

Potential Clinical and Laboratory Prognostic Factors for Prediction of Need for ICU admission in Acute Aluminum Phosphide Poisoning

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Abstract

Background: Aluminum phosphide (AIP) is a type of fumigant which became a leading factor for suicidal poisoning in developing countries. In the absence of specific antidote, acute aluminum phosphide poisoning is considered a major public health problem. Determination of prognosis is a major concern for clinical toxicologists. Prognostic factors facilitate appropriate disposition to limited intensive care unit (ICU) beds. **Aim:** The aim of this study was to determine the impact of clinical and laboratory findings for prediction of need for intensive care unit admission in acute aluminum phosphide poisoning. **Patients and methods:** This cross sectional study was conducted on 114 acute aluminum phosphide poisoned patients admitted to Tanta University Poison Control Center (TUPCC) from May 2017 to November 2019. For these patients characteristic clinical manifestations, laboratory investigations and outcome were recorded. **Results:** Acute aluminum phosphide poisoned patients who required ICU admission represented 69.3% of cases and 91.1% of all cases died. A significant difference was recorded between patients who needed and those who didn't need ICU admission as regard Glasgow Coma Scale (GCS), pulse, random blood sugar, pH, HCO₃ and serum potassium. Moreover, respiratory rate, oxygen saturation, systolic blood pressure and serum Na were found to be good predictors for ICU admission need in acute aluminum phosphide poisoned patients. **Conclusion:** Respiratory rate, oxygen saturation, systolic blood pressure and serum Na can predict need for ICU admission in acute aluminum phosphide poisoned patients.

Key words

Aluminum phosphide, Intensive Care Unit, Prediction.

Introduction

Aluminum phosphide (AIP) is a type of fumigant. It is used to protect stored grains (Khan et al., 2020). Aluminum phosphide is easily accessible and cheap fumigant so it became a leading factor for suicidal poisoning in developing countries (Mehrpour et al., 2008, Mehrpour et al., 2012b and Nakhaee et al., 2017). When AIP gets in contact with moisture or acidity, a highly toxic phosphine gas is released (Fayyaz, 2015).

Phosphine gas is absorbed through the gastrointestinal tract. It is a mitochondrial poison which inhibits mitochondrial cytochrome oxidase, oxidative phosphorylation and cellular oxygen utilization leading to multi-organ failure (Rigobello et al., 2002, Singh et al., 2006, Proudfoot, 