticoagulants. The purpose of this study is to find the clinical outcome of the patients in our setting. **Materials and Methods**: This descriptive retrospective study was done for the rodenticide poisoning cases of five (5) years from 1st April 2015 to 31st March 2020. Sample size was calculated as 231. Collected data was entered in Microsoft excel 2010 and converted into SPSS 11.5 for statistical analysis. **Result**: 268 cases of five years were included. Out of which 33 were excluded from study due to incomplete data and mixed poisoning. Thus 235 cases were analyzed, 186 were aluminum phosphide, 15 were zinc phosphide and 34 were super warfarins.94 were male and 141 were female. Nausea, vomiting, dizziness and pain were the main clinical features.4 died from Aluminum phosphide group, 8 died from zinc phosphide group and none died from super warfarin group. Ionotropic support and mechanical ventilation needed in 80% of patients who died. **Conclusion**: Rodenticide poisoning is a common mode of self-harm. The metal phosphides (Zinc phosphide and Aluminium phosphide) are highly toxic with high mortality rate than super warfarin group. Among metal phosphides, Aluminium phosphide is more toxic than Zinc phosphide probably because Aluminium phosphide is unstable compound. The major cause of death in metal phosphide seems due to involvement of cardiovascular and metabolic system.*

Keywords: Aluminium phosphide, Zinc phosphide, super warfarin, mortality

1. Introduction

Rodenticides are commonly known as rat poison, are typically non-specific pest control chemicals which are made and sold for the purpose of killing rodents [1].

Rodents not only contains rats and mice but also squirrels, woodchucks, chipmunks, porcupines, nutria and beavers.

Besides being directly toxic to the mammals that ingest them, including dogs, cats, and humans, many rodenticides present a secondary poisoning risk to animals that hunt or scavenge the dead corpses of rats. Common classification of rodenticides according to their toxicity [2].

Highly Toxic Rodenticide
Highly toxic rodenticides are those substances with a single dose LD**50** of less than 50 mg/kg body weight. Examples: Aluminium phosphide, Sodium mono-fluoroacetate, Strychnine, Zinc phosphide, Yellow phosphorus, Arsenic, Thallium.

Moderately Toxic Rodenticides
Among the moderately toxic rodenticides, those with LD**50** of more than 500 mg/kg body weight. Examples: alpha-naphthyl-thiourea (ANTU), DDT.

Low Toxicity Rodenticides
Low toxicity rodenticides are those with LD500 and 5000 mg/kg body weight. Examples: Red squill, Norbomide, Anticoagulants (warfarin-type rodenticides). They have different toxicological profiles with variable fatality rates in humans when consumed by accident or intentionally [4].

Metal phosphides commonly used are Aluminium phosphide (ALP) and Zinc phosphide. Aluminium phosphide is used as a fumigant while zinc phosphide is used as bait [5, 6].

The mechanism of toxicity: In metal phosphide poisoning both Aluminium and Zinc phosphidecombines with moisture and acid in stomach and phosphine gasis released which has been thought to be toxic because it inhibits cytochrome c oxidase, catalase and peroxidase and thus blocks protein and enzyme synthesis and causes tissue necrosis in liver, heart, lung alveoli and brain. While phosphine does inhibit cytochrome C oxidase in vitro, the inhibition is much less in vivo. It has been shown recently in nematodes that phosphine rapidly perturbs mitochondrial morphology, inhibits oxidative respiration by 70%, and causes a severe drop in mitochondrial membrane potential. This failure of cellular respiration is likely to be due to a mechanism other than inhibition of cytochrome C oxidase. In addition, phosphine and hydrogen peroxide can interact to form the highly reactive hydroxyl radical and phosphine also inhibits catalase and peroxidase; both mechanisms result in hydroxyl radical associated damage such as lipid peroxidation. The

major lethal consequence of phosphide ingestion, profound circulatory collapse, is secondary to factors including direct effects on cardiac myocytes, fluid loss, and adrenal gland damage. [4]

The common clinical features of poisoning are headache, dizziness, nausea/vomiting, cough, dyspnoea, chest tightness, thirst, liver failure, jaundice, tetany, delirium, convulsion, coma and death. [7]

Anticoagulants commonly used as a rodenticide are Bromadiolone. It is a second-generation 4-hydroxycoumarin derivative and Vitamin K antagonist. It is commonly referred to as "Super Warfarin" due to its potency. OtherWarfarin analogues are: Brodifacoum, Difethialone, Diphacinone, Pindone of which warfarin is first generation anticoagulant and rest are second generation anticoagulant.

