Study of the Causes of Mortality in Acute Aluminium Phosphide Poisoning

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KEYWORDS
Aluminium phosphide,
Suicide,
Mortality,
Shock,
Acidosis,
Arrhythmia

ABSTRACT

Aluminum phosphide (ALP) poisoning has aroused interest in the past three decades. The percentage of poisoning is low, but the mortality is high and no effective antidote available. The objective of this study was to find out predictors of mortality for patients with acute aluminium phosphide poisoning. All patients with acute aluminium phosphide poisoning admitted to Poisoning Control Center, Ain Shams University Hospital between the years 2015 to 2017 were prospectively studied and compared between survival and non survivor patients. Data collected include demographic data, clinical manifestations, laboratory parameters, ECG and treatment offered to the patients. A total 31 patients were enrolled comprising 20 males and 11 females, 84% were suicide and mortality rate was 35%. Shock and cardiac arrhythmia were observed in 52% and39% respectively, while 26% presented with coma. Abnormal blood sugar and metabolic acidosis were found in 19% and 45% respectively. Fifty two percent of the patients needed inotropic therapy and 32% received N-acetylcysteine (NAC). Risk factors increasing mortality were found as shock, tachycardia, coma, metabolic acidosis, hyperglycemia, cardiac arrhythmia and the need for inotropic drug therapy. The study concluded that aluminum phosphide is a low-cost highly-toxic rodenticide. The circulatory collapse, metabolic acidosis and cardiac arrhythmia are the major causes of death. The role of NAC must be reassessed in larger scale. So, intensive observation in ICU and aggressive symptomatic management should be urgently taken into consideration.

Introduction

Aluminium phosphide (ALP) is used for crop protection in storage and transport, it improved the quantity and quality of agricultural products, yet the risk of mortality associated with phosphide poisoning in humans is high (Hassanian, 2014). After ingestion, phosphine will be released due to contact between aluminium phosphide and water/acid in the gastrointestinal (GI) tract. Phosphine gas can rapidly be absorbed by the lungs, GI tract and can easily be distributed in all tissues (Gurjar et al., 2011). At the cellular level, phosphine gas inhibits cytochrome C oxidase, oxidative phosphorylation, catalase and deplete glutathione, which may result in cellular wall damage (Anand et al., 2013).

Hypotension and shock ensue within 3-6 hours of ingestion due to arrhythmia, conduction disturbance, myocardial damage and excessive vomiting with fluid loss. Also shock can be attributed to wide spread small vessel injury leads to peripheral vasodilatation and direct toxic effects of phosphine on adrenal cortex accompanied by decreased cortisol levels. The major complications of ALP

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poisoning include cardiac arrhythmias, circulatory failure and severe metabolic acidosis. In survivors, the cardiotoxicity and hypoxia disappear within 5-7 days due to excretion of phosphine (Nosrati et al., 2013). The objective of this study was to find out predictors of mortality for patients with acute aluminium phosphide poisoning.

Subjects and Methods

This study is a prospective study included cases with acute ALP poisoned patients who were admitted to Poisoning Control Center; Ain Shams University Hospital (PCCA) from January 2015 to December 2017. The total number of cases is 31 cases; the diagnosis of acute ALP was based on history of consumption of the poison (obtained from the patient or the closest relative). Patients with an unclear diagnosis of poisoning or consumption of more than one substance were excluded from the study. The cases were classified according to the outcome into two groups: survivors and non survivors group. The data gathered included : age, gender, mode of poisoning (suicidal/ accidental), the delay in presentation to hospital, period of stay, conscious level, heart rate, blood pressure, presence or absence of vomiting, laboratory parameters include (blood sugar, acid base status, liver enzymes and renal function tests), ECG, the need of inotropic drugs and N acetylcysteine (NAC) therapy. All patients were admitted to ICU after initial resuscitation. A baseline electrocardiogram was recorded and blood samples for biochemical investigations were taken at time of presentation to the hospital.

Ethical consideration:

Permission for the study was obtained from the Director of the Poison Control Center, Ain Shams University Hospitals and the regional ethics committee. All data were stored anonymously. Relatives of recruited patients provided written informed consent for participation.

Statistical Analysis

Data were analyzed using IBM SPSS 22 statistical soft ware. The categorical data were reported in number and percentage and continuous data were reported as mean and standard deviation. The difference of frequency between two groups was analyzed by chi-square test for categorical data and by t- test for continuous data. Odds ratio (logistic regression analysis) were used to detect the predictor of outcome. P value less than 0.05 was considered statistically significant.

