PATTERN AND PREDICTORS OF DEATH FROM ALUMINUM AND ZINC PHOSPHIDE POISONING: A TWO YEARS PROSPECTIVE STUDY

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ABSTRACT

Background: Aluminum phosphide (ALP) and zinc phosphide pesticides are widely used as grain preservatives in Egypt. It was associated with high morbidity and mortality rates. **Aim**: To investigate the pattern and predictors of mortality related to aluminum and zinc phosphide poisoning. **Methods**: A two years prospective study was conducted on 399 patients with acute aluminum and zinc phosphide poisoning (124 non-survivors and 275 survivors) who were admitted to Menoufia University Poison and Dependence Control center (MPCC), during the period from 1st of July 2016 to the end of June 2018. For each patient, demographic and poisons data were taken. Also, clinical examination, laboratory investigations, ECG and Echocardiography were performed. **Results**: Phosphide poisoning predominated among cases aged < 18 years (46.8%), females (68.7%), students (48.9%) who attempted suicide (92.2%). The majority of non-survivors took aluminum phosphide tablets (97.6%) while most of survivors took zinc phosphide powder (78.5%). There was a significant difference as regards poisoning severity score (PSS), severe PSS among non-survivors (91.9%) while mild PSS among survivors (77.4%). Severe left ventricular dysfunction was the most predicting factor for mortality in the studied cases (OR= 9.35; CI: 1.156-5.26, P <0.001) followed by higher dose, need for mechanical ventilation, more than 2 hours' time delay, acidosis and hypotension. **Conclusion**: The mortality from aluminum and zinc phosphide poisoning can be predicted by intake one tablet or more of AlP with delayed time from intake to seek medical care, left ventricular dysfunction, hypotension and acidosis. These finding necessitate the need for public health policies to restrict the accessibility and use of phosphides in Egypt with the awareness of the public to the high fatality of these poisons.

Keywords: Pattern; Predictors; Deaths; Aluminum phosphide (ALP); Zinc phosphide.

INTRODUCTION

Aluminum and zinc phosphides pesticides are used worldwide to prevent the harmful effects of pests and rodents on stored grains. Phosphine gas was released when aluminum phosphide contact with water or hydrochloric acid in the stomach. The toxic effects of phosphine was due inhibition of cytochrome oxidase system in the mitochondria and consequent impairment of tissue respiration **(Valmas et al., 2008).** In addition, phosphine had a fatal direct toxic effects on cardiac muscles and adrenal glands leading to a circulatory collapse and may lead to death **(Shah et al., 2009)**.

Aluminum phosphide (AlP) poisoning mortality rates vary from 40% to 80% worldwide (**Chaudhry & Rai, 2014**). The identified fatal dose of AlP is 0.15–0.5 g. The time elapsed from ingestion of AlP to death ranged from about from1to48 hours, most of the patients die within the first 24 hours from poison intake. The most common cause of death as reported by previous studies was cardiac dysrhythmia (**Wahab et al., 2009**). Also, the mortality rate of zinc phosphide poisoning is around 37–100% (**Sogut, 2011**).

Aluminum and Zinc phosphide, effective grain preservatives are frequently used in silos of Menoufia governorate. However, exposure to these cheap and easily accessible chemicals has provoked poisoning with a high fatality rate. Therefore, this study was conducted to identify the pattern of aluminum and zinc phosphide poisoning and investigate their predictive factors of mortality.

SUBJECTS AND METHODS

This was a prospective study conducted on all cases of aluminum and zinc phosphide poisoning who were admitted to Menoufia University Poison and Dependence Control Center (MPCC), Egypt during the period from the 1st of July 2016 to the end of June 2018. MPCC is the only referral center for the cases of poisoning in Menoufia governorate.

