

Prognostic Value of Blood Lactate and C-Reactive Protein Levels in Acute Phosphides Poisoned Patients Admitted to Menoufia University Poison Control Center

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ABSTRACT

KEYWORDS

Aluminum phosphide,
Blood lactate,
CRP,
Poisoning,
Prognostic value.

Metal phosphides as a whole and aluminum phosphide (AIP) in particular, are potent pesticides. Although phosphides are highly toxic and had a high mortality, it cannot be discarded due to their wide-spectrum applications. The aim of the study was to evaluate the prognostic value of blood lactate and C-reactive protein (CRP) levels in acute phosphide poisoned patients admitted to Menoufia University Poison Control Center. This was an analytical cross-sectional study done on cases of acute phosphide poisoning admitted to Menoufia Poison Control Center (MPCC) from the 1st of August 2022 to 31 December 2022 which fulfilled the inclusion criteria. Patient's data were collected in clinical sheets after taking written consent from patients or their guardians. Only moderately and severely poisoned patients were included in the study according to Poison Severity Score. History, clinical examination, and Echocardiography were performed. Blood samples were taken for routine laboratory investigations and measuring blood lactate and CRP levels. A total of 57 patients (26 males and 31 females) were included in the present study. In all cases, the manner of poisoning was suicide. The age in non survivors (85.9%) ranged between 14-63 years while in survivors (14.1%) was 17-39 years. Increase tablet number, low pH, low PO₂, high respiratory rate, low temperature, low diastolic blood pressure, high pulse rate, low ejection fraction, low potassium level, high CRP and blood lactate levels were significant predictors for phosphide poisoning mortality. Aluminum phosphide is highly toxic with a high mortality rate. CRP and blood lactate levels can be used as predictors for mortality.

Introduction

Metal phosphides as a whole and aluminum phosphide (AIP) in particular, are potent pesticides. They have an important role in protection of crops during transportation and storage. Although these substances are highly toxic with a high mortality, it cannot be written off due to their wide-spectrum applications (Bumbrah et al., 2012).

Aluminum phosphide is purchasable in a form of 3-grams tablets, (56% aluminum

phosphide and 44% ammonium carbonate in each tablet). The lethal dose is 150–500 mg, for a subject weighing 70 Kg (Mehrpour et al., 2019).

Metal phosphides cause many harmful effects on human body, in the form of oxidative stress, electrolyte disturbances, metabolic disorders, circulatory failure, and damage to most organs (Sciuto, et al., 2016). Cardiac shock, hypotension and severe metabolic acidosis are considered as the most important causes of death in these cases (Mehrpour, et al., 2019).

The increased fatality of ALP is due to effects of phosphine gas (PH₃) that is released after exposure of the tablet to water and/or acid. This gas will affect mitochondrial

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Cytochrome c oxidase activity and causes its dysfunction leading to reduction in production of adenosine triphosphate (ATP) molecules and causing an increase in hemolysis of RBCs. increased oxidative stress is the most proposed mechanisms (Hosseini et al., 2020).

ALP-poisoning is considered as an important cause of fatal poisoning worldwide. Most patients deteriorate despite proper supportive care, as there is no known specific antidote (Oghabian and Mehrpour, 2016).

All inflammatory processes affecting human body will lead to a significant increase in pro-inflammatory proteins levels in serum i.e. acute phase proteins. Among these proteins, C-reactive protein (CRP) is one of the most important prognostic biomarkers. The results concluded that CRP levels can be considered as a cheap prognostic test for evaluation of severity of acute poisoning (Sawiniec et al., 2004).

Blood lactate level was used as a prognostic tool by Broder and Weil in 1964 when they noted that a blood lactate level more than 4 mmol/L was found to be associated with bad prognosis in patients suffering from shock (Broder and Weil, 1964). In critically ill patients, blood lactate level has been considered as a prognostic factor. It can be elevated due to many causes but shock and tissue hypoperfusion may be the most common cause of elevation (Anderson et al., 2013). Blood lactate level has also been used as a prognostic factor in patients with some poisonings (Inoue et al., 2011; Lee, et al., 2012).

This study aimed to evaluate the prognostic value of blood lactate and C-reactive protein levels in acute phosphide poisoned patients that were admitted to Menoufia University Poison control Center.

