Original Article

Toxicoepidemiology of Phosphide Poisoning in the Central Part of Iran

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ABSTRACT

Objective: Aluminum phosphide (ALP) and zinc phosphide (ZnP) are toxic agrochemical pesticides, which are commonly used as an agent of self-harm in developing countries. Because of high toxicity of phosphides, we evaluated toxico-epidemiology ALP and ZnP poisoning in with respect to outcome. **Methods:** We performed a cross-sectional study with retrospective chart review including the records for patients admitted due to phosphide poisoning (ALP, ZnP) in a poisoning referral center in Khorshid Hospital, affiliated with Isfahan University of Medial Sciences, Isfahan, Iran. Demographic characteristics, clinical manifestations, outcome (survived or death), and length of hospital stay for the patients were recorded in a data collecting form. Binary backward stepwise logistic regression was used for outcome prediction. **Findings:** Sixty patients were evaluated in the study. The mean age of patients was 27.61. Thirty-nine patients were men. 96.7% of the patients ingested it intentionally. Most of the patients on admission were conscious (66.7%). Abnormality of EKG was noted in 8.3%. The mortality in ALP and ZnP poisoning was 39.2% and 22.2%, respectively. Serum bicarbonate and base excess in the venous blood gas analysis, systolic blood pressure, and serum sodium level were significantly different between patients with ALP and ZnP poisoning on admission time ($P < 0.05$). On admission, systolic blood pressure was an important predictive factor for mortality (odds ratio 4.87; 95% confidence interval: 1.5–15.45; $P = 0.007$). **Conclusion:** The rate of mortality in phosphide poisoning is high. Knowing predictive factors for mortality help physicians for selecting patients in intensive care unit admission and aggressive treatment.

KEYWORDS: *Aluminum phosphide, mortality, outcome, overdose*

INTRODUCTION

Phosphides, including aluminum phosphide (ALP) and zinc phosphide (ZnP), are toxic agrochemical pesticides in developing countries as it is cheap, effective, free from toxic residue, and do not affect seed viability. In addition, ALP is commonly used as an agent of self-harm in developing countries.[1,2] Its death rate in Asian countries is reported to be between 60% and 90%.[3,4] In a study in India, phosphide poisoning accounts for about 68% of the reported causes of poisoning in this subcontinent.[5] The mortality rate of ALP poisoning has been reported about 40%–45% in different parts of Iran.[6,7] Although most reports of ALP poisoning are related to young populations from rural areas in Asia, some cases of phosphine poisoning have been reported in European countries during the last three decades.[8-10]

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The toxic effects of phosphides are due to phosphin gas (PH3) released when it mixes with aqueous solutions (water or hydrochloride acid in stomach) which is rapidly absorbed by inhalation, ingestion, and skin or mucosal contacts.[2,11,12] Phosphin inhibits mitochondrial cytochrome oxidase and catalase, and the result is cell hypoxia.[13,14] While phosphine does inhibit cytochrome C oxidase *in vitro*; the inhibition is much less *in vivo*.[15] In addition, phosphine can interact to form the highly reactive hydroxyl radical which results in hydroxyl radical-associated damage including 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.[16,17]

Breathing ALP can irritate the nose, throat, and lungs causing coughing, wheezing, and shortness of breath. The gastrointestinal, cardiovascular, nervous systems, kidneys, liver, and lungs are the most affected organs in phosphide poisoning.[14,18-21]

Some studies suggest that glutathione, melatonin, Vitamin C and carotenes, N-acetylcysteine, methylene blue, calcium, and magnesium may reduce the effects caused by oxidative phosphines.[5,22,23] However, no definite treatment has been determined yet.

As the phosphide poisoning is common in our referral poisoning emergency center[24] hospitalized with high mortality, we evaluated the toxicoepidemiology of phosphide poisoning and the outcome in a 10-year cross-sectional study.