Both Brodifacoum and Bromadiolone act by the same mechanism as warfarin but are 100 times more potent and have much longer serum and tissue half-lives. Coagulation factors II, VII, IX, X, and other vitamin K–dependent proteins require gamma-carboxylation of glutamic acid residues to be biologically active. Active vitamin K is regenerated by epoxide reductase enzymes, which are blocked by warfarin and its analogues. The limited availability of active vitamin K produces a bleeding diathesis or complication.

The treatment of poisoned patients includes gastric lavage, followed by infusion of active charcoal, vitamin K1 therapy, and FFP transfusion in the case of bleeding [4].

Rationale:
The rationale of this study is to find out clinical profile of rat killer poisoning cases so that we can be prepared beforehand to manage these cases in better way and maybe we could decrease the mortality rate from rat killer poisoning which seems to be high at present.

Hypothesis:
Clinical profile and mortality of rat killer poisoning cases might be different at this centre.

Objectives:
1) General Objective: To find out the clinical profile of rat killer poisoning cases at BPKIHS.
2) Specific Objective: To find out the mortality rate of rat killer poisoning cases at this centre.

2. Review of Literature

D K Suneetha et al, March 2016 in International Journal of scientific study, included 56 cases of rat killer poison. In all the cases route of exposure was oral, majority cases were in the age group 11-30 years. The most common poisoning agent was Zinc Phosphide 32%, followed by aluminium phosphide 21% and yellow phosphorus 14%. Mortality rate was high with aluminium phosphide 5 case out of 12 and zinc phosphide 3 case out of 18. LFT derangement was with yellow phosphorus. ICU admissions were more with aluminium phosphide [1].

Yu et al. Springer Plus 2013, This study mainly included anticoagulant rodenticide and showed most of the rodenticide poisoning patients were middle-aged adults and both genders were equally affected. Many patients had a past history of major depressive disorder or schizophrenia. In analysis, it was revealed that patients with brodifacoum suffered higher incidences of bleeding complications, such as ecchymosis (50.0%) and hematuria (50.0%), than patients with bromadiolone poisoning [4].

Dr. Murali Nalabothu et al in International Journal of Scientific and Research Publications, It says among the patients presenting with Rodenticide poisoning most of them presented with ingestion of Yellow Phosphorous, Zinc Phosphide followed by Superwarfarins and Aluminium phosphide. Patients who presented early and started with N Acetylcysteine had good prognosis. Patients who presented with consumption Superwarfarin had excellent prognosis [8].

ShashidharaKuppegala et al in International Journal of Research in Medical Sciences. This study included a total of 31 patients out of 64 had consumed Aluminium Phosphide. They had consumed a mean of 7.5 grams of aluminium phosphide each. The mean values of Poison consumption were 5.11 grams among patients who survived and 11.66 grams in those who expired. Only 10 out of the 31 cases (34.5%) had received Magnesium Sulphate immediately at presentation to the hospital. While 17 more of the cases had received it later during the course of hospital stay. The average time since consumption to treatment with magnesium sulphate was 4.83 hours in the expired patients while it was 6.18 hours in the patients who had survived. Among the rodenticides, Aluminium Phosphide was the foremost common cause for mortality in this study. Even though Magnesium sulphate's usage had shown better prognosis in this study, in view of less number of cases a large randomized controlled trial is necessary to prove Magnesium Sulphate's therapeutic potential in improving the outcome.

3. Materials and Methods

This descriptive retrospective study was done for the rodenticide poisoning cases of five (5) years from 1st April 2015 to 31st March 2020 which were admitted at Emergency ward (EW) of B. P. Koirala Institute of Health Sciences (BPKIHS), Dharan, a tertiary care hospital in Eastern Nepal. All patients ≥16 years of age presenting in Emergency ward with history of rat killer (rodenticide) poisoning were included in the study. The rodenticide poisoning was confirmed by history from patient or care taker or from the brand name of the poison container that the care take or patient had brought in EW. Patients with mixed poisoning, case file with inadequate or incomplete data were excluded from the study.

All the samples required for this study was collected from the medical record section BPKIHS. The case files were searched in the system with ICD code T60.4 and case details were taken as per the proforma.

Sample size calculation:
Sample size was calculated as **231** assuming 20.3% of overall mortality from all kind of rodenticide poisoning 20.3% [9].

The study considers 95% confidence interval and 80% power to estimate the sample size.