Patients and methods

This was an analytical cross section study conducted on cases of acute phosphide poisoning admitted to Menoufia Poison Control Center (MPCC) in the period from 1st of August 2022 to 31 December 2022. The diagnosis was obtained on positive history of exposure, presence of the container, clinical picture suggestive of poisoning with shock and metabolic acidosis or positive bed side test for the detection of phosphine gas in stomach contents (silver nitrate test).

Inclusion criteria were moderately and severely poisoned patients according to Poison Severity Score (Persson et al., 1998).

Exclusion criteria were patients with co-ingestion, liver, kidney or heart disease, diabetes mellitus, patients who had received medical management prior to admission or those who had asymptomatic or mild criteria according to Poison Severity Score.

Approvals from the head of MPCC and Ethical Committee at Menoufia University Hospital were obtained (Approval Number 8/2022FORE1). Special clinical sheets were developed for collection of patient's data after taking written consent from the patients or their guardians.

To ensure confidentiality, patient's data were kept anonymous.

Collected data included socio-demographic data: (age, sex, marital status, educational level and residence), poisoning data (manner and cause of poisoning, amount of phosphide poison, delay time before presentation). Clinical examination was done including vital signs, level of consciousness (according to Glasgow Coma Scale) and systemic examination. Echocardiography was done for evaluation of left ventricular function (Yancy et al., 2013). Blood samples were taken for routine laboratory investigations and blood lactate and C-reactive protein levels at

the Clinical Biochemistry Laboratory department of Menoufia University hospital. The concentrations of CRP were measured using the CRP a latex enhanced turbidimetric immunoassay kits (MISPA- i3 AGAPPE DIAGNOSTIC LTD. 'Agappe Hills', Dist. Ernakulam, Kerala, India-683 562). Lactate concentration is determined using an enzymatic colorimetric method by spectrophotometer (Kit was purchased from Egyptian Company for Biotechnology, Obour City, Cairo, Egypt).

Statistical analysis

With the aid of the IBM SPSS software program version 20.0 (Armonk, NY: IBM Corp) grouped statistics were obtained as percentages and numbers. To evaluate two groups, the Chi-square test was used. In the occasion that more than 20% of the cells have an anticipated count lower than, the Fisher Exact or Monte Carlo correction test was instead used. Range (minimum and maximum), mean, standard deviation (SD), and median were used to convey quantitative data. Student t-test was used to assess two groups for quantitative variables that were normally distributed. The Mann Whitney test, on the other side, was used to compare two groups for quantitative variables that were not normally distributed. At the 5% level, significance of the findings was determined.

Results

A total of 57 patients (26 males and 31 females) were included in the present study. All were AIP cases as zinc phosphide poisoned cases were asymptomatic or mild according to poison severity score and so they were excluded. In all cases, the manner of poisoning was suicide and through oral route. Patients were categorized into two groups regarding outcome into survivors (14.1%) and non survivors (85.9%). There were no statistically significant differences between both groups regarding age, sex, marital status, educational level, residence, the cause of suicide and delay hours. The age of non survivors ranged between 14-63 years while in survivors ranged between 17-39 years. In total cases, number of females was more slightly increased than males, cases were mainly single and most commonly of basic education (47.4%), mainly from rural areas (71.9%), social problems were the main trigger of suicide (66.7%) and delay time before arrival to hospital was more increased in non survivors than in survivors.

On the other hand, there were statistically significant differences between both groups regarding hospital stay duration, tablet number and severity of poisoning. While hospital stay duration was significantly higher in survivors. Tablet number of aluminum phosphide was higher in non-survived cases and most of non-survived cases at presentation were severe according to poison severity score (Table 1 and Figure1).

Table (1): Comparison between survivors and non-survivors of acute aluminum phosphide poisoned cases according to sociodemographic data and toxicological data (n= 57).