Methods

We performed a cross-sectional study with retrospective chart review for patients admitted due to phosphide poisoning (ALP, ZnP) between February 2005 and September 2014 hospitalized in the poisoning referral center, Khorshid hospital, in Isfahan, central part of Iran. The institutional board of human studies at Isfahan University of Medical Sciences (Research Project Number 292134) approved the study protocol. All patient information was protected and kept confidential. We reviewed the medical chart of patients hospitalized because of phosphide poisoning. Patients discharged by their own consent and patients with a history of ingestion but without any clinical manifestations during observation period were excluded from the study. For each patient, the different variables including gender, age, route of exposure (ingestion, skin, inhalation, mucosal) and type of poisoning (suicide, accidental)), kind of phosphide (ALP, ZnP), clinical manifestations on admission, abnormalities of electrocardiogram (ECG), mean dose of sodium bicarbonate (mEq/mL) used, other treatments (including administration of Vitamin C, Vitamin E, calcium gluconate, N-acetyl cysteine, and magnesium sulfate), history of psychiatric disorder, previous suicide attempt, duration of hospitalization, and outcome (survived and death) were recorded in a data gathering form.

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) software (IBM SPSS Statistics for Windows, Version 22.0, Armonk, NY, USA: IBM Corp.). Binary backward stepwise logistic regression analysis was used for outcome prediction. ANOVA, Chi-square, or Fisher Exact, Pearson/spearman correlation tests were used for analysis. P <0.05 was considered statistically significant.

Results

From February 2005 to September 2014, sixty patients poisoned with phosphides had inclusion criteria. The number of patients increased from one patient in 2005 to 2015 patients in 2014. Fifty-one (85%) patients were poisoned with ALP and 9 (15%) cases were poisoned with ZnP. Among those poisoned with ALP, 62.7% were males, with a mean age of 26.84 ± 7.89 years, and among those poisoned with ZnP, 77.8% were males, with a mean age of 32.00 ± 15.35 years. There was no significant difference between the patients with ALP and ZnP poisoning in terms of demographic characteristics, including age, gender, employment status, marital status, type of poisoning (intentional and unintentional), history of previous suicide, history of depression, addiction, and alcohol consumption [P > 0.05, Table 1].

Results regarding on admission clinical manifestations are shown in Table 2. Most of the patients were conscious (66.7%). 11.66% of patients had vomiting immediately after poison ingestion (six patients in AlP and one in ZnP) poisoning. In 48.34% of patients, the vomiting also occurred during hospitalization. Abnormality of EKG was wide QRS complex and junctional rhythm which was noted in 8.3% of all of cases. Furthermore, there was no significant difference between ALP and ZnP poisoning according to the clinical manifestations as well as complications [P > 0.05, Table 2].

Evaluation of the laboratory findings indicated that serum sodium (Na) at the admission to the hospital and discharge (or death) was significantly greater in those poisoned with ALP than those with ZnP (P < 0.05). Even though there was no significant difference between ALT and AST in the two types of poisoning at admission to the hospital (P > 0.05), the two parameters were far higher in those poisoned with ALP than those poisoned with ZnP at discharge or before death (P < 0.05). At admission to

Table 1: Comparison of patient's demographic characteristics between aluminum phosphide and zinc phosphide poisoning

Characteristics	Total (*n*=60), *n* (%)	ALP (*n*=51), *n* (%)	ZnP (*n*=9), *n* (%)	*P*
Gender				
Male	39 (65.0)	32 (62.7)	7 (77.8)	0.47
Female	21 (35.0)	19 (37.3)	2 (22.2)	
Age (years)	27.62±9.39	26.84±7.89	32.00±15.35	0.13
Homemaker	16 (26.7)	15 (29.4)	1 (11.1)	0.09
Other jobs	44 (73.3)	36 (70.6)	8 (88.9)	
Marital status				
Single	35 (58.3)	32 (62.7)	3 (33.3)	0.14
Married	25 (41.7)	19 (37.3)	6 (66.7)	
Type of exposure				
Intentional	58 (96.7)	50 (98)	8 (88.9)	0.28
Unintentional	2 (3.3)	1 (2)	1 (11.1)	
History of previous suicide	14 (23.3)	12 (23.5)	2 (22.2)	1.00
History of previous psychological problems	16 (26.7)	15 (29.4)	1 (11.1)	0.42
Addiction	5 (8.3)	4 (7.8)	1 (11.15)	0.57
Alcoholism	1 (1.7)	1 (2)	0	1.00