Now using the following formula,

$$\text{Sample size (n)} = \frac{z^2pq}{d^2}$$

where z=1.96 at 95% confidence interval
p=20.3% i. e. about 20%
then q= 80%
d= 20% of p at 80% power
i. e 20% of 20= 4
Putting the values in the equation,

$$\text{Sample size (n)} = \frac{1.96^2 \text{ x}20\text{x}80}{4^2} = 384$$

Now adding 10% at calculated sample size to reduce various biases, the sample size now will be 384+384 x 10%= **422.4**
Now using finite sample size formula (sample size)
(N) =Calculated Sample Size/ (1+ Calculated Sample Size/Estimated Population)

According to medical record the lightning injury of last year (2019-04-01 to 2020-03-31) was 52 then, N = 422.4 / (1+422.4/52)] = 422.4/ (1+8.12) = 46.3 x 5 years = 231.5.

Thus, the sample size will be **= *231***

Statistical Analysis:
Collected data was entered in Microsoft excel 2010 and converted into SPSS 11.5 for statistical analysis. Descriptive statistical data like percentage, frequency, mean, SD, were calculated along with graphical and tabular presentation. For inferential statistics, Chi square test, independent t-test or Mann-Whitney u-test was applied between death, risk factors, lab parameters and demographic variables.

Ethical Consideration:
Ethical clearance was taken from "Institutional Review Committee (IRC) " of BPKIHS.

4. Results

The total registered number of rodenticide poisoning cases during 1st April 2015 to 31st March 2020 were 268. Out of which the clinical details were not complete in 7 case files and 26 had mixed poisoning which were excluded from the study (total 33). Thus, the total number of rodenticide poisoning included in this study were **235** excluding the mixed poisoning cases and inadequate case files, of which 186 (79.15%) were Zinc Phosphide poisoning, 15 (6.38%) were AluminiumPhosphide poisoning and 34 (14.46%) were Super coumarin group poisoning.

The circumstances of poisoning were intentional i. e. suicidal in 224 (95.31%) and 11 (4.68%) were accidental.85 (36.17%) had history of drinking alcohol while taking rodenticide poison, 59 (25.1%) cases consumed poison with cold drink, 73 (31.06%) consumed mixing with water and 18 (7.66%) consumed without any additives. Most of the patients were not able to say the exact name of the poison but they could remember the characteristics of the poison (colour, form, smell).94 (40%) cases were male and 141 (60%) cases were female. The rest of the demographic data are given in table 1.

Table 1: Clinical characteristics of patients with zinc phosphide poisoning.

Characteristics		
Sex	Male	94 (40%)
	Female	141 (60%)
Age (Median)	35years (16-66 years)	
Religion	Hindu	214 (91.06%)
	Muslim	8 (3.4%)
	Kirat	9 (3.83%)
	Christian	4 (1.7%)
Region	Hill	87 (37.02%)
	Terai	148 (62.98%)
Amount of exposure	<1 Packet	116 (49.36%)
	>1 Packet	61 (25.96%)
	Unknown	58 (24.68 %)

As per table 1 a major portion of the patient was taken by female, median age was 35 years. Hindu was the majority religion and 63% presented from terai region.

Table 2: Occupation of victim

Occupation	n (%)
Labourers	75 (31.96)
Housewife	53 (22.55)
Student	61 (25.96)
Farmer	25 (10.64)
Drivers	5 (2.13)
Private job	13 (5.53)
Government job	3 (1.28)

Table 2 shows majority of the poisoning victims were labourers, housewives and students.

Table 3: Clinical features and vital signs at presentation

Clinical Presentation n (%)	
1. Common Presenting Symptoms	
a. Nausea and Vomiting	125 (53.19)
b. Dizziness	91 (38.72)
c. Abdominal Pain	78 (33.19)
d. Palpitation	35 (14.89)
e. Dyspnoea	26 (11.06)
f. Drowsiness	20 (8.51)
2. Temperature (Perepheral in ^oC)	
a. <37.5	219 (93.19)
b. >37.5	16 (6.8)
3. Pulse Rate	
a. <60bpm	10 (4.25)
b. 60-100bpm	182 (77.44)
c. >100bpm	43 (18.29)
4. Respiratory Rate	
a. <20	155 (65.96)
b. >20	80 (34.04)
5. Blood Pressure	
a. Low (<90/60 mmHg)	35 (14.89)
b. Normal (90/60-139/89)	178 (75.74)
c. High (>140/90mmHg)	22 (9.36)
6. Oxygen Saturation	
a. Normal (>90%)	230 (97.87)
b. Low (<90%)	5 (2.13)

7. Glasgow Coma Scale	
a. <9	4 (1.70)
b. 9-12	4 (1.70)
c. 13-15	227 (96.59)

Nausea, vomiting, dizziness and pain abdomen were more common clinical features in the rodenticide poisoning cases. Fever was not very common feature in the presentation but a considerable number of patients had low blood pressure. Rodenticide poisoning has not prominent effect on CNS, but few cases had low GCS which could be because of indirect effect of poison on CNS like due to hypoxia or hypotension or hypoglycaemia.