	Total (n = 57)	Survivor (n = 8) 14.1%	Non survivor (n = 49) 85.9%	Test of sig.	P
Age (years)					
Median (Min. – Max.)	22 (14 – 63)	21 (17 – 39)	22 (14 – 63)	U= 196.0	1.000
Sex					
Male	26 (45.6%)	2 (25%)	24 (49%)	$\chi^2=$	^{FE} p=
Female	31 (54.4%)	6 (75%)	25 (51%)	1.594	0.269
Marital status					
Single	33 (57.9%)	5 (62.5%)	28 (57.1%)	$\chi^2=$	^{MC} p=
Married	20 (35.1%)	3 (37.5%)	17 (34.7%)	0.337	1.000
Unmarried	4 (7%)	0 (0)	4 (8.2%)		
Education					
Illiterate	7 (12.3%)	1 (12.5%)	6 (12.2%)		
Basic	27 (47.4%)	3 (37.5%)	24 (49%)	$\chi^2=$	^{MC} p=
Secondary	17 (29.8%)	3 (37.5%)	14 (28.6%)	0.977	0.941
High	6 (10.5%)	1 (12.5%)	5 (10.2%)		
Cause of suicide					
Family problems	38 (66.7%)	6 (75%)	32 (65.3%)	$\chi^2=$	^{MC} p=
Psychiatric history	11 (19.3%)	0 (0%)	11 (22.4%)	2.607	0.214
undetermined	8 (14%)	2 (25%)	6 (12.2%)		
Delay hours					
Median (Min. – Max.)	2.5 (1 – 5)	2 (1 – 5)	3 (1 – 5)	U= 149.0	0.291
Residence					
Urban	16 (28.1%)	2 (25%)	14 (28.6%)	$\chi^2=$	^{MC} p=
Rural	41 (71.9)	6 (75%)	35 (71.4%)	.043 ^a	1.000
Tablet number					
Median (Min. – Max.)	1(0.25 – 3)	0.50(0.25 – 1)	1(0.25 – 3)	U= 66.50*	0.002*
Severity of poisoning					
Moderate	13 (22.8%)	8 (100.0%)	5 (10.2%)	$\chi^2=$	^{FE} p
Severe	44 (77.2%)	0 (0%)	44 (89.8%)	31.498	<0.001*

n: number, Min. – Max: Minimum- Maximum, SD: Standard deviation, t: Student t-test, U: Mann Whitney test, χ^2 : Chi square test, MC: Monte Carlo, FE: Fisher Exact p: p value for comparing between the studied groups, *: Statistically significant at $p \leq 0.05$.

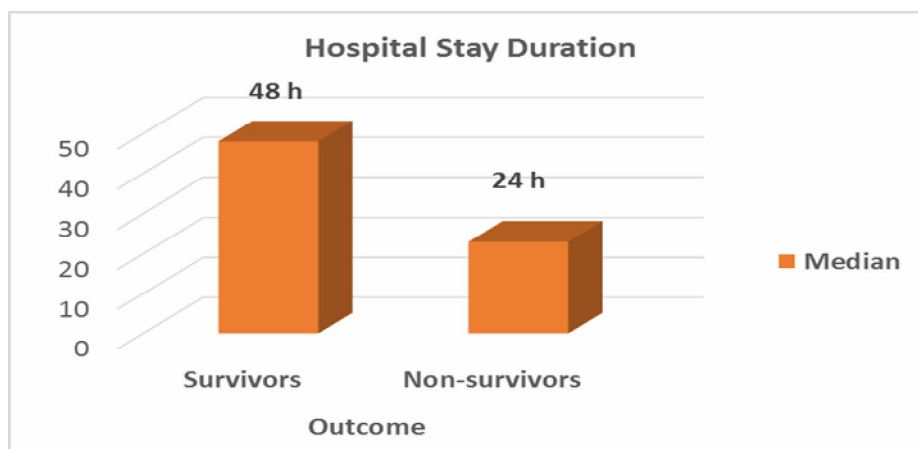


Fig. (1): Comparison between survivors and non- survivors of acute aluminum phosphide poisoned cases as regards hospital stay duration (U=6.000* and P <0.001).

Comparison of the clinical parameters between two groups has been illustrated in (Table2). It showed that there were statistically significant differences between both groups regarding temperature, respiratory rate, systolic blood pressure,

diastolic blood pressure, ventricular Ejection fraction (EF%), Glasgow coma scale (GCS) and pulse rate, where the non survivors had higher respiratory rate, pulse rate and lower temperature, systolic and diastolic blood pressure, GCS and ejection fraction.