A total of 399 cases of phosphide (180 aluminum phosphide and 219 zinc phosphide poisoned cases) poisoning were recruited in the present study. Data was obtained from the patient or his relative about; socio-demographic data (including age, sex, residence, marital status, occupation and smoking habits), intoxication data (type of phosphide poison, manner of intake, dose, formula, time elapsed since intake), data regarding clinical assessment; clinical poison severity score according to the method described by **Persson et al. (1998)**. Investigations like measurement of arterial blood gases, blood electrolytes, pH, liver and kidney function tests were performed. Silver nitrate test in gastric content were analyzed for all the studied cases (**Ahmed, 2017**). Serum cholinesterase levels were assessed to all the studied cases (**Waber, 1966**). In cases with positive silver nitrate test and decreased serum cholinesterase levels, thin layer chromatography was performed for exclusion of mixed toxicity with organophosphate or carbamate (**Kaplan et al.,1989**).

ECG and Echocardiography were performed and left ventricular function was assessed by ejection fraction (**Yancy et al., 2013**). In addition, the outcome of all cases was reported.

Ethical considerations*:* The study was approved by Menoufia University ethical committee. Informed valid consent was taken from each patient or their relatives prior to the beginning of the study. To insure confidentiality, samples were coded and anonymously stored.

Statistical analysis: The statistical analysis was performed by using IBM personal computer and statistical package SPSS version 22. The description of the data was done in the form of percentage (%) for qualitative data and mean and standard deviation (SD) for qualitative data. For comparison between survivors and non-survivor groups, student t- test was used for quantitative variables and the Chi-square (χ^2) test was used for categorical variables. Binary logistic regression analysis was performed to test the independent effects of the predictors of death from aluminum and zinc phosphides poisoning. P value <0.05 was considered statistically significant.

RESULTS

Three hundred ninety nine patients had aluminum (180 cases) and zinc (219 cases) phosphide poisoning. The commonest age of the studied cases was less than 18 (46.9%) and from 18-30 (33.6%) years. Two hundred seventy four patients (68.7%) were female and 125 (31.3%) were male. Most of cases were from rural area (65.9%). About half of the cases were students (48.9%) and about two thirds of the series were single (67.2%). The need for admission to ICU and mechanical ventilation (respiratory rate > 35/min, PaO_2 < 50 mmHg at home air, $PaCO_2$ >60 mmHg, blood pressure < 80/50, severe metabolic acidosis and patient isn't respond to supportive to treatment) were significantly more among non-survivors than survivors (P<0.001) as shown in **Table 1**.

There was a significant difference between survivors and non-survivors as regards the poison type (P<0.001). The majority of non-survivors took aluminum phosphide tablets (97.6%) while most of survivors took zinc phosphide powder (78.5%). Suicide attempts were the predominant manner in all cases (92.2%) while accidental intake presented in only (7.8%) of them. Oral intake was the route of intake in all cases. Tablets in a dose of more than one were significantly predominant among non-survivors than survivors while zinc phosphide powder in a dose of less than half packet was significantly predominant among survivors than non-survivors. The referred cases were significantly more in non-survivors than survivors (P<0.001). Time delay between poison ingestion and medical care of > 2 hours was significantly more predominant in non-survivors than survivors (P<0.001) as shown in **Table 2**.

As regards clinical manifestations in **Table 3**, the presentation with consciousness changes, hypothermia, bradycardia, hypotension, tachypnea, cyanosis, abdominal pain, vomiting and severe poisoning severity score was significantly more frequent in non-survivors than survivors (P<0.001).

Investigations of the studied patients were illustrated in **Table 4**. It showed that, elevated serum creatinine, hypokalemia, hyperkalemia, hypomagnesaemia, metabolic acidosis and hypoxemia were significantly more prevalent among the non-survivors than survivors. Moreover, moderate to severe left ventricular dysfunction as assessed by Echocardiography were more presented in the non-survivors than survivors (P<0.001).