The Chi-square or Fisher's exact test used for analysis; the results are presented as *n* (%) or mean±SD where appropriate. SD=Standard deviation, ALP=Aluminum phosphide, ZnP=zinc phosphide

Table 2: Comparison of clinical manifestations between aluminum phosphide and zinc phosphide poisoning at admission to the hospital

Clinical manifestations	Total (*n*=60), *n* (%)	ALP (*n*=51), *n* (%)	ZnP (*n*=9), *n* (%)	*P*
Low level of consciousness	20 (33.3)	18 (35.3)	2 (22.2)	0.48
Vomiting	36 (60.0)	30 (58.8)	6 (66.7)	0.72
Nausea	23 (38.3)	19 (37.3)	4 (44.4)	0.72
Heartburn	3 (5.0)	3 (5.9)	0	1.00
Abdominal pain	12 (20.0)	8 (15.7)	4 (44.4)	0.06
Diarrhea	3 (5.0)	2 (3.9)	1 (11.1)	0.39
Restlessness	16 (26.7)	15 (29.4)	1 (11.1)	0.42
Thirst	7 (11.7)	7 (13.7)	0	0.58
Shock	15 (25.0)	15 (29.4)	0	0.09
Arrhythmia	5 (8.3)	5 (9.8)	0	1.00
Dyspnea	9 (15.0)	9 (17.6)	0	0.33
Cyanosis	9 (15.0)	9 (17.6)	0	0.33
Pulmonary crackle	4 (6.7)	4 (7.8)	0	1.00
Blurred vision	4 (6.7)	4 (7.8)	0	1.00

The Chi-square or fisher exact test used for analysis; the results are presented as *n* (%) or mean±SD where appropriate. ALP=Aluminum phosphide, ZnP=Zinc phosphide, SD=Standard deviation

the hospital, serum bicarbonate in the venous blood gas analysis (HCO_3) was significantly lower in patients with ALP poisoning (P = 0.01). Base excess (BE) was also significantly higher in those poisoned with ALP than in those poisoned with ZnP (P = 0.04). However, after the treatment, at the discharge time, these two factors did not have a significant difference between the two groups of poisoning [P > 0.05, Table 3].

Furthermore, there was no significant difference in the treatment protocol (P > 0.05) and length of hospital stay (P = 0.64) between ALP and ZnP poisoning. 63.3% of the patients survived and 36.7% died. Even though the hospital mortality in ALP poisoning with a frequency of 39.2% was greater than ZnP poisoning with 22.2%, the difference was not statistically significant [Table 4].

There was significant correlation between mortality and on admission systolic blood pressure (P = 0.001; r = −0.42), O_2 saturation (P = 0.03; r = −0.47), sodium bicarbonate (HCO_3) (P = 0.002; r = −0.42), and BE (P = 0.003; r = −0.42) in venous blood gas analysis, blood glucose (P = 0.03; r = −0.32), prescription of magnesium sulfate (P = 0.000; r = −0.46), and calcium gluconate (P = 0.001; r = −0.42). Binary backward logistic regression analysis revealed that on admission venous HCO3, BE, admission systolic blood pressure,

Table 3: Comparison of hemodynamic and laboratory parameters of patients between aluminum phosphide and zinc phosphide poisoning at admission time (T0) and before discharge or death (T1)