Table 4: Outcome and hospital stay of rodenticide poisoning

	n (%)	Ward stay (mean days)	ICU admission n (%)	ICU stay (mean days)	Survived n (%)	Died n (%)	LAMA n (%)
Aluminium Phosphide	15 (6.38)	5.4	7 (46.66)	5.2	10 (66.67)	4 (26.67)	1 (6.67)
Zinc Phosphide	186 (79.15)	4.5	28 (15.05)	3.5	168 (90.32)	8 (4.30)	10 (5.37)
Bromadiolone	34 (14.46)	3.6	0	-	33 (97.06)	0	1 (2.94)
Total	235 (100)	4.5	35 (14.89)	4.35	211 (89.78)	12 (5.10)	12 (5.10)

In table 4, we have tried to show the comparative effects of the different rodenticide. Aluminium phosphide poisoning shows most toxic effect among the rodenticides in terms of hospital stay and percentage of death. Zinc phosphide seems toxic than Aluminium phosphide. Bromadiolone and Brodifacoum is least toxic among the rodenticide and it did not cause any mortality during the hospital stay.

Table 5: Rodenticide poisoning and their effect on LFT and ABG

	Zinc Phosphide		Aluminium Phosphide		Bromadiolone and Brodifacoum
	Outcome		Outcome		Outcome
	Survivors	Expired	Survivors	Expired	Survivors
Liver Function Test					
Total Bilirubin	0.82	0.92	0.73	0.87	0.69
AST	46.23	105.34	40.54	215.32	42.12
AST 5th day	50.08	-	45.61	-	32.00
ALT	45.34	72.21	62.34	123.51	33.32
ALT 5^{th} day	87.45	-	67.43	-	
PT-INR	1.0	1.12	1.3	1.4	1.4
PT-INR 5^{th} day	1.2	-	1.2	-	1.3
Arterial blood gas (ABG)					
PH	7.32	7.02	7.32	6.93	7.37
$HCo3^-$	17.22	10.12	15.40	7.32	20.32
Lactate	2.5	6.4	3.5	12.3	1.9

In table 5, the lab parameters (LFT and ABG) values are worse in the patients who died. Lactate and liver enzymes are considerably high in the patients who died in both the Zinc Phosphide and Aluminium Phosphide group. In Aluminium phosphide group the lactate is raised more compared to Zinc phosphide group. The liver enzymes mainly AST is raised up to 5 times the normal value in the expired group of Aluminium phosphides and up to 2 to 3 times in expired group of Zinc phosphide group. Similarly, PH is very much decreased in expired group of Aluminium phosphide and Zinc phosphide. There is not much effect of poison on PT/INR.

Table 6

S. N.	Clinical characteristics and laboratory findings	No. of Patient (out of 235)	Patients who survived (out of 211)	Patients who died (out of 12)	Pt. on LAMA (out of 12)
1.	Duration from exposure to hospital visit in hours (mean)		5.9	8.6	7.6
2.	HR >100/min at presentation	43 (18.3%)	24 (9%)	10 (83.3%)	9 (75%)
3.	SBP <90mmHg at presentation	35 (14.9%)	17 (8%)	8 (66.7%)	10 (83.3%)
4.	DBP<60mmHg at presentation	35 (14.9%)	17 (8%)	8 (66.7%)	10 (83.3%)
5.	RR >20/min at presentation	80 (34%)	59 (27.9%)	10 (83.3%)	11 (91.7%)
6.	Na^+ >145mmol/L at presentation	11 (4.7%)	8 (66.7%)	1 (8.3%)	2 (16.7%)
7.	K^+ >5.5mmol/L at presentation	32 (13.6%)	25 (11.8%)	4 (33.3%)	3 (25%)
8.	Acidosis at presentation	43 (18.3%)	26 (12.3%)	11 (91.6%)	6 (50%)
9.	AKI (Acute kidney injury)	25 (10.6%)	10 (4.7%)	8 (66.7%)	7 (58.3%)
10.	Hypoglycaemia	51 (21.7%)	40 (18.9%)	6 (50%)	5 (41.7%)
11.	ET tube intubation	28 (11.9%)	12 (5.7%)	10 (83.3%)	8 (66.7%)
12.	Inotropic Support	29 (12.3%)	10 (4.7%)	10 (83.3%)	9 (75%)

