Clinical Data, Laboratory Investigations and Electrocardiographic Changes as Predictors of Mortality in Acute Aluminum Phosphide Poisoning

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ABSTRACT

Aluminum phosphide (AlP) is a fumigant widely used in Egypt. Limited data is available about mortality prediction in AlP poisoned patients. This work was designed to evaluate role of clinical data, laboratory investigations and ECG findings in predicting mortality in these cases. A cohort retrospective study was conducted on 113 cases of AlP poisoning admitted to Tanta University Poison Control Center. History, clinical data and results of laboratory investigations were recruited. The mortality rate was 68.1%. There was a significant difference between survivors and non- survivors as regards mode of poisoning, GCS, vital signs (except heart rate), ECG abnormalities, pH, HCo3, K, creatine level and white blood cell count. The multivariate analysis logistic regression revealed that ECG changes and pH level were the only independent variables that can predict mortality. Analysis of receiver operating curve of blood pH level revealed a good area under the curve (AUC) of 0.881. At a cut off value of ≤7.28 the blood pH level had a sensitivity of 71.43. It was concluded that AlP poisoning causes high mortality rate. Mortality could be predicted by ECG changes and blood pH level. At a cut off value of ≤7.28 the blood pH level was able to predict mortality. ECG assessment and blood pH monitoring are highly recommended in cases of AlP poisoning for early prediction of mortality.

Introduction

Pesticides poisoning is one of the major public health problems. According to International Programme on Chemical Safety, consumption of pesticides results in 370000 deaths per year. Many of these deaths result from Aluminum phosphide (AlP) Poisoning (Prashar and Ramesh, 2018). In Egypt, it is widely used as a grain preservative because of its low cost and easy availability. Moreover, it is highly potent against broad spectrum of insect species, not affecting seed viability and leaves little residues on food grains (Mostafazadeh et al., 2011; Nagy et al., 2015).

When AlP comes in contact with water, moisture in the air, or hydrochloric acid in the stomach, it liberates toxic phosphine gas (PH3), which is rapidly absorbed by inhalation, ingestion and dermal contact (Navabi et al., 2018). The exact mechanism of action of AlP is still unknown; however, several mechanisms have been described for phosphides toxicity. Cellular damage and cardio-respiratory failure are the most common mechanisms of mortality and morbidity induced by AlP poisoning (Proudfoot, 2009; Hassanian - Moghaddam et al., 2016).

According to Bumbrah et al. (2012), cytotoxic phosphine gas generated due to acid

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hydrolysis of AlP affects heart, lungs, kidneys and gastrointestinal tract. AlP toxicity causes nausea, restlessness, abdominal pain, palpitation, pulmonary edema, cyanosis, hypotension, shock and cardiac arrhythmias. Other rare effects include hepatitis, acute tubular necrosis, disseminated intravascular coagulation and respiratory alkalosis. These detrimental effects have been described by several authors over a long period of time.

Predicting outcome and mortality in critically ill patients is a key component and a major concern of any health care system. Detrimental effects of AlP poisoning are a big problem with major consequences. In the same time, there is a striking lack of recent studies regarding predictors of mortality in acute AlP toxicity (Mathai and Bhanu, 2010; Khan, 2015).

Hereafter, the current study aimed to use clinical data, laboratory investigations and ECG findings in acute AlP intoxication as simple tools for prediction of mortality.

Subjects and methods:

Study design: This is a cohort retrospective study which was carried out in Tanta University Poison Control Center (TUPCC) after being approved by the research ethical committee of Tanta Faculty of Medicine (approval code: 33156/05/19).

Subjects: This study included data of 113 cases suffering from acute AlP poisoning in a 2 years period from the start of October 2016 to the end of September 2018. The data were recruited from the hospital records after approval from the head of TUPCC.

Inclusion criteria:

Cases (from both genders and all ages) who are suffering from acute AlP poisoning are included.