Hemodynamic and laboratory parameters	Total (*n*=60)	ALP (*n*=51)	ZnP (*n*=9)	*P*
SBP (mmHg)				
T0	100.02±25.12	97.38±26.06	115.00±10.61	0.05
T1	104.64±21.74	102.23±23.00	114.28±12.72	0.19
DBP (mmHg)				
T0	64.65±12.80	63.90±13.26	68.89±9.28	0.28
T1	65.77±10.53	65.79±9.81	65.71±13.97	0.98
PR (bpm)				
T0	85.02±15.84	84.98±16.75	85.22±10.08	0.96
T1	83.91±9.01	83.88±10.03	84.00±3.65	0.97
RR (bpm)				
T0	18.61±8.85	19.04±9.49	16.22±2.90	0.38
T1	17.32±2.05	17.26±2.21	17.57±1.39	0.72
Temperature (°C)				
T0	36.84±0.34	36.82±0.36	36.93±0.09	0.36
T1	36.91±0.25	36.89±0.26	36.96±0.21	0.57
O_2 saturation (%)				
T0	93.75±3.74	93.61±3.93	95.00±0.02	0.63
T1	92.50±5.21	92.57±5.62	92.00±5.36	0.92
Na (mEq/L)				
T0	142.11±4.67	142.69±4.59	139.33±4.27	0.04
T1	140.30±4.02	142.14±2.67	136.00±3.46	0.01
K (mmol/L)				
T0	3.89±0.50	3.90±0.52	3.82±0.45	0.66
T1	3.70±0.42	3.71±0.49	3.67±0.21	0.88
BUN (mmol/L)				
T0	13.22±4.30	13.27±4.28	13.00±4.66	0.86
T1	17.67±10.34	18.55±20.11	15.00±4.58	0.77
Cr (mg/dL)				
T0	0.99±0.30	1.01±0.33	0.92±0.14	0.46
T1	1.22±1.12	1.28±1.30	1.03±0.21	0.76
FBS (mg/dL)				
T0	110.86±42.92	113.41±46.92	101.22±21.36	0.45
T1	122.00±46.21	127.25±57.52	111.50±20.51	0.73
WBC $\times 10^9$/L				
T0	10.75±5.11	10.96±5.32	9.83±4.18	0.55
T1	8.87±4.75	8.87±2.75	8.36±4.75	0.65
Hb (g/dL)				
T0	15.32±1.95	15.43±2.05	14.81±1.31	0.39
T1	14.96±2.92	14.96±2.92	13.06±1.26	0.06
Hct (%)				
T0	45.37±4.99	45.68±5.24	43.94±3.55	0.35
T1	44.58±6.83	44.58±6.84	43.15±4.65	0.55
Plt $\times 10^9$/L				
T0	242.91±87.78	248.66±88.37	218.67±66.27	0.34
T1	206.00±80.90	206.00±80.90	200.36±76.23	0.84
ALT (U/L)				
T0	26.76±47.32	26.97±10.75	25.40±13.72	0.94
T1	98.20±35.5	119.00±21.15	49.67±34.77	0.001
AST (U/L)				
T0	31.36±11.86	32.39±15.73	24.80±8.40	0.76
T1	60.60±56.29	65.86±15.39	48.33±13.32	0.002
PT (s)				

Contd...

Table 3: Contd...

Hemodynamic and laboratory parameters	Total (*n*=60)	ALP (*n*=51)	ZnP (*n*=9)	*P*
T0	15.07±1.72	15.07±1.58	15.08±2.59	0.98
T1	19.61±8.55	20.18±9.22	18.60±8.46	0.78
PTT (s)				
T0	47.23±29.81	42.70±23.44	45.17±29.18	0.16
T1	53.09±34.38	59.57±14.77	41.75±13.45	0.43
INR				
T0	1.33±0.25	1.33±0.24	1.32±0.35	0.91
T1	2.16±1.60	2.26±1.75	1.99±1.52	0.80
pH				
T0	7.29±0.17	7.29±0.18	7.32±0.05	0.68
T1	7.37±0.11	7.37±0.12	7.37±0.07	0.95
HCO_3 (mEq/L)				
T0	17.30±5.98	16.57±5.81	22.82±4.43	0.01
T1	20.65±4.67	20.33±4.96	22.55±1.63	0.39
PCO_2 (mmHg)				
T0	34.92±10.07	34.03±9.88	41.60±9.75	0.08
T1	36.19±10.51	35.83±10.62	38.27±11.12	0.67
BE				
T0	8.37±7.18	8.95±7.41	4.48±3.82	0.04
T1	4.75±3.58	5.47±3.64	2.05±1.53	0.09