Based on binary logistic regression analysis for relevant predictors of mortality in aluminum and zinc phosphide poisoning cases, severe left ventricular dysfunction was the most predicting factor for mortality in the studied cases (OR= 9.35; CI: 1.156-5.26, P <0.001) followed by aluminum phosphide dose of ≥ 1.7 gram (OR= 7.65 and 7.30; respectively). Moreover, need for mechanical ventilation (for hypoxemia or severe metabolic acidosis or hypotension not responding to supportive treatment), more than 2 hours' time delay, metabolic acidosis and hypotension were significant predictors of death in phosphide poisoning cases (OR= 5.36, 2.65, 1.86 and 1.46; respectively) as shown in **Table 5**.

Table 1: Characteristics of aluminum and zinc phosphide poisoning cases as regards their outcome

Characteristics	Non-survivors (n=124) No (%)	Survivors (n= 275) No (%)	Total (n=399) No (%)	χ^2	p-value
Age (years): < 18 18-30 30-40 > 40	65 (52.4) 42 (33.9) 10 (8.1) 7 (5.6)	122 (44.4) 92(33.5) 42(15.2) 19(6.9)	187 (46.9) 134 (33.6) 52 (13.0) 26 (6.5)	4.8	0.187
Sex: Males Females	31 (25.0) 93 (75)	94(34.2) 181(65.8)	125 (31.3) 274 (68.7)	3.35	0.067
Residence: Rural urban	90 (72.6) 34 (27.4)	173(62.9) 102(37.1)	263 (65.9) 136 (34.1)	3.56	0.059
Marital Status: Single married	89 (71.8) 35 (28.2)	179 (65.1) 96 (34.9)	268 (67.2) 131 (32.8)	1.73	0.188
Occupation: Employers Farmers &workers House wife Students Not working	9 (7.3) 10 (8.1) 30 (24.2) 68 (54.8) 7 (5.6)	14 (5.2) 51 (18.5) 65 (23.6) 127(46.2) 18(6.5)	23 (5.8) 61 (15.3) 95 (23.8) 195 (48.9) 25 (6.2)	8.27	0.082
Smoking habits: Smokers Non-smokers	8 (6.5) 116 (93.5)	34 (12.4) 241 (87.6)	42 (10.5) 357 (89.5)	3.17	0.075
Admission to ICU: Yes No	108 (87.1) 16 (12.9)	149 (54.2) 126 (45.8)	257 (89.5) 42 (10.5)	40.39	<0.001
Duration of stay in ICU: (days:	2.10±0.34	4.56±0.21	3.51±0.68	71.51	<0.001
Duration of hospital stay (days)	2.91±0.16	9.12±0.03	6.39±0.12	620.77	<0.001
Need for mechanical ventilation: Yes No	98 (79.0) 26 (21.0)	33 (8.4) 242 (91.6)	131 (32.8) 268 (67.2)	174.14	<0.001

Table 2 : Poisoning data of aluminum and zinc phosphide poisoning cases as regards their outcome.

Poisoning data	Non-survivors (n=124) No (%)	Survivors (n= 275) No (%)	Total (n=399) No (%)	χ^2	p-value
Types of poisoning: Zinc phosphide Aluminum phosphide	3 (2.4) 121 (97.6)	216 (78.5) 59 (21.5)	219 (54.9) 180 (45.1)	200.02	<0.001
Manner of poisoning: Suicidal accidental	119 (95.9) 5 (4.1)	249 (90.5) 26 (9.5)	368 (92.2) 31 (7.8)	3.51	0.061
Route of intake: Oral	124 (100.0)	275(100.0)	399 (100.0)	-	-
Formula: Tablets Powder	121 (97.6) 3 (2.4)	59 (21.5) 216 (78.5)	180 (45.1) 219 (54.9)	200.02	<0.001
No of Alp tablets*: (n=180) < 1 ≥1	18 (14.5) 103 (83.1)	53 (19.3) 6 (2.2)	71 (17.8) 109 (27.3)	93.53	<0.001
No of zinc phosphide powder packets: (N=219)** **<0.5** **0.5-1**	0 3(2.5)	213 (77.4) 3 (1.1)	213 (53.4) 6 (1.5)	107.98	<0.001
Time from intake to admission (hours): 1-2 > 2	12 (9.7) 112 (90.3)	226(82.2) 49(17.8)	238 (59.6) 161 (40.4)	186.66	<0.001
Referred from other medical care unit: Yes No	63 (50.8) 61 (49.2)	89(32.3) 186(67.6)	152 (38.1) 247 (61.9)	12.33	<0.001