Table (2): Comparison between survivors and non- survivors of acute aluminum phosphide poisoned cases as regards clinical presentation and echocardiography (n=57)

	Total (n = 57)	Survivor (n = 8)	Non survivor (n = 49)	Test of sig.	P
Temperature					
Mean ± SD.	37.1 ± 0.1	37.2 ± 0.1	37.1 ± 0.1	t= 2.904*	0.005*
Respiratory rate					
Median (Min. – Max.)	28 (18 – 35)	20 (18 – 28)	28 (18 – 35)	U= 46.50	<0.001*
Systolic blood pressure					
Median (Min. – Max.)	80 (50 – 110)	105 (100 – 110)	70 (50 – 100)	U= 12.0	<0.001*
Diastolic blood pressure					
Median (Min. – Max.)	50 (30 – 80)	70 (60 – 80)	40 (30 – 70)	U= 26.50	<0.001*
Ventricular Ejection fraction (EF%) measured by Echocardiography					
Mean ± SD.	44 ± 9.7	55.6 ± 3.2	42.1 ± 9.1	t= 7.819	<0.001*
GCS					
Median (Min. – Max.)	15 (3 – 15)	15 (15 – 15)	15 (3 – 15)	U= 108.0*	0.043*
Pulse					
Mean ± SD.	101.3 ± 13.3	87.6 ± 4.4	103.6 ± 12.9	t= 6.610*	<0.001*

n: number, Min. – Max: Minimum- Maximum, SD: Standard deviation, GCS: Glasgow coma scale, t: Student t-test, U: Mann Whitney test, p: p value for comparing between the studied groups, *: Statistically significant at $p \leq 0.05$.

Regarding laboratory investigations, there were statistically significant differences between both groups regarding pH, PO₂, HCO₃, potassium, blood glucose, lactate and CRP levels. The non-survivors experienced a significant lower pH, PO₂, potassium level and higher glucose level, CRP and blood lactate level compared to survivors.

On the contrary, there were no statistically significant differences between both groups regarding prothrombin time, INR, liver enzymes (ALT and AST), sodium, magnesium, creatinine, cholinesterase level and hemoglobin level (Table 3).

Table (3): Comparison between survivors and non- survivors of acute aluminum phosphide poisoned cases as regards laboratory investigations (n=57).

	Total (n = 57)	Survivor (n = 8)	Non survivor (n = 49)	Test of sig.	P
pH					
Mean ± SD.	7.22 ± 0.08	7.31 ± 0.02	7.21 ± 0.07	t= 8.801*	<0.001*
PCO₂					
Median (Min. – Max.)	39 (20 – 49)	42.5 (38 – 47)	39 (20 – 49)	U= 122.50	0.092
PO₂					
Mean ± SD.	74.5 ± 10.9	92.3 ± 3.5	71.6 ± 8.7	t= 11.772*	<0.001*
HCO₃					
Median (Min. – Max.)	15 (8.7 – 23)	21 (19 – 23)	14 (8.7 – 19)	U= 7.0*	<0.001*
Prothrombin Time (PT)					
Median (Min. – Max.)	14 (13.2 – 31.8)	13.6 (13.2 – 16.1)	14.9 (13.2 – 31.8)	U= 135.0	0.168
International Normalized Ratio (INR)					
Median (Min. – Max.)	1.1 (1 – 2.5)	1 (1 – 1.2)	1.1 (1 – 2.5)	U= 145.0	0.251
ALT					
Median (Min. – Max.)	15 (3 – 35)	14.5 (3 – 22)	15 (3 – 35)	U= 162.50	0.449
AST					
Mean ± SD.	27 ± 9.5	26.5 ± 10.8	27.1 ± 9.4	t= 0.153	0.879
Sodium					
Mean ± SD	138.1 ± 3	138.5 ± 3.3	138 ± 3	t= 0.427	0.671
Potassium					
Median (Min. – Max.)	3.6 (2.5 – 5.5)	4.3 (2.8 – 5.2)	3.5 (2.5 – 5.5)	U= 110.50*	0.048*
Magnesium					
Median (Min. – Max.)	2.2 (1.5 – 2.9)	2.3 (2.1 – 2.6)	2.2 (1.5 – 2.9)	U= 180.0	0.727
Creatinine					
Median (Min. – Max.)	0.7 (0.1 – 1.3)	0.7 (0.1 – 1)	0.7 (0.1 – 1.3)	U= 191.50	0.919
Cholinesterase level					
Mean ± SD.	629.9 ± 226.5	663.5 ± 278.6	624.4 ± 219.8	t= 0.450	0.655
Glucose					
Median (Min. – Max.)	165 (90 – 350)	128.5 (90 – 210)	173 (100 – 350)	U= 84.50	0.008*
Hemoglobin					
Mean ± SD.	13 ± 1	13.1 ± 1.1	13 ± 1	t= 0.317	0.752
CRP					
Mean ± SD.	11.5 ± 4.4	6.6 ± 1.5	12.3 ± 4.2	t= 7.017*	<0.001*
Lactate					
Median (Min. – Max.)	55(23 – 97)	37.5(23 – 48)	56(25 – 97)	U= 19.0*	<0.001*