Exclusion criteria:

1- Cases with ingestion or exposure to other substances in addition to AlP.
2- Cases with other major medical conditions (e.g. cardiovascular disease, renal or hepatic failure).
3- Cases treated for acute AlP poisoning in any medical center before admission to TUPCC.
4- Cases with incomplete hospital records.

Data confidentiality was maintained by making code numbers (available to investigators only) for each patient. All the data were analyzed anonymously.

For each case, the following data were collected:

a) History:

1- Personal history with emphasis on age, gender and residence.
2- Toxicological history including; mode of poisoning, route of exposure and delay time.

b) Clinical data:

1- Vital signs (pulse, blood pressure, temperature and respiratory rate).
2- Level of consciousness by Glasgow Coma Scale (GCS).
3- Electrocardiographic (ECG) monitoring.

c) Results of laboratory investigations: (on admission)

Including results for:

- Arterial blood gases (ABG) with emphasis on pH and bicarbonate level.
- Serum sodium and potassium.
- Liver enzymes: serum aspartate transaminase (AST) and alanine transaminase (ALT) activities.
- Kidney function tests: blood urea and serum creatinine.

- Complete blood count (CBC) with emphasis on white blood cell count.
- Random blood sugar (RBS)

Statistical analysis:

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0 **(**Armonk, NY: IBM Corp**).** The Kolmogorov- Smirnov, Shapiro and D'agstino tests were used to verify the normality of distribution of variables. Comparisons between groups for categorical variables were assessed using Chi-square test (Fisher or Monte Carlo). Student t-test was used to compare two groups for normally distributed quantitative variables while Mann Whitney test was used to compare between two groups for abnormally distributed quantitative variables. Univariate analysis and multivariate analysis logistic regression were done to identify the variables that can predict mortality. Receiver–operating characteristic (ROC) curves for predicting the probability of mortality were generated from the data. Area under ROC curve, sensitivity, specificity, positive predictive values (PPV) and negative predictive values (NPV) were calculated. The area under ROC curve (AUC) is graded as follows: 0.90-1= excellent; 0.80-0.90= good; 0.70-0.80 = fair and 0.60-0.70 = poor. Significance was adopted at $p < 0.05$ for interpretation of results of tests (Dawson and Trapp, 2001).

Results:

During the study period, 113 cases have fulfilled the inclusion criteria. Their sociodemographic data (age, gender and residence) and toxicological data (mood of poisoning, route of exposure and delay time between exposure and arrival to hospital) are presented in table (1).

Table (1): Distribution of sociodemographic and toxicological data of cases of aluminum phosphide poisoning included in the study (113 cases).

Age (years)	Min- Max.		11- 50
	Median		20
Gender	Female	n	70
		%	61.9
	Male	n	43
		%	38.1
Residence	Urban	n	16
		%	14.2
	Rural	n	97
		%	85.8
Mode of poisoning	Accidental	n	11
		%	9.7
	Suicidal	n	102
		%	90.3
Route of exposure	Ingestion	n	109
		%	96.5
	Inhalation	n	4
		%	3.5
Delay (Hours)	Range		0.5- 14
	Median		2

n: number, Min.- Max.: minimum- maximum.

Table (2) shows that 77 cases died with an overall mortality rate of 68.1% (this represented the non–survivor group). There was no statistically significant difference between survivors and non- survivors as regards age, gender, residence, route of exposure and delay time. On the other hand, a statistically significant difference between the two groups as regards mode of exposure was detected.

Table (2): Comparison between survivor and non-survivor groups following aluminum phosphide poisoning regarding sociodemographic and toxicological data (number = 113).