Results

As regard the gender, 65% of the patients were male, 84% were suicidal and all the non survivor patients were due to suicidal cause. The period of the stay was less than one day in71% of the patients and more than one day in 29% of the patients. The mean age was 23 ± 14 years in survivor group versus 25 ± 11 years in the non-survivor and the delay time was 5 ± 2h in the survivor group versus 4 ± 2h in the non-survivor. The statistical analysis revealed no significant difference between non survivor and survivor groups as regards, sex, mode of poisoning, age and delay time of presentation

to hospital and there is significant difference between both groups as regards period of stay at hospital and patients who died usually die in the first day. So, if patients pass the first day, there is a good chance to survive (Table 1).

Table (1): Statistical analysis of the gender, mode of poisoning, period of stay, age and delay time of presentation to hospital for both studied groups.

Parameters	Non survivor group n = 11	Survivor group n= 20	Total n = 31	Statistic test	p
Gender					
Males	7	13	20 (65%)	X^2_{ChS}= 0.006	0.9
Females	4	7	11 (35%)		
Mode of poisoning					
Suicidal	11	15	26 (84%)	X^2_{ChS} = 3.3	0.07
Accidental	0	5	5 (16%)		
Period of stay at hospital					
<1 day	11	11	22 (71%)	X^2_{ChS} = 6.9	0.008*
>1 day	0	9	9 (29%)		
Age					
Age (years) Mean ±SD	25 ± 11	23±14	24 ± 13	t = 0.4	0.7
Delay time of presentation to hospital					
Delay Time (hours) Mean ±SD	4 ± 2	5 ± 2	4.5 ± 2	t = 1.1	0.3

n: number, SD: standard deviation, * significant

As regard the clinical presentation, 39% of the patients presented with abnormal heart rate (nine patients in the non survivor group: four patients had no palpable pulse, three patients had tachycardia and two patients had bradycardia, versus three patients in the survivor group: one patient with no palpable pulse, one patient had tachycardia and one patient had bradycardia. Fifty two percent were shocked (all the patients in the non survivor group presented with shock versus five patients in the survivor group), 26% had coma and 48% had vomiting. The statistical analysis revealed significant difference between both groups as regard heart rate, blood pressure and level of consciousness (Table 2).

Table (2): Statistical analysis of the clinical parameters for both studied groups.

Parameters	Non survivor group n= 11	Survivor group n =20	Total n = 31	Chi-square test (χ^2)	p
Heart rate (HR)					
Abnormal Not palpable Tachycardia Bradycardia	9 (4) (3) (2)	3 (1) (1) (1)	12 (39%)	13.4	0.0002*
Normal	2	17	19 (61%)		
Blood pressure (BP)					
Shock	11	5	16 (52%)	15.9	0.0006*
Normal	0	15	15 (48%)		
Conscious level					
Coma	6	2	8 (26%)	7.4	0.006*
Normal	5	18	23 (74%)		
Vomiting					
Yes	7	8	15 (48%)	1.6	0.2
No	4	12	16 (52%)		

n: number, * significant

The laboratory analysis of studied patients revealed, 19% of the studied patients had abnormal blood sugar, 45% had metabolic acidosis, 10% had abnormal liver function test and no patients had renal abnormalities. The statistical analysis revealed significant difference between both groups as regards blood sugar and acid base status and no significant difference as regards liver and renal function tests (Table 3).

Table (3): Statistical analysis of the laboratory analysis for both studied groups.

Parameters	Non survivor group n= 11	Survivor group n =20	Total n= 31	Chi-square test (χ^2)	p
Random blood glucose level					
Abnormal a) Hyperglycemia b) Hypoglycemia	 5 1	 0 0	6 (19%)	13.5	0.0002*
Normal	5	20	25 (81%)		
Acid – base status					
Metabolic acidosis	10	4	14 (45%)	14.4	0.0001*
Normal	1	16	17 (55%)		
Abnormal liver function tests					
Yes	1	2	3 (10%)	0.007	0.9
No	10	18	28 (90%)		
Abnormal renal function tests					
Yes	0	0	0 (0%)	0	1
No	11	20	31(100%)		

n: number, * significant

The cardiac arrhythmia was apparent in 39% of the patients, (seven patients in the non survivor group had ECG abnormalities: two patients had ventricular tachycardia (VT), two patients had bradycardia, one patient had premature ventricular contractions (PVC), one patient had atrial fibrillation (AF) and one patient had ischemic changes, versus five patients in the survivor group: one patient had bradycardia, one patient had PVC, two patients with ischemic changes and one patient had nodal rhythm). The statistical analysis revealed significant difference between both groups as regard ECG abnormalities (Table 4).