n: number, Min. – Max: Minimum- Maximum, SD: Standard deviation, ALT: alanine transaminase, AST: aspartate aminotransferase, CRP: C- reactive protein, t: Student t-test, U: Mann Whitney test, p: p value for comparing between the studied groups, *: Statistically significant at $p \leq 0.05$

A significant negative correlation was shown between CRP on one side and hospital stay (p value 0.039) and ejection fraction (p value 0.051) on the other side. Also, there was

a significant negative correlation between lactate level on one side and hospital stay (p value 0.002) and pH (p value 0.031) on the other side (Table 4).

Table (4): Correlation between hospital stay, blood PH, HCO₃ and Ejection fraction on one side and blood Lactate and CRP levels on the other side in acute aluminum phosphide poisoned cases (n=57).

	CRP		Lactate	
	r	p	r _s	p
Hospital stay	-0.274	0.039*	-0.401	0.002*
pH	-0.087	0.519	-0.285	0.031*
HCO ₃	0.010	0.942	-0.245	0.066
Ejection fraction	-0.260	0.051	-0.225	0.093

r: Pearson coefficient, r_s: Spearman coefficient, *: Statistically significant at p ≤ 0.05

The logistic regression analysis for the parameters affecting mortality was discussed in table (5). It represented that high glucose level, decreased hospital stay period, increased tablet number, low pH, low oxygen pressure, low temperature, high respiratory

rate, low diastolic blood pressure, low ejection fraction, high pulse rate, low potassium level, high CRP and blood lactate levels were significant predictors for phosphide poisoning mortality.

Table (5): Logistic regression analysis for the predictors of mortality in acute aluminum phosphide poisoning (n=57).

Parameters	p	Univariate
		OR(LL – UL 95% C.I)
Age (years)	0.484	1.027(0.954 – 1.105)
Sex	0.221	2.880(0.529 – 15.694)
Marital status(single)	0.878	1.129(0.240 – 5.308)
Glucose	0.021*	1.034(1.005 – 1.063)
Education (illiterate)	0.984	1.024(0.107 – 9.838)
Hospital stay	0.026*	0.660(0.458 – 0.952)
Delay hours	0.314	1.362(0.746 – 2.488)
Tablet number	0.008*	47.112(2.700 – 821.969)
pH	0.027*	0.0(0.0 – 0.0)
PCO ₂	0.107	0.875(0.743 – 1.029)
PO ₂	0.028*	0.591(0.370 – 0.944)
HCO ₃	0.989	–
Temperature	0.013*	0.0(0.0 – 0.128)
Respiratory rate	0.003*	1.519(1.151 – 2.006)
Systolic	0.994	–
Diastolic	0.012*	0.775(0.635 – 0.946)
Ejection fraction	0.005*	0.757(0.623 – 0.919)
GCS	0.996	–
Pulse	0.009*	1.161(1.038 – 1.299)
ALT	0.336	1.054(0.947 – 1.172)
AST	0.876	1.006(0.929 – 1.090)
Potassium	0.018*	0.256(0.083 – 0.791)
Sodium	0.665	0.947(0.742 – 1.209)
Mg	0.898	1.194(0.079 – 17.994)
Creatinine	0.595	2.390(0.096 – 59.344)
CRP	0.006*	1.703(1.162 – 2.497)
Lactate	0.002*	1.190(1.068 – 1.325)

OR: Odd's ratio, C.I: Confidence interval, LL: Lower limit, UL: Upper Limit, #: All variables with p<0.05 was included in the multivariate, *: Statistically significant at p ≤ 0.05.