*AlP in 3 grams tablet (57% AlP; 1.7 g/tablet)

**: Zinc phosphide in 5 grams powder packet (80% zinc phosphide; 4 g/packet)

Table 3: Clinical manifestations of aluminum and zinc phosphide poisoning cases as regards their outcome.

Clinical manifestations	Non-survivors (n=124) No (%)	Survivors (n= 275) No (%)	Total (n=399) No (%)	χ^2	p-value
Level of consciousness: Conscious Drowsy or confused	52 (41.9) 72 (58.1)	217(78.9) 58(21.1)	269 (67.4) 130 (32.6)	53.19	. <0.001
Temperature: Normal Hypothermia	109 (87.9) 15 (12.1)	271(98.5) 4(1.5)	380 (95.2) 19 (4.8)	21.34	<0.001
Pulse: Normal Tachycardia Bradycardia	10 (8.1) 46 (37.1) 68 (54.8)	210 (76.4) 60 (21.8) 5 (1.8)	220 (55.1) 106 (26.6) 73 (18.3)	211.13	<0.001
BP: Normal Hypotension	27 (21.8) 97 (78.2)	216 (78.5) 59 (21.5)	243 (60.9) 156 (39.1)	115.68	<0.001
RR: Normal Rapid	25 (20.2) 99 (79.8)	207(75.3) 68(24.7)	232 (58.1) 167 (41.9)	106.66	<0.001
Skin colour: Normal Cyanosed	63 (50.8) 61 (49.2)	272(98.9) 3(1.1)	335 (84.0) 64 (16.0)	146.84	<0.001
Vomiting: Present Absent	118 (95.2) 6 (4.8)	208(75.6) 67(24.4)	326 (81.7) 73 (18.3)	21.80	<0.001
Abdominal pain: Present Absent	92 (74.2) 32 (25.8)	219(79.6) 56(20.4)	311 (77.9) 88 (22.1)	1.47	0.225
Poisoning severity score: None Mild Moderate Severe	0 0 10 (8.1) 114 (91.9)	56(20.4) 213(77.4) 6(2.2) 0	56 (14.0) 213 (53.4) 16 (4.0) 114 (28.6)	381.49	<0.001

Table 4: Investigations of aluminum and zinc phosphide poisoning cases as regards their outcome.

Investigations	Non-survivors (n=124) No (%)	Survivors (n= 275) No (%)	Total (n=399) No (%)	χ^2	p-value
Creatinine: Normal Elevated	107 (86.3) 17 (13.7)	270 (98.2) 5 (1.8)	377 (94.5) 22 (5.5)	23.20	<0.001
K level: Normal Hypokalemia hyperkalemia	94 (75.8) 24 (19.4) 6(4.8)	251(91.3) 21(7.6) 3(1.1)	345 (86.5) 45 (11.3) 9(2.2)	11.73	0.001
Magnesium level: Normal Hypomagnesaemia	113 (91.1) 11 (8.9)	267(97.1) 8(2.9)	380(95.2) 19(4.8)	6.70	0.010
pH: Normal Acidosis	14 (11.3) 110 (88.7)	253 (92.0) 22 (8.0)	267 (66.9) 132 (33.1)	251.47	<0.001
NaHCO$_3$: Normal Decreased	19 (15.3) 105 (84.7)	215 (78.2) 60 (21.8)	234 (58.6) 165 (41.4)	139.24	<0.001
PaO$_2$: Normal Hypoxia	10 (8.1) 114 (91.9)	272(98.9) 3(1.1)	282 (70.7) 117 (39.3)	340.02	<0.001
PaCO$_2$: Normal Increased	102 (82.3) 22 (17.7)	272(98.9) 3(1.1)	374 (93.7) 25 (6.3)	40.35	<0.001
EF: Normal **(50-70%)** Mild dysfunction(40-49%) Moderate dysfunction(30-39%) Severe dysfunction (<30%)	0 2 (1.6) 44 (35.5) 78 (62.9)	216(78.5) 53(19.3) 6(2.2) 0	216 (54.2) 55 (13.8) 50 (12.5) 78 (19.5)	365.35	<0.001
Silver nitrate screening: Negative Positive	0 124 (100)	78(28.4) 197(71.6)	78 (19.5) 305 (80.5)	43.72	<0.001
Decreased cholinesterase level **Yes** **No**	6 (4.8) 118(95.2)	10(3.6) 265(96.4)	16 (4.1) 383(95.9)	0.32	0.571