	Outcome		**Test of Sig.**	**p value**
	Survivors (n = 36)	**Non survivors (n = 77)**		
Age (years)				
Min. –Max.	15 – 50	11 – 50	U= 1238.50	0.362
Median	21	20		
Sex				
Male	13 (36.1%)	30 (39%)	χ^2= 0.085	0.771
Female	23 (63.9%)	47 (61%)		
Residence				
Rural	28 (77.8%)	69 (89.6%)	χ^2= 2.826	0.093
Urban	8 (22.2%)	8 (10.4%)		
Mode of poisoning				
Accidental	9 (25%)	2 (2.6%)	χ^2= 14.011*	0.001*
Suicidal	27 (75%)	75 (97.4%)		
Route of exposure				
Ingestion	33 (91.7%)	76 (98.7%)	χ^2= 3.555	0.095
Inhalation	3 (8.3%)	1 (1.3%)		
Delay time (hours)				
Min. –Max.	0.5 – 14	0.5 – 14	U= 1083.50	0.161
Median	3	2		

χ^2: Chi square test, U: Mann Whitney test, p: p value, *: Statistically significant at $p \leq 0.05$, n: number, Min.- Max.: minimum- maximum.

Table (3) revealed a comparison between survivors and non-survivors considering Glasgow coma scale (GCS), vital signs and the presence of ECG abnormalities. Statistically significant difference could be detected between survivors and non-survivors as regards all these parameters except the heart rate. ECG finding among survivors, non-survivors and the whole study population were recorded in table (4).

Table (3): Comparison between survivor and non-survivor groups following aluminum phosphide poisoning regarding the clinical data and ECG findings (number = 113)

	Total (n = 113)	Outcome		Test of Sig.	p value
		Survivors (n = 36)	Non survivors (n = 77)		
GCS					
Min. –Max.	3 – 15	12 – 15	3 – 15	U=973.0*	0.002*
Median	15	15	15		
Pulse (b/min)					
Min. –Max.	40 – 143	60 – 134	40 – 143	U=1237.5	0.547
Median	98	90	99		
Respiratory rate (cycle/min)					
Min. –Max.	16 – 55	16 – 27	17 – 55	U=859.0*	0.001*
Median	22	21	23.5		
Systolic blood pressure (mmHg)					
Min. –Max.	40 – 160	60 – 160	40 – 130	t=5.439*	<0.001*
Mean ± SD.	89.7 ± 23.7	105.7 ± 25	79.8 ± 16.5		
Diastolic blood pressure (mmHg)					
Min. –Max.	30 – 100	30 – 100	30 – 80	t=5.303*	<0.001*
Mean ± SD.	55.4 ± 16.9	66.6 ± 17.8	48.5 ± 12		
Blood pressure					
Normal (%)	36 (31.9%)	23 (63.9%)	13 (16.9%)	χ^2=24.968*	<0.001*
Abnormal (%)	77 (68.1%)	13 (36.1%)	64 (83.1%)		
ECG					
Normal (%)	20 (17.7%)	12 (33.3%)	8 (10.4%)	χ^2=8.865	0.003*
Abnormal (%)	93 (82.3%)	24 (66.7%)	69 (89.6%)		

χ^2: Chi square test, U: Mann Whitney test, t: student t test, p: p value, *: Statistically significant at $p \leq 0.05$, n: number, Min.- Max.: minimum- maximum.

Table (4): ECG findings among survivors, non-survivors and the whole study population following aluminum phosphide poisoning (number = 113)

ECG finding		Total (n = 113)	Outcome	
			Survivors (n = 36)	Non-survivors (n = 77)
Normal ECG		20 (17.7%)	12 (33.3%)	8 (10.4%)
ECG changes	Sinus tachycardia	45 (39.8%)	16 (44.4%)	29 (37.7%)
	Ventricular fibrillation	4 (3.5%)	0 (0%)	4 (5.2%)
	Atrial fibrillation	13 (11.5%)	3 (8.3%)	10 (13%)
	Sinus bradycardia	13 (11.5%)	2 (5.6%)	11 (14.3%)
	Ventricular tachycardia	4 (3.5%)	0 (0%)	4 (5.2%)
	Extrasystole	6 (5.3%)	1 (2.8%)	5 (6.5%)
	Inverted T-wave	4 (3.5%)	1 (2.8%)	3 (3.9%)
	S-T elevation	4 (3.5%)	1 (2.8%)	3 (3.9%)

ECG: electrocardiogram.