For predicting the mortality using an equation, two equations were constructed:

First using CRP, glucose, temperature and Pulse

$$\text{Logit (Mortality)} = 0.517 * (\text{CRP on admission}) + 0.021 * (\text{Glucose}) - 3.833 * (\text{Temperature}) + 0.285 * (\text{Pulse}) + 109.655$$

$$\text{Omnibus Test (31.395, } p < 0.001)$$

$$\text{Nagelkerke R Square} = 0.762$$

Second using Lactate, glucose, temperature, respiratory rate and EF

$$\text{Logit (Mortality)} = 0.158 * (\text{Lactate on admission}) + 0.017 * (\text{Glucose}) - 6.346 * (\text{Temperature}) + 0.140 * (\text{RR}) - 0.256 * (\text{EF}) + 236.638$$

$$\text{Omnibus Test (32.606, } p < 0.001)$$

$$\text{Nagelkerke R Square} = 0.784$$

Discussion

Phosphide intoxication is a widespread problem in developing countries like Egypt because this pesticide is easily accessible and of low price (Elshama 2022).

In the current study, median age of patients was 22 (Range from 14 – 63) years. As, this group is characterized by high activity, common economic problems, and a great deal of social and work difficulties. These findings are similar to those in studies carried out by Farzaneh et al. (2018) in Iran and Abdelkader et al. (2023) in Egypt.

All cases included in this study were suicidal. Many studies found that the suicide mode of poisoning was the predominant as Badawi et al. (2018); Ghonem et al. (2020); Ahmed et al. (2021), and Sakr et al. (2023). This can be explained by a variety of factors, including availability, low price, and higher lethality of this poison (Hegazy et al., 2019).

Regarding sex, females (54.4%) were more than males (45.6%). As females usually experience more difficult circumstances, social and cultural responsibilities, emotional troubles as love failure and marital conflict (Deraz et al., 2022). Most studies agreed with that result as El-Ebiary et al. (2015) (72.5%); Ghonem et al. (2020) (57.3%) and Deraz et al. (2022) (62.5%). On the contrary, Abdelkader

et al. (2023) stated that males outnumbered females (58.5% were males), and he explained that by financial instability and career insecurity facing males.

Considering residence, most cases were from rural regions according to the conducted study. Rural instances made up the bulk according to findings by Hegazy et al. (2019). It could be as a result of phosphide widespread use, unrestricted access, and affordable price (Elshama 2022).

The mortality rate in the current research was high (85.9%). As moderate and severe cases were only included with exclusion of mild cases. Besides, inappropriate first aid administered at home or in primary health care facilities, lack of a specific antidote for phosphide intoxication and treatment relies mainly on supportive treatment along with some medications from clinical trials, such as antioxidants (Elshama 2022). Higher mortality rate was noted by other researchers, as El-Ebiary et al. (2015) (67.5%) and Elhosary and Hodeiba (2020) (73.9%). Lower mortality rates were detected by Abdelkader et al. (2023) (39.1%).

In the current study, statistical difference was found between survivors and non-survivors regarding duration of hospital stay. The duration of hospital stay was significantly higher in survivors. As it is a poison with rapid fatality and patients usually

die within a short period, so, if patients pass the first day, there is a good chance to survive (Tawfik 2018). Similar results were found by El-Sarnagawy (2017) and Elhosary and Hodeiba (2020).

There is a highly statistically significant difference between both groups (survivors and non-survivors) in the present study regarding, systolic and diastolic blood pressure. Both are significantly lower in non-survivors. Also, non survivors had a statistically significant higher pulse rate. Similar results were found by El-Sarnagawy (2017); Tawfik (2018) and Ghonem (2020). Massive intravascular fluid loss carried on by vascular wall insufficiency following phosphine gas absorption, which results in hypovolemic shock could be the cause. Additionally, profound circulatory collapse resulted from the cardio-toxic effects of phosphine gas (Farahani et al., 2016). Pulse rate was observed to be a significant factor that could affect the outcome, which was in line with El-Sarnagawy (2017).

Non survivors had a significant higher respiratory rate than survivors. Ghonem et al. (2020) and Ahmed et al. (2021) had similar results and explained that tachypnea may be a consequence of aluminum phosphide poisoning effect on the respiratory system. Abd-Allah et al. (2022) concluded that phosphine gas causing damaging effect on the alveolar membrane.