In table 6, the clinical characteristics and laboratory findings are compared between patients who survived, patients who died and patients who left the hospital against medical advice (LAMA). The factors with significant differences were duration from exposure to hospital visit, abnormal vital signs at presentation (Heart rate, Low blood pressure, tachypnoea), acidosis, hypernatremia, hyperkalaemia, in

hospital AKI and in hospital hypoglycaemia, ET tube intubation and inotropic requirement.
There were no significant differences in sex, alcohol co-ingestion, temperature at presentation, oxygen saturation at presentation and GCS score at presentation.

The patients who presented early (within 6 hours of ingestion) to hospital have lower rate of mortality. Tachycardia, hypotension and dyselectrolytemia were more common in expired group. Almost 34.4%% of intubated patients expired, about 34.5% of patient on inotropic support expired and 25.6%% of patients with metabolic acidosis expired.

The treatment modalities are IV fluids given in all the patients (100%). Gastric aspiration was done in 27.5% cases and gastric lavage in 15.2% cases about 14.9% cases needed oxygen support. Endotracheal intubation was performed in 11.9% of patients and inotropic support was needed in 12.3%. Almost all (94.2%) patients were initially admitted in Internal medicine ward, 2.12% of the patients went home against medical advice, 3.8% of the cases were admitted in ICU from ER and 11% of the patients were admitted in ICU from ward after clinical deterioration. The median length of hospital stay was 4 days (range 2-10 days).

The overall mortality rate in our study was 5.1% (12 patients).2.12% (5) patients went on LAMA from ER and 2.97% (7) patients went on LAMA from ICU after clinical deterioration and non-improvement due to financial issues. So, if we add LAMA from ICU and hospital death the mortality rate would reach to 8.08%.

Out of total death which was 12 in number, 11 death were from suicidal attempt and 1 death was from accidental ingestion.10 out of 12 patients who died in hospital developed systemic features cardiovascular or respiratory within 24 hours of ingestion and rest 2 developed the systemic features after 24-48 hours of ingestion. The ECG reading were present in 6 of the hospital death patients. Ventricular tachycardia, ventricular fibrillation and asystole were present in 2, 1 and 3 patients respectively.

Aluminium phosphide
A total of 15 patients (6.38%) had consumed Aluminium phosphide. As the details given on the container the concentration usually comes in 56%, weight of each tablet of Aluminium phosphide was 3gram and in 10gram per sachet when in sachet form. Out of 15 patients, 5 patients had consumed single tablet of 3 grams, 4 patients had consumed 2 tablets i. e.6 grams by each and 6 patients had consumed sachet form of Aluminium phosphide, out of which 4 consumed full sachets and 2 had consumed the sachet partially. So, the mean amount of consumption was about 5.9grams. Average ward stay was 5.4 days. The total deaths from Aluminium phosphide poisoning was 4 (26.67%), 1 patient went on LAMA and 10 patients were successfully discharged from hospital.7 patients (46.7%) needed ICU care and 8 patients were managed in ward. Liver enzymes were increased almost 4-5 times, PH was less than 7.00 and lactate was more than 12.3 in those expired with Aluminium phosphide poisoning.

Zinc phosphide:
Total of 186 patients (79.15%) consumed Zinc phosphide. As per the details on container one packet of Zinc phosphide contains about 10 grams in 80% concentration. Out of 186 patients only 72 patients (38.7%) had consumed from fresh packet but 114 patients had consumed from open packet and amount was unknown. Average ward stay was 4.5 days. The total death from Zinc phosphide poisoning was 8 (4.3%), 10 patients (5.3%) went on LAMA and 168 patients (90.3%) were successfully discharged from hospital.28 patients (15%) needed ICU admission. The liver enzymes were elevated by 2 times, PH was in the range of 7.00 and lactate was around 6 among those expired.