Comparison between survivors and non-survivors considering results of ABG analysis, electrolyte level, liver enzymes, kidney function tests, RBS level and white blood cell count detected a significant statistical difference in pH, HCo_3, K and creatine level and white blood cell count (Table 5).

Table (5): Comparison between survivor and non-survivor groups following aluminum phosphide poisoning regarding results of laboratory investigations (number = 113)

	Total (n = 113)	Outcome		Test of Sig.	p
		Survivors (n = 36)	Non survivors (n = 77)		
pH					
Min.- Max.	7.1 – 7.5	7.2 – 7.5	7.1 – 7.5	t= 9.472*	<0.001*
Mean ± SD.	7.3 ± 0.1	7.4 ± 0.1	7.2 ± 0.1		
HCo_3 (mEq/L)					
Min.- Max.	5.3 – 33	10 – 33	5.3 – 33	U= 449.5*	<0.001*
Median	15.3	19.5	11.8		
ALT (U/L)					
Min.- Max.	2 – 85	5 – 44	2 – 85	U= 1204.0	0.261
Median	20	18.5	21		
AST (U/L)					
Min.- Max.	6 – 90	10 – 40	6 – 90	U= 1244.5	0.383
Median	21	21.5	21		
Urea (mg%)					
Min.- Max.	15 – 53	15 – 53	15 – 48	U= 1209.0	0.275
Median	28	30	27		
Creatine (mg%)					
Min.- Max.	0.5 – 2.4	0.5 – 1.4	0.5 – 2.4	U= 911.0*	0.004*
Median	1	0.8	1		
Na (mg%)					
Min.- Max.	130.6 – 155	132.5 – 152	130.6 – 155	t= 0.493	0.623
Mean ±SD.	140.4 ± 4.8	140.1 ± 4.5	140.6 ± 5		
K (mg%)					
Min.- Max.	2.2 – 5.4	2.6 –5.3	2.2 – 5.4	t= 2.855*	0.005*
Mean± SD.	3.5 ± 0.7	3.8 ± 0.7	3.4 ± 0.6		
Random blood Sugar (mg%)					
Min.- Max.	42 – 413	79 – 410	42 – 413	U= 1151.5	0.148
Median	130	120	137		
WBCs ($\times 10^3$)					
Min.- Max.	2.7 – 18.7	2.7 – 18.7	5 – 16.6	U= 1013.0*	0.021*
Median	11.7	8.7	11.8		

U: Mann Whitney test, t: student t test, p: p value, *: Statistically significant at $p \leq 0.05$, n: number, Min.- Max.: minimum- maximum.

Univariate analysis detected a statistically significant relationship between mortality and all the following; the presence of ECG changes, GCS, blood pressure disorders, respiratory rate, pH, HCo_3, K and creatine level and white blood cell count. While the multivariate analysis logistic regression revealed that the presence of ECG changes and the pH level are the only independent variables that can predict mortality (Table 6).

Table (6): Univariate analysis and multivariate analysis logistic regression differentiation between survivors and non-survivors following aluminum phosphide poisoning (number = 113).

	Univariate		#Multivariate	
	p	**OR (95% C.I)**	**p**	**OR (95% C.I)**
ECG changes	0.004*	4.312 (1.574– 11.817)	0.048*	7.291 (1.017– 52.262)
GCS	0.028*	0.469 (0.238– 0.923)	0.577	1.133 (0.730– 1.760)
Blood pressure disorders	<0.001*	8.710 (3.526– 21.519)	0.126	3.129 (0.725– 13.505)
Respiratory rate	0.002*	1.180 (1.065– 1.307)	0.063	1.249 (0.988– 1.578)
PH	<0.001*	0.183 (0.098– 0.341)	<0.001*	0.141 (0.049– 0.404)
Hco3	<0.001*	0.781 (0.707– 0.863)	0.810	0.979 (0.822– 1.166)
Creatine	0.007*	9.049 (1.827– 44.828)	0.323	4.780 (0.214– 106.535)
K	0.007*	0.426 (0.228– 0.796)	0.147	0.429 (0.137– 1.345)
WBCs ($\times 10^3$)	0.014*	1.168 (1.032– 1.322)	0.598	1.056 (0.863– 1.291)

OR: Odd's ratio, C.I: Confidence interval, #: All variables with p<0.05 were included in the multivariate regression, *: Statistically significant at $p \leq 0.05$.