Table (4): Statistical analysis of the ECG abnormalities for both studied groups

Parameters	Non survivor group n= 11	Survivor group n =20	Total n= 31	Chi-square test (χ^2)	p
Cardiac Arrhythmia					
Total number	7	5	12 (39%)	4.5	0.03*
VT	(2)	(0)			
Bradycardia	(2)	(1)			
PVC	(1)	(1)			
AF	(1)	(0)			
Ischemic changes	(1)	(2)			
Nodal rhythm	(0)	(1)			
Normal	4	15	19 (61%)		

n: number, * significant

The inotropic therapy was given in 52% of the patients and in 32% NAC was given and there is significant difference between both groups as regard the usage of inotropes and NAC (Table 5).

Table (5): Statistical analysis of the given therapy for both studied groups.

Parameters	Non survivor group n= 11	Survivor group n =20	Total n= 31	Chi-square test (χ^2)	p
Inotropic therapy					
Yes	11	5	16 (52%)	15.9	0.00006*
No	0	15	15 (48%)		
NAC therapy					
Yes	6	4	10 (32%)	3.9	0.05*
No	5	16	21 (68%)		

n: number, * significant

The mortality of the moderate to severe patients who received NAC was 60% versus 83% in those moderate to severe who did not receive NAC and no significant difference between both groups as regard the mortality (Table 6).

Table (6): Statistical comparison of mortality in patients who received NAC and patients did not receive NAC in the moderate to severe patients

	NAC therapy n =10	No NAC therapy n= 6	Chi-square test (x)	p
Survivor group	4 (40%)	1 (17%)	0.95	0.3
Non survivor group	6 (60%)	5 (83%)		
Total	10	6		

n: number.

A logistic regression analysis of the significant parameters revealed that the tachycardia, shock, coma, hyperglycemia, metabolic acidosis, cardiac arrhythmia and usage of inotropic therapy are predictors of mortality (Table 7).

Table (7): Logistic regression analysis of the significant parameters to detect predictors associated with aluminium phosphide poisoning-related mortality.

Parameters	Non survivor group n= 11	Survivor group n =20	OR (95%CI)	p
Heart rate (HR)				
Abnormal	9	3	26 4 to 182	0.001*
Normal	2	17		
Blood pressure (BP)				
Shock	11	5	65 3 to 1294	0.006*
Normal	0	15		
Conscious level				
Coma	6	2	11 2 to 71	0.01*
Normal	5	18		
Random blood glucose level (mg/dl)				
Abnormal	6	0	49 2 to 999	0.01*
Normal	5	20		
Acid – base status				
Metabolic acidosis	10	4	40 4 to 411	0.002*
Normal	1	16		
ECG results				
Cardiac arrhythmia	7	5	5 1 to 26	0.04*
Normal	4	15		
Inotropic therapy				
Yes	11	5	65 3 to 1294	0.006*
No	0	15		

n: number, * significant, OR = odds ratio, CI = confidence interval.

Discussion

Aluminum phosphide is a highly toxic pesticide that is used widely in agriculture since 1940s. Aluminum phosphide poisoning is common either accidental or suicidal reasons and is a serious public health problem in developing countries. Because of high toxicity and no existence of effective antidote, ALP poisoning has a high mortality rate (Vahdati et al., 2015).

The grading of the patients was done according to poisoning severity score (Persson et al., 1998), and revealed that 11 patients were asymptomatic, 4 patients had minor toxicity (presented with vomiting only) and 16 patients had moderate to severe toxicity (presented with end organ damage). The study revealed that the mortality rate was 35% (11 patients out of 31 patients). Similar results were obtained by Sulaj et al. (2015) who studied 317 ALP cases with 140 fatalities and death ratio of 44%. Also, Nosrati et al. (2013) found in Iran between 2000 and 2007 that ALP poisoning caused 146 deaths with mortality rate 24%. Higher incidence of mortality was noted by Aziz and Husain, (2015) where they studied 100 patients with ALP poisoning and total 78 patients died, depicting a 78% mortality rate. In contrast lesser mortality rate was detected by Lauterbach et al. (2005) where they reported in Germany, between 1983 and 2003, two deaths only out of 188 cases of ALP poisoning and in UK only one resulted in a death out of 93 cases of ALP poisoning.