T0 is admission time and T1 is discharge time. Chi-square or Fisher's exact test used for analysis. ALP=Aluminum phosphide, ZnP=Zinc phosphide, SBP=Systolic blood pressure, DBP=Diastolic blood pressure, PR=Pulse rate, RR=Respiratory rate, O_2sat=Oxygen saturation, Na=Sodium, K=Potassium, BUN=Blood urea nitrogen, Cr=creatinine, FBS=Fasting blood sugar, WBC=White blood cell, Hb=Hemoglobin, Hct=Hematocrit, Plt=Platelets, ALT=Alanine aminotransferase, AST=Aspartate aminotransferase, PT=Prothrombin time, PTT=Partial thromboplastin time, HCO_3=Bicarbonate, PCO_2=The partial pressure of carbon dioxide, BE=Base excess

Table 4: Comparison of the treatment approaches for the poisoned patients between aluminum phosphide and zinc phosphide poisoning

Treatment approach	Total (*n*=60)	ALP (*n*=51)	ZnP (*n*=9)	*P*
Calcium gluconate	8 (13.3)	8 (15.7)	0	0.33
Magnesium sulfate	9 (15.0)	9 (17.6)	0	0.33
Methylene blue	1 (1.7)	1 (2)	0	1.00
Vitamin C	14 (23.3)	13 (25.5)	1 (11.1)	0.67
Vitamin E	5 (8.3)	4 (7.8)	1 (11.1)	0.57
N-acetylcysteine	10 (16.7)	9 (17.6)	1 (11.1)	1.00
Dopamine	18 (30.0)	17 (33.3)	1 (11.1)	0.25
Hydrocortisone	10 (16.7)	9 (17.6)	1 (11.1)	1.00
Methylprednisolone	5 (8.3)	5 (9.8)	0	1.00
Metoclopramide	19 (31.7)	17 (33.3)	2 (22.2)	0.70
Milk of magnesium	3 (5.0)	2 (3.9)	1 (11.1)	0.39
Oil lavage	15 (25.0)	15 (29.4)	0	1.00
Endotracheal intubation	17 (28.3)	17 (33.3)	0	0.05
Mechanical ventilation	7 (11.7)	7 (13.7)	0	0.58
Time from ingestion to hospital presentation (min)	240 (120–420)	240 (120–420)	270 (187.50–390)	0.64
Length of hospitalization (day)	14.00 (4.50–24)	14.00 (4–24)	15.00 (6.50–51.50)	0.23
In hospital mortality	22 (36.7)	20 (39.2)	2 (22.2)	0.46

The Chi-square or Fisher's exact test used for analysis. ALP=Aluminum phosphide, ZnP=Zinc phosphide

blood glucose, and prescription of calcium gluconate were correlated with mortality [Table 5].

Discussion

The general objective of this study was to evaluate different factors in patients with phosphide poisoning during 2005–2014 years. The results of this study among 60 cases showed the prevalence in our referral centers was increasing during the years. Another study in our country has also showed this increasing rate from 2007 to

Table 5: Factors for outcome prediction in patients with phosphide poisoning

Variable	*P*	OR (95% CI)
Hypotension	0.007	4.87 (1.52–15.45)
Metabolic acidosis	0.019	13.00 (1.51–111.46)
Shock	0.002	8.50 (2.24–32.17)
Thirst	0.019	13.87 (1.54–124.81)
Dyspnea	0.01	8.40 (1.56–45.20)

OR=Odds ratio; CI=Confidence interval

2010.[25] Although most of the patients were men (65%), in some studies, men did not outnumbered women.[25] Poisoning was more observed in men, young, single, with suicidal intention similar to other studies.[19,26-30]

Sixty percent of patients were conscious on admission. Loss of consciousness was reported in those who referred late or ingested other medication including sedative-hypnotics. Shadnia *et al*. also reported loss of consciousness in 28.13%.[26] Several studies reported neurological toxicity of ALP and ZnP resulting in clinical effects such as headache, stupor, restlessness, agitation, anxiety, ataxia, paresthesia, and central nervous system depression, leading to coma and seizures.[31-34] In our study, we reported one case of seizure after overdosing ALP, one patient with stupor, two patients with coma, and restlessness as a common manifestation of this type of poisoning.