Table (7) and figure (1) show the results of analysis of ROC curve of blood pH level as a predictor of mortality. Blood pH level has an AUC of 0.881 which is graded as good AUC. The optimal cut-off value of blood pH was identified. At a cut off values of ≤7.28 the blood pH level had a sensitivity of 71.43 and a specificity of 97.22. Moreover, PPV (the probability that a patient at a certain cut off value will die) have been calculated and was 98.2%

Table (7): Receiver operating characteristic (ROC) curve for blood pH level for prediction of mortality following aluminum phosphide poisoning (number = 113).

	AUC	**P**	**95% C.I**	**Cut off**	**Sensitivity**	**Specificity**	**PPV**	**NPV**
pH	0.881	<0.001*	0.817 – 0.945	≤7.28	71.43	97.22	98.2	61.4

AUC: Area under a Curve, p value: Probability value, CI: Confidence Intervals, PPV: Positive predictive value, NPV: Negative predictive value, *: Statistically significant at $p \leq 0.05$.

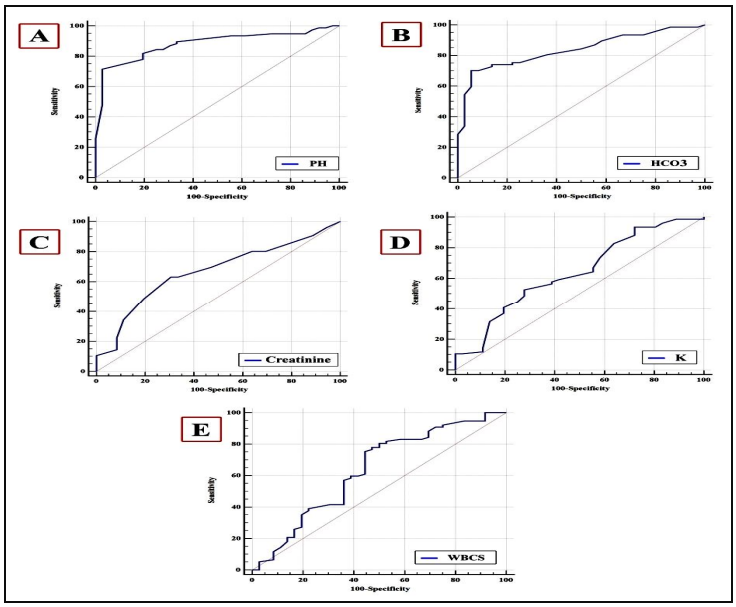

Fig. (1): Receiver operating characteristic (ROC) curve for blood pH level for prediction of mortality following aluminum phosphide poisoning.

Discussion

Aluminum phosphide is a potent pesticide. It is used for crops protection during storage and transportation (Bumbrah et al., 2012). Acute AlP toxicity is associated with high risk of morbidity and mortality (Singh et al., 2014). Therefore, the current study was designed to evaluate role of clinical data, laboratory investigations and ECG changes in mortality prediction in acute AlP poisoning.

Sociodemographic characteristics, and toxicological data in this study were comparable to results gathered from different poison control centers in Egypt and across the developing world (El Naggar and El Mahdy, 2011; Hosseinian et al., 2011; Vijayanath et al., 2011; Soltaninejad et al., 2012).

The mortality rate in this study was about 68 %. This rate is near to rates reported by Masoud and Barghash (2013) and El-Ebiary et al. (2015) from Egypt (64% and 67.5% respectively). This high mortality rate might be due to the easy availability and cheap price of AlP and the absence of specific antidote (Sulaj et al., 2015).