The study revealed that male patients represent 65% of the cases and the mean age of the patients was 23±14 years in survivor group versus 25±11 years in the non survivor and no significant difference between both groups as regard gender and age. The predominance of males may be due to that aluminium phosphide is not a household product and it is commonly available in agricultural field and crop stores where male are more exposed.

Similar results were obtained by Aziz and Husain (2015) who studied 100 patients with ALP poisoning at Allied Hospital Faisalabad and found that 63% of the cases were males with mean age was 26.7 ± 7.9 years. Also, Khodabandeh et al. (2014) in their study found that male–female ratio in favor of men (55:45) and with mean age of patients was 26 ± 8 years. In contrast, Taghaddosinejad and Esmaeil. (2016) studied 63 Patients with ALP poisoning; they found that most of the patients were females (56.5%). Also, Sulaj et al. (2015) studied 317 AIP intoxications, the victims mainly belong to the third or fourth decade of life.

The study revealed that 84% of the cases were suicidal and 16%were accidental and no significant difference between both groups as regard mode of poisoning. Similar result was obtained by Sulaj et al. (2015) who did a study in five-year period registered a total of 317 AIP intoxication and (94%) were suicidal. Also, Nosrati et al. (2013) found in Iran more than 90 % of ALP poisonings were suicidal. While in contrast, Lauterbach et al. (2005) reported in Germany 188 cases of ALP poisoning between 1983 and 2003, 65 % of which was accidental and the remaining was intentional poisoning.

As regard clinical manifestation the results revealed that 39% of the patients had abnormal heart rate, 52% were shocked, 26% had coma and 48% had vomiting. The statistical analysis revealed significant difference between both groups as regard heart rate, blood pressure and level of consciousness. Similar results was obtained by Aziz and Husain, (2015) who studied 80 patients with acute ALP poisoning and found tachycardia in 85% of the patients, bradycardia in 15% and hypotension was observed in 94%. Moreover, Sulaj et al. (2015) found that among 140 patients, whom died out of 317 in his study of ALP, 92% had

cardiovascular collapse, 37% had coma and 46% had vomiting. Also, Erfantalab et al. (2017) demonstrated that blood pressure and pulse rate were significantly different between ALP survivors and non-survivors groups.

As regard laboratory analysis the results revealed that 19% had abnormal blood sugar, 45% had metabolic acidosis, 10% had abnormal liver function tests and no one had renal abnormalities. The statistical analysis revealed significant difference between both groups as regard blood sugar and acid base status. Similar results were obtained by Sulaj et al. (2015) who found that among 140 patients whom died out of 317 in his study of ALP poisoning, 88% had metabolic acidosis and 16% had hyperglycemia. Also, Erfantalab et al. (2017) demonstrated that the blood pH, and serum bicarbonate level were significantly different between survivors and non-survivors groups.

The results revealed that 39% had ECG abnormalities; the statistical analysis revealed that there was significant difference between non survivor and survivor groups as regarded ECG abnormalities. Also, Aziz and Husain, (2015) studied 80 patients with acute ALP poisoning and found that 10 % developed cardiac arrhythmia and the most frequent arrhythmia was atrial fibrillation (31% of patients) followed by ventricular fibrillation (20%), ventricular tachycardia (17%) and AV block (12%). Moreover, Sulaj et al. (2015) in their study of 317 ALP poisoned patients found that 44% had sinus tachycardia and 11% had other cardiac rhythm disorders.

The results revealed that 52% of the patients need inotropic therapy and in 32% NAC was received and there were significant difference between both groups as regard inotropic and NAC used. As regard NAC there was limitation in our study where we had 16 patients graded moderate to severe and must take NAC according to our local guidelines but due to unavailability of the drug only 10 patients received NAC so we cannot use their results in the prediction of outcome. At the same time we got benefit from this limitation in comparing between patients who received NAC and patients who did not received NAC in the 16 moderate to severe cases. As showing the mortality rate in the patients who received NAC was 60% (6 died out of 10 patients) while mortality in the group did not receive NAC was 83% (5 died out of 6 patients) and the statistical analysis revealed no significant difference between both groups.

Similar results obtained by Karanth and Nayyar (2003) where they found that treatment with NAC in ALP poisoning had no therapeutic advantage. In contrast, Bhat et al. (2015) found that among 26 patients treated with NAC out of 100 ALP poisoning patients, mortality in the treated patients was 2% versus 21.6% in non treated patients and the difference was statistically significant. Moreover, Tehrani et al. (2013) found that NAC prolong survival time, stabilize blood pressure and pulse rate. Also, Agarwal et al. (2014) found that NAC lower mortality, hospitalization time, and the frequency of intubation and mechanical ventilation.