Ventricular ejection fraction was considerably lower in non-survivors than in survivors, according to echocardiography. The toxic impact of AIP on the myocardium may be responsible for this outcome. The primary target of AIP poisoning is the heart. Within 12 to 24 hours of exposure to AIP, cardiovascular issues like persistent hypotension, dysrhythmia, and congestive heart failure manifest (Abd-Allah et al., 2022). This result was in agreement with Abdelkader et al. (2023).

Temperature was significantly lower in non-survivors. The same result was noted by Ghonem et al. (2020) who suggested that this might be related to vomiting and a condition of shock.

Concerning level of consciousness, Glasgow coma scale (GCS) was higher in survived than in non-survived. This could be explained by presence of peripheral vasodilation, severe vomiting with fluid loss, hypoxia, and persistent shock which were present in these cases (Abd-Allah et al. 2022). Also, Ghonem et al. (2020) declared that the conscious level depend on degree of hypoxia and hypotension. While this finding disagreed by Erfantalab et al. (2017) who noted that the patient may remain conscious till the end.

Regarding laboratory investigations, ABG findings demonstrated significant differences in pH, PO₂, and HCO₃ findings between survivors and non-survivors. pH, PO₂, and HCO₃ were considerably lower in non-survivors. Severe metabolic acidosis, cytochrome c oxidase inhibition and wide tissue hypo-perfusion may be the main contributing factors (Katwal et al. 2021). These results were agreed with results of studies done by Ghonem et al. (2020); Elgazzar et al. (2022) and Abdelkader et al. (2023).

Regarding blood electrolytes, there was a significant lower potassium level in non survivors. Hypokalemia might be as result of severe vomiting in most of non-survivors (Hegazy et al., 2019). The Same results were found by El-Ebiary et al. (2015) and Elhosary and Hodeiba (2020).

It was found that serum glucose was significantly higher in non survivors. As AIP poisoning may cause pancreatic β -cell dysfunction, glucose tolerance impairment, and mitochondrial dysfunction, which all end up with hyperglycemic state (El-Ebiary, et al.,

2015; Ghonem, et al., 2020, and Ahmed, et al., 2021).

In comparison to survivors, serum lactate was considerably higher in non-survivors. As AIP engage with the mitochondrial electron transport chain and inhibit cytochrome c, resulting in energy deficiency, oxidative stress which contributes to hyperlactatemia and metabolic acidosis (Anand et al., 2013).

Similar results were found by Erfantalab et al. (2017) and Elhosary and Mahmoud (2020) who declared that blood lactate level could be effectively used as a prognostic factor in acute AIP poisoning.

Meanwhile, little is known about C-reactive protein (CRP) role in poisoning (Kim 2022). The exact role of CRP in phosphide intoxication hasn't been discussed in the previous researches. The current study illustrated its role and its effect in the outcome and mortality prediction in phosphide poisoning, as C-reactive protein was significantly higher in non-survivors. Furthermore, there was significant negative correlation between CRP on one side and hospital stay and ejection fraction on the other side, and high CRP was significant predictors for phosphide poisoning mortality.

C-reactive protein is a reactive substance in acute lesions, and it is increased in inflammation and trauma. Toxins may cause inflammation in tissues and organs in the body, which will increase plasma CRP levels. So, its level may give idea about the degree of inflammation caused by phosphide. In the same direction, Wu et al. 2016 found that patients with severe organophosphorus poisoning had severe inflammation in tissues causing multiple organ impairment causing high plasma CRP levels. Sawiniec et al. (2004) concluded that C-reactive protein a useful prognostic marker in acute poisoning.

The present study showed that high glucose level, increase tablet number, low pH, low oxygen pressure, high respiratory rate, low temperature, low diastolic blood pressure, high pulse rate, low potassium level, high CRP and blood lactate levels were significant predictors for phosphide poisoning mortality.

El-Ebiary et al. (2015) noted that alarming risk factors included hypotension, hyperglycemia, low serum bicarbonate, Suicidal ingestion, altered consciousness, and hypokalemia. Also, Farzaneh, et al. (2018) concluded that low systolic blood pressure, low bicarbonate level and low GCS were demonstrated for predicting the mortality rate of AIP-poisoned patients.