EF: ejection fraction

Table 5: Binary logistic regression analysis for relevant predictors of mortality in aluminum and zinc phosphide poisoning cases.

Predictors	P-value	OR	95% C.I.	
			Lower limit	Upper limit
EF:				
Normal		Reference		
Mild dysfunction	0.180	0.36	0.081	1.603
Moderate dysfunction	0.006	2.16	1.032	4.668
Severe dysfunction	<0.001	9.35	1.156	5.264
Types (Aluminum phosphide)	<0.001	7.65	1.65	3.697
≥ 1.7 gram of AlP	<0.001	7.30	1.317	5.293
Need for mechanical ventilation	<0.001	5.36	1.695	3.689
Time delay (> 2 hours)	0.021	2.65	0.023	0.392
pH (acidosis)	0.005	1.86	1.368	1.871
Hypotension	0.014	1.46	1.080	1.977

OR, odds ratio; CI, confidence interval

Duration of hospital stay, duration of stay in ICU, poisoning severity score, O_2 levels

were included in regression model but weren't significant.

DISCUSSION

Aluminum and zinc phosphide are extremely toxic compounds and resulted in a high case fatality rate (124/399 cases; 31.1 %) in the present study. This result was in agreement with a retrospective 7 years study from 2000–2007 in Tehran that showed an overall mortality rate of 31 % due to aluminum phosphide (**Shadnia et al., 2009)**.

The case fatality rate from aluminum phosphide alone was about 67% (121/180 cases) and constituted the majority of deaths among all phosphide deaths (121/124 cases; 97.6%). Moreover, acute toxicity from aluminum and zinc phosphide constituted the first leading cause of death (124/153 cases; 81% of total deaths) among all deaths from poisoning cases admitted to Menoufia University Poison and Dependence Control Center (MPCC), Egypt during the period of the present study. These results reflect the bad prognosis and high fatality of phosphide poisoning that need urgent intervention and implementation of policies to ban its use and accessibility. Also, this study revealed that, left ventricular dysfunction, aluminum phosphide poisoning with dose ≥1.7 gram, hypotension, blood pH, and time delay from consumption of poisoning to treatment were found to be the most important predictors of death in phosphide poisoning cases.

Suicide was the most common manner of phosphide poisoning as these compounds are easy to access and cheap with rapid action and high fatality. This may direct persons to use aluminum and zinc phosphide for suicide. Similar findings were reported by previous study from Iran (**Mehrpour et al., 2008)** and India (**Louriz et al., 2009)**. Many factors such as family conflicts, drug and alcohol abuse, emotional distress, depression, medical diseases, social isolation, and financial and work-related problems may contribute to suicidal acts (**Konradsen et al., 2003)**

The most common age for phosphide poisoning in the present study was less than 18 years followed by those aged from 18-30 years as these ages are more susceptible to social and psychological stress so become more vulnerable to commit suicide **(Alinejad et al., 2017)**. They also may have conflict with other family members or friends and emotional problems **(Chang et al., 2011)**. Also, **Mathai and Bhanu (2010)** found that the majority of acute aluminum phosphide poisoning patients were young and in the age group from 21 to 40 years.