Vomiting was the most common symptom in our patients (60%) similar to others.[20,21,31] Gastrointestinal manifestations are the earliest symptoms that appear after ingestion. ALT and AST were far higher in those poisoned with ALP than those poisoned with ZnP at discharge. Probably liver toxicity was more prominent in those poisoned with ALP.

Metabolic acidosis was the most abnormality laboratory finding observed in our study similar to other studies.[1,3,26,27,30,31,35] Wide QRS complex and junctional rhythm was observed only in 8.3% of our cases. Several studies also noted ECG abnormalities in 36%–91% of patients.[36,37] In a study in India, cardiac dysrhythmias were described as atrial fibrillation (4% to 61%), junctional rhythm (4% to 100%), ventricular and atrial extrasystoles, and ventricular tachycardia.[38] We found no significant correlation between ECG abnormalities and outcome. However, in the study by Shadnia *et al*. in 20 poisoned patients, there was a significant correlation between cardiac manifestation, ECG findings, and TnT-positive results with mortality in acute ALP poisoning.[26] This difference in results may be related to the low number of patients with arrhythmia in our study or may be due to different severity of toxicity.

Outcome was correlated with on admission venous HCO_3, BE, systolic blood pressure, and blood glucose in our patients. Other studies showed that lack of vomiting after ingestion, high dose of ALP, electrolytes abnormalities, acute renal failure, impaired hemostasis and hyperleukocytosis were correlated with mortality.[31,36,38-43] In our study, some of these factors were not found to influence significantly the outcome. As electrolyte abnormality, acute renal failure and hyperleukocytosis were not observed in any cases at admission time. However, similar to our results, hyperglycemia, acidosis, and hypotension have been associated with mortality in some studies.[26,38-41] In addition, we found patients received calcium gluconate had the better outcome. However, the large odds ratio in some factors evaluated in our study may reduce the strength of this association in clinical practice. The difference of our predictive factors with other studies may be due to the different time of evaluation, as we analyzed on admission clinical manifestations and laboratory findings for outcome prediction. However, in other studies, this issue is not obvious whether the correlation between mortality with clinical manifestations was on admission time or during hospitalization.

The overall mortality rate was 36.7%. The mortality rate of ALP poisoning is highly variable, ranging from 37% to 100%.[38,39,42,44,45] The lower mortality compare to some studies may be due to lower ingested dose of phosphide which was not reported in this study and could be a limitation of the study. There is no standard antidote for this type of poisoning[2,3,16] and because the most type of poisoning is intentional, it is recommended to minimize access to these poisons and set strict rules for their usage, suicide prevention, and public education.

There are some limitations in our study. The number of patients with ZnP poisoning was small to be able definitely compare the different factors between these two types of phosphides. We also evaluate the clinical manifestations and laboratory findings on admission time. Since many signs and symptoms appear during hospitalization, the study comparing all clinical manifestations during different times of hospitalization until discharge or death would be more helpful.

It is concluded that the rate of phosphide poisoning is increasing during the years in our referral poisoning emergency center. It is more common in men and young people with suicidal intention. Mortality was higher in ALP than ZnP. On admission, systolic blood pressure was an important predictive factor for mortality. Serum bicarbonate, systolic blood pressure, Na level, and BE were significantly different between patients with ALP and ZnP poisoning.

Authors' Contribution

N. Eizadi-Mood, A. Yaraghi, and A.M. Sabzghabaee did the study's concept and design and data interpretation. N. Eizadi-Mood, M. Momenzadeh, A. Yaraghi, A.M. Sabzghabaee, and S. Alfred revised it critically for important intellectual content. M. Sadeghi performed the acquisition and interpretation of data and drafted the article. All authors did final approval of the version to be published.

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Conflicts of interest

There are no conflicts of interest.

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