Early detection of these potential hazards could assist in effective early action, reduce mortality, and improve outcomes.

Conclusion

Aluminum phosphide is widely used especially in developing countries. Supportive treatments are still prioritized because there is currently no known antidote. Preventing poisoning is inexpensive and fundamental solution. According to the study's results, in addition to other well-known clinical and laboratory findings, blood lactate levels and C-reactive protein can be used to predict mortality from acute aluminum phosphide poisoning.

Recommendations:

- Governments should spread public awareness about dangers of aluminum phosphide
- Strict guidelines should be established to control AIP purchases and look for other alternatives.

- Early treatment of cardiogenic shock and intervention in cases of ALP toxicity enhance prognosis.
- Use of C-reactive protein and blood lactate levels to predict mortality in acute aluminum phosphide poisoning.

Limitation of the study:

Small sample size of the patients with low survival rate was the main limitations.

Conflict of interest: The authors declared that they have no conflict of interests.

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القيمة التنبؤية لمستويات اللاكتات والبروتين التفاعلي سى في الدم في المرضى المصابين بالتسمم الحاد بالفوسفيد والذين أدخلوا بمركز علاج التسمم والإدمان بمستشفى جامعة المنوفية

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تعتبر الفوسفات المعدنية ككل وفوسفيد الألومنيوم على وجه الخصوص من المبيدات الحشرية المهمة والتي تستخدم في حماية المحاصيل أثناء النقل والتخزين، على الرغم من أنها شديدة السمية وتسبب معدلات وفيات عالية. تهدف هذه الدراسة إلى تقييم القيمة التنبؤية لمستويات اللاكتات في الدم والبروتين التفاعلي سى في مرضى التسمم الحاد بالفوسفيد والذين تم حجزهم في مركز السموم بجامعة المنوفية خلال الفترة من ١ أغسطس ٢٠٢٢ إلى ٣١ ديسمبر ٢٠٢٢ والذين استوفوا معايير التضمين. وقد تم عمل استمارة طبية لكل حالة تشمل البيانات الخاصة بها بعد أخذ موافقة كتابية من المرضى أو أولياء أمورهم. استند التشخيص إلى تاريخ التعرض، وتحديد العبوة، والدلالات السريرية. ثم تم تضمين المرضى المصابين بالتسمم المتوسط والشديد فقط في الدراسة وفقاً لدرجة شدة السمية. ثم تم تجميع البيانات الاجتماعية الديموغرافية، تاريخ التسمم، تم إجراء الفحص السريري بما في ذلك العلامات الحيوية ومستوى الوعي وفقاً لمقياس غلاسكو للغيوبة والفحص الشامل. تم إجراء تخطيط صدى القلب وتم تقييم وظيفة البطين الأيسر. وتم أخذ عينات من الدم للفحوصات المخبرية الروتينية ومستويات اللاكتات والبروتين التفاعلي في الدم. ، وتم تقسيم الحالات تبعاً لمصيرهم الى أحياء وأموات.

وقد كان من أهم نتائج البحث أن العدد الكلى للحالات كان ٥٧ مريضاً (٢٦ ذكور و ٣١ إناث). وقد كان التسمم في كل الحالات انتحارياً. تراوحت أعمار المرضى المتوفيين (٩٠،٨٥%) بين ١٤-٦٣ سنة. كانت زيادة عدد الأقراص، وانخفاض درجة الحموضة، وانخفاض نسبة الأكسجين، وارتفاع معدل التنفس، وانخفاض درجة الحرارة، وانخفاض ضغط الدم الانبساطي، وارتفاع معدل النبض، وانخفاض وظيفة القلب، وانخفاض مستوى البوتاسيوم، وارتفاع مستويات CRP ومستويات اللاكتات في الدم من العوامل الهامة للتنبؤ بمصير مرضى التسمم بالفوسفيد.

الخلاصة: أن هذه الدراسة أظهرت أن فوسفيد الألومنيوم شديد السمية ذو معدل وفيات مرتفع. وأنه يمكن استخدام بروتين سى التفاعلي ومستويات اللاكتات في الدم كمؤشرات للوفيات الناتجة عن التسمم بفوسفيد الألومنيوم.