Aluminum and zinc phosphide poisoning was more common in females than males in our study with a ratio of about 2:1 as females attempt suicide greater than in males due to females restricted freedom, harmful traditional practices and frustration of females in Egyptian community from other family members. These results were in agreement with another Egyptian study **(El Naggar et al., 2011).** On the contrary, **Darbari et al., 2008 and Saha et al., 2015** reported that aluminum phosphide poisoning more common in males than females. This disagreement may be due different life style and different traditions between different countries.

More than two thirds of cases were single (67.2%) as married persons are more emotionally and psychologically stable. Also, about half of the cases were students (48.9%), this could be explained as students are susceptible to more emotional stress and conflict with others and hence more tendency to commit suicide. Housewives, farmers and workers were also common as these slices are more involved in grain storing and so phosphides toxicity.

Most of cases belonged to the rural area. Menoufia is an agriculture governorate, also grain storing is more in rural than urban areas. This is in accordance with previous studies **Kapoor et al. (2006)** who found that 65.1% of

aluminum phosphide poisoning cases were from rural areas.

The first presentation of phosphide poisoning were normal in most patients especially who transferred to the hospital within the first hours of poison ingestion. Systemic symptoms developed within the first 24 hours as vomiting and abdominal pain, hypotension, tachycardia, tachypnea, cyanosis and changes in the levels of consciousness. Finally, the patients proceed to multi-organ failure and death within the first 24 to 48 hours after poisoning. Similar findings were also reported in many previous studies from different countries **(Anger et al., 2000; Nocera et al., 2000; Navabi et al., 2018).**

The fatality from phosphide poisoning showed a statistically significant relationship with many variables including aluminum phosphide in a dose $\geq$ 1.7 gram (one tablet or more). Aluminum phosphide in Egypt is available in a 3 grams tablet form (containing 57% aluminum phosphide active ingredient equivalent to 1.7 g/tablet) while zinc phosphide is formulated as a 5 grams powder packet (containing 80% zinc phosphide active ingredient; 4 g/packet) **(National Pesticide Information Center, 2011).** This was supported by **Hajouji et al. (2006)** who stated that death from aluminum phosphide is common if more than 1.5 g is ingested.

Zinc phosphide is less dangerous than aluminum phosphide because it does not produce as much phosphine gas. This is due to zinc phosphide requires acidic conditions for appreciable hydrolysis and subsequent formation of phosphine, whereas aluminum phosphide will hydrolyze to form phosphine in neutral pH **(Knight, 2006)**.

Left ventricular dysfunction was still the most predictor factor for mortality in phosphide poisoning in this study after multivariate logistic regression analysis; severe and moderate left ventricular dysfunction was the most predicting factor of death followed by the need for mechanical ventilation, aluminum

phosphide in a dose ≥ 1.7 gram, hypotension and acidosis as cardiogenic shock secondary to toxic myocarditis induced by phosphine release was reported as the most common cause of death following aluminum phosphide poisoning in many previous studies **(Louriz et al., 2009; Mehrpour et al., 2012; Proudfoot et al., 2009).**

Metabolic acidosis, hypokalemia, hyperkalemia, hypomagnesaemia, hypoxemia and elevated serum creatinine, were significantly more prevalent among the non-survivors than survivors. Hypokalemia might be contributed to vomiting that presented in 95.2% of non-survivors. These findings were in agreement with **Gurjar et al. (2011)** who reported that manifestations like refractory shock, metabolic acidosis, severe hypoxia and electrolyte imbalance were observed in cases of AlP poisoning and they are associated with poor prognosis. This could be explained by the release of phosphine gas which inhibits mitochondrial cytochrome oxidase and cellular oxygen utilization and so causing cellular hypoxia and metabolic acidosis **(Wang et al., 2007)**.