Glasgow coma scale registered significant difference between survivors and non-survivors in the current study. In the same line of this result, Louriz et al. (2009) and El-Ebiary et al. (2015) reported significantly lower values of GCS in non survivors when compared to survivors. Disturbed consciousness level induced by AlP poisoning may be due to cerebral anoxia resulting from refractory shock (Mehrpour et al., 2012). Such phosphides induced neural injury was previously mentioned by Guale et al. (1994). They described central nervous system intoxication as clinical sign in phosphides poisoning. In post-mortem reports; Tripathi and Pandey (2007) reported distinct changes in the cerebral and cerebellar cortex due to the effect of phosphides on human brain.

Systolic and diastolic blood pressure recorded significant lower mean values for non-survivors when compared to survivors. Furthermore, a significantly higher percentage of non- survivors showed abnormal blood pressure. These results were similar to results

reported by El-Ebiary et al. (2015) and El-Sarnagawy (2017). Aluminum phosphide induced hypotension is due to direct myocardial depressive effect, adrenal insufficiency with inadequate systemic vasoconstriction, massive intravascular fluid loss as a result of increased permeability of capillaries and decreasing left ventricular ejection fraction (Marashi et al., 2011; Farnaghi et al., 2013). Moreover, hypovolemia due to vomiting could contribute to hypotension (Louriz et al., 2009).

The present study showed that respiratory rate was significantly higher in non-survivors compared to survivors. Comparable results were obtained by El-Ebiary et al. (2015). The increase in respiratory rate could be a compensatory response to metabolic acidosis which is the most common type of acid base disturbance in AlP poisoned patients (Jaiswal et al., 2009).

The ECG changes were significantly registered in non- survivors when compared to survivors. Parallel results were obtained by Shadnia et al. (2009) and El-Sarnagawy (2017). Sinus tachycardia was the most common change among survivors and non-survivors while all other changes were more common among non-survivors than survivors. Similar results were reported by El-Sarnagawy (2017). The ECG abnormalities induced by AlP poisoning could be attributed to cardio toxic effect of phosphine gas. It promotes generation of reactive oxygen species leading to lipid peroxidation with focal areas of necrosis in the myocardium (Akkaoui et al., 2007). Furthermore, it leads to inhibition of mitochondrial cytochrome C oxidase with subsequent depletion of myocardial energy like ischemia (Singh et al., 2006). These effects leads to changes in cardiac transmembrane action potentials causing dysrhythmia, and ischemia-like effect on ECG (El-Ebiary et al., 2015).

The mean blood pH and HCo_3 levels were significantly lower in non-survivors compared to survivors. Similar results were obtained by Mathai and Bhanu (2010) and Navabi et al. (2018). Accumulation of lactic acid resulting from AlP induced blockage of oxidative phosphorylation and poor tissue perfusion may be the cause of metabolic acidosis which is considered the most common acid-base abnormalities in acute AlP toxicity. (Agarwal et al., 2014; Berry et al., 2015).

The mean K level was significantly lower in non-survivors compared to survivors. El-Sarnagawy (2017) reported alike results. Farzaneh et al. (2018) reported insignificant lowering in K level in non-survivors. Hypokalemia after metal phosphides toxicity may be due to vomiting. In addition, catecholamine release could be a contributing factor (Proudfoot, 2009).

The median level of creatinine and WBCs count were significantly higher in non-survivors compared to survivors. Masoud and Barghash (2013) reported similar results. These effects could be explained by action of phosphine gas which inhibits the cytochrome C oxidase activity and causes free radical damage of tissues. Organs that have high oxygen demands (the brain, heart, kidneys and liver) appear to be very sensitive to this damage (Louriz et al., 2009).