Bromadiolone, Brodifacoum
34 patients (18.27%) consumed Bromadiolone a super coumarin type of anticoagulant rodenticide. Commercially it is available in 0.005% concentration. Common commercial names are: Tiger cake, DR. PEST, Mortein cake, Hit cake. Both Bromadiolone and Brodifacoum have similar potency and comes in similar concentration. Out of 34, 15 cases had consumed Bromadiolone, 11 had consumed Brodifacoum and in the remaining 8 cases the ingredient was not mentioned and was assumed as anticoagulant poisoning by the description given by patient and the attendant as bluish cake Rat killer. The mean amount was approximately 11grams. Liver enzymes were not elevated and PT/INR was mildly elevated. ABG parameters were also normal. There was no death due to anticoagulant rat killer. One patient went on LAMA. Average ward stay was 3.6 days. None of the patient required ICU admission, Intubation or inotropic support.

5. Discussion

Rodenticide is inexpensive and readily available everywhere in Nepal. Anyone can find it in general stores, grocery shops or department stores and most people keep rodenticides at their home for using it against household rodents. Rodenticide poisoning is third most common cause of poisoning in Nepal after Organophosphate and Medicines. [11] It is a common deliberate self-harm agent.

In one study the result showed the rodenticide poisoning is common in male they found 60% in male and about 40% in female. [2] But in our study, we found it is more common in female which is about 60%. This may be because here in Nepal most of the family is male dominated and could be due to non-fulfilment of their day to day needs or lack of representation of their opinion in family decisions.

In this study, we can see that the poisoning is more common among low income and less educated group this could be because of difficulty in managing financial burden of family and lack of education regarding the poison. This finding is supported by another study done at Mysore India. [1]

The common clinical presentations were nausea, vomiting, dizziness, abdominal pain. Palpitation and shortness of breath were less common. Fever at presentation was not a common feature. Similar findings were present with slight changes in percentage in other studies done in India and other countries. [2, 6, 8]

The onset of systemic features was early and more severe in Aluminium phosphide poisoning group than the Zinc phosphide and Bromadiolone group.12 out of 15 patients of Aluminium phosphide poisoning patients developed systemic features within 24 hours of ingestion. The severity of the features was shown by presence of low blood pressure, decreased oxygen saturation, elevated liver enzymes, metabolic acidosis and high lactate level. In about 74% of patients of Zinc phosphide poisoning, the systemic features developed within 48 hours and was less severe in nature. This is supported by Trakulsrichal et al and study done by Alex TP. The Systemic features appears early and is more severe in Aluminium phosphide poisoning as Aluminium phosphide is unstable in contrast Zinc phosphide is relatively stable compound. [2, 12]

The mortality rate was highest with Aluminium phosphide than Zinc phosphide in our study which was 26.7% and 4.3% respectively. In Suneetha et at also the mortality rate is higher with Aluminium phosphide than Zinc phosphide. [1] The mortality rate of Aluminium phosphide and Zinc phosphide in Sunitha et al was 41.7% and 16.7% respectively. This higher rate of mortality in other studies could be because of other constituents present in the poison or the higher concentration of poison in the packet. In this study 6.7% of Aluminium phosphide poisoning patient and 5.4% of Zinc phosphide poisoningpatients had left the treatment against medical advice (LAMA) and most of the cases who went on LAMA were in poor clinical condition and had poor prognosis. So, if we add LAMA patient to the mortality, the mortality rate would slightly increase.

The treatment of all types of rodenticide poisoning is more of supportive with adequate IV fluids, early gastric aspiration and lavage, Oxygen supplementation and close monitoring for development of cardiac complication and early intervention. The antidote for Bromadiolone and Brodifacoum is available as Vitamin K and if needed fresh frozen plasma can also be given in patients with bleeding manifestation. But there is no specific antidote for metal phosphides, we are forced to depend only on supportive management.

Few studies have tried N-acetylcysteine and Magnesium sulphate for the management of metal phosphide but they didn't get promising result. [9, 10]

6. Conclusion and Recommendation

Rodenticide poisoning is a common mode of self-harm. The metal phosphides (Zinc phosphide and Aluminium phosphide) are highly toxic with high mortality rate than super warfarin group. Among metal phosphides, Aluminium phosphide is more toxic than Zinc phosphide probably because Aluminium phosphide is unstable compound. The metal phosphide poisoning primarily affects the gastrointestinal, Cardiovascular, Respiratory and Metabolic systems. There is no specific antidote for metal phosphide and we need to depend on supportive care only for its management. The major cause of death in metal phosphide seems due to involvement of cardiovascular and metabolic system. So, we need to monitor these systems very closely. As super warfarin group is less toxic to human body with no mortality in our study, the government should have policy to encourage its use as rodenticide by general public and should restrict the use of metal phosphide so that we could prevent some unnatural deaths.

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