It has been found that organs with a higher need for oxygen as heart, lung, kidney, and liver are more sensitive to damage by phosphine gas as phosphine also produces oxygen free radicals in body tissues. In AlP poisoning patients may remain conscious till the late stage but can have manifestations like headache, dizziness, confusion, convulsion and coma due to affection of aerobic metabolism of cerebral cells by inhibition of mitochondrial cytochrome oxidase enzyme. Blockage of oxidative phosphorylation and poor tissue perfusion lead to accumulation of lactic acid and metabolic acidos**is (Goel & Aggarwal, 2007).**

Moghadamnia (2012) and **Chugh, et al. (1990)** reported that decreased plasma magnesium and decreased or increased potassium levels may occur and presence

of metabolic acidosis indicates moderate to severe AlP overdose.

The present study showed that 80.5% of the cases were positive silver nitrate screening test. It is a chemical qualitative color test that was done to detect phosphine (PH3) in stomach contents. Similarly **Soltaninejad et al. (2009)** indicated that PH3 detection with silver nitrate test is the commonest and most valuable in clinical and forensic tests that can be done on bio samples such as stomach content and expiratory air and is sensitive enough to detect very low concentrations of PH3 as 0.05 mg L-1(0.05 ppm).

Positive silver nitrate screening test was more prominent in non-survivors than survivors and this may be due to the fact that non-survivors usually ingest higher amounts of the poison hence more concentration of PH3 in the stomach content.

In this study only 4.1% showed decreased cholinesterase level (by about 65% of normal) but with no relevant manifestations of cholinesterase inhibition. Many investigations previously reported that phosphides inhibit cholinesterase activity as **Saidi and Shojaie. (2012)** who found that plasma cholinesterase levels were inhibited in rat group receiving ALP. Also **Hashemi-Domeneh et al. (2016)** and **Mittra et al. (2001)** stated that both ALP and phosphine cause cholinesterase inhibition, but this inhibition is unlikely to be clinically relevant.

In addition, More than 2 hours' time delay to attend medical care was still predictors of death in phosphide poisoning patients on multivariate logistic regression. Similarly, **Navabi et al.,** reported that the elapsed time from consumption to treatment was 1.3±0.7 and 2.0±1.4 hours for survivors and non- survivors of ALP poisoning, respectively. Therefore, accelerating the transport of poisoned individuals to treatment centers, followed by careful attention to blood pressure and blood pH at the time of treatment, would

be expected to reduce the risk of mortality in cases of aluminum phosphide poisoning.

Regarding period of stay in hospital, it was longer in survivors than non-survivors as most non-survivors came to emergency room with very severe cardiogenic shock that caused death in less than 48 hours in the majority of the cases.

A significantly higher percentage of non survivors than survivors were referred cases. this Only one factor of the cause of the previous finding is improper management; other factors may play a role as remoteness of the cases taking long hours to reach medical care or delay due to undetected cases (non- revealing suicidal patients). Also, gastric lavage might have been done by water which enhances release of phosphine gas in the stomach and hence more toxicity

The present study had some limitation as it was conducted in a single center, the findings may not be representative to the general population in Egypt but it can provide useful data for healthcare providers about the pattern of phosphide poisoning and risk factors of mortality from these types of pesticides.

CONCLUSIONS & RECOMMENDATIONS

The results of this study showed that mortality can be predicted in patients with phosphide poisoning based on their type whether aluminum or zinc phosphide, formula and dose of poison. In addition to, left ventricular function, blood pressure, blood pH, and time delay from consumption to treatment. These finding necessitate the need for public health policies to restrict the access and use of phosphides in Egypt with the awareness of the public to the high fatality of these poisons and to encourage the healthcare providers to take more effective measures to treat patients with phosphide poisoning.

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