The present study reported a significant relationship between mortality and ECG changes, GCS, blood pressure, respiratory rate, pH, HCo_3, K, creatine level and white blood cell count based on univariate regression analysis. Navabi et al. (2018) reported a significant relationship between mortality and delay time, blood pressure, number of ingested tablets, vomiting, pH and HCO3 level.

In the current study, multivariate analysis revealed that, ECG changes and pH level are the only independent variables that can predict

mortality. On the other hand, Navabi et al. (2018) reported blood pressure, pH level and delay time as the most important predictive variables of mortality.

Analysis of ROC curve for blood pH level detected a sensitivity of 71.43 (able to predict, 71.43% of cases that died) and a specificity of 97.22 with an optimal cut-off value of ≤7.28. This result was slightly different from result of (Masoud and Barghash (2013)) who reported a ROC curve analysis with a sensitivity and specificity of 100% and a best cut of value of ▪ 7.27.

Conclusion:

The present study concluded that AlP poisoning cause high rate of mortality. The presence of ECG changes and the blood pH level are the only variables that could predict mortality based on the multivariate regression analysis. Analysis of ROC curve for pH level as a predictor of mortality revealed that at a cut off values of ≤7.28 the blood pH level was able to predict 71.43% of cases that died.

Recommendations:

Assessment of ECG and blood pH level is highly recommended in cases of AlP poisoning for early prediction of mortality in these cases. Further studies on a larger scale of cases are recommended to detect other predictors of mortality.

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دور البيانات السريرية والفحوصات المختبرية وتغيرات رسم القلب في التنبؤ بالوفيات في حالات التسمم الحاد بفوسفيد الألومنيوم

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فوسفيد الالومنيوم واحد من المبيدات الحشرية التي تستخدم على نطاق واسع في التبخير في مصر. تتوفر بيانات محدودة عن المنبئات المتعلقة بالوفيات في المرضي المسممين بفوسفيد الالومنيوم. لذلك، كان الهدف من هذا العمل هو تقييم دور البيانات السريرية والفحوصات المختبرية ونتائج رسم القلب في التنبؤ بالوفيات في هذه الحالات. أجريت الدراسة بأثر رجعي على ١١٣ حاله من حالات التسمم بفوسفيد الالومنيوم التي احتجزت في مركز مكافحة السموم بجامعة طنطا في الفترة من بداية أكتوبر ٢٠١٦ لنهاية سبتمبر ٢٠١٨. وتم تجميع البيانات الخاصة بالتاريخ المرضي والفحوصات السريرية ونتائج الفحوصات المختبرية. نسبة الوفيات كانت ٦٨,١%. كان هناك فارق ذا دلالة احصائية بين الناجين وغير الناجين فيما يتعلق بطريقه التسمم، مقياس جلاسكو للغيبوبة، العلامات الحيوية (باستثناء معدل ضربات القلب)، وجود تغيرات في رسم القلب، مستوى حامضية الدم، مستوى بيكربونات الصوديوم، مستوى البوتاسيوم، مستوى الكرياتين وعدد كرات الدم البيضاء. وكشف الانحدار اللوجستي لتحليل متعدد المتغيرات ان تغييرات رسم القلب ومستوى حامضية الدم فقط هي المتغيرات المستقلة التي يمكنها التنبؤ بالوفيات. وقد وجد تحليل منحني (ROC) لمستوى حامضية الدم منطقة جيدة تحت المنحنى. فعند قيمة قاطعة $\geq$ ٧,٢٨ يكون لمستوى حامضية الدم حساسية بنسبة ٧١,٤٣. وقد استنتج ان حالات التسمم بفوسفيد الالومنيوم تتسبب في معدل وفيات مرتفع. ويمكن التنبؤ بالوفيات من خلال تغييرات رسم القلب ومستوى حامضية الدم. وعند قيمة قاطعة $\geq$ ٧,٢٨ يستطيع مستوى حامضية الدم التنبؤ بالوفيات. لذا ينصح بتقييم رسم القلب ومستولى حامضية الدم في حالات التسمم بفوسفيد الالومنيوم للتنبؤ المبكر بالوفيات.