The logistic regression analysis revealed that tachycardia, shock, coma, hyperglycemia, metabolic acidosis, cardiac arrhythmia and the need of inotropic therapy were predictors of mortality in acute ALP. Similar results were obtained by Mathai and Bhanu (2010) where they found that hypotension requiring vasoactive drugs, low pH, coma and need for mechanical ventilation were predictive of mortality from acute ALP poisoning. While Sulaj et al. (2015) found that the negative prognostic factors related to the fatalities were high dosage of the toxic agent, the long delay (a mean of 4 hours); the lack of vomiting, and the depth of coma upon presentation.

Conclusion

Aluminum phosphide is a low-cost highly-toxic rodenticide. The circulatory collapse, metabolic acidosis and cardiac arrhythmia are the major causes of death. The role of NAC must be reassessed in larger scale. So, intensive observation in ICU and aggressive symptomatic management should be urgently taken into consideration.

Recommendations

- People handling this fumigant must be aware of its lethal aspects.
- They should be prohibited from keeping and using this poison at the home.
- They should be advised to cover the tablets in open fields after use.
- They should keep their tablets away from the reach of children and other family members.
- Official health care system should restrict the open sales of this pesticide.
- Vendors and shop keepers should not sell the tablets to young people and children without proper verification and confirmation.
- If possible all of the phosphide derivatives compounds should be forbidden forever for everyone.

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دراسة أسباب الوفاة فى حالات التسمم الحاد بفوسفيد الالمونيوم

هانى محمد توفيق

مركز علاج التسمم مستشفيات جامعة عين شمس

أثار تسمم فوسفيد الألومنيوم الاهتمام بزيادة عدد الحالات في العقود الثلاثة الماضية. على الرغم من أن النسبة المئوية للتسمم منخفضة ، إلا أن معدل الوفيات مرتفع للغاية و لا يوجد ترياق فعال لعلاج مثل هذة الحالات. كان الهدف من هذه الدراسة هو اكتشاف تنبؤات للوفاة بالنسبة للمرضى الذين تعرضوا للتسمم الحاد بفوسفيد الألومنيوم . درسنا 31 مريضا يعانون من التسمم الحاد بفوسفيد الألومنيوم وتم دخولهم مركز علاج التسمم مستشفى جامعة عين شمس بين عام ٢٠١٥ إلى عام ٢٠١٧ وتمت مقارنة بين المرضى الناجين والمرضى المتوفين . وتشمل البيانات التي تم جمعها البيانات الديموغرافية ، والأعراض والعلامات السريرية ، والمعايير المختبرية ، و رسم القلب والعلاج المقدم للمرضى. النتائج: تم تسجيل ٣١ مريضا وكان منهم ٢٠ ذكرا و ١١ أنثى ، وكان نسبة المنتحرين ٨٤ ٪. و معدل الوفيات ٣٥ ٪. لوحظ هبوط بضغط الدم وتغير فى ضربات القلب في ٥٢ ٪ و ٣٩ ٪ على التوالي ،وكان ٢٦ ٪ أصيبوا بغيبوبة. و لوحظ اختلال فى نسبة السكر بالدم و زيادة حموضة الدم في ١٩ ٪ و ٤٥ ٪ على التوالي. لوحظ اضطراب فى تخطيط القلب في ٣٩ ٪ من المرضى.وكان ٥٢ ٪ من المرضى يحتاجون إلى علاج لرفع ضغط الدم وتلقى ٣٢ ٪ ان استيل سيستايين . ووجدنا العوامل التالية تزيد من خطر الوفاة : هبوط ضغط الدم، عدم انتظام ضربات القلب ،حدوث غيبوبة ، زيادة حموضة الدم، ارتفاع نسبة السكر في الدم ، اضطراب تخطيط القلب والحاجة إلى علاج لرفع ضغط الدم. و نخلص إلى أن فوسفيد الألومنيوم هو مبيد عالي السمية منخفضة التكلفة، ويعتبر هبوط الدورة الدموية، زيادة حموضة الدم وعدم انتظام ضربات القلب هي الأسباب الرئيسية للوفاة. لم يكن هناك ترياق فعال متاح للعلاج ويحتاج ترياق ان استيل سيستايين الى إعادة تقييم على نطاق اوسع، لذا يجب أخذ الملاحظة المكثفة في وحدة العناية المركزة والتدخل العلاجى السريع لاى أعراض تظهر دون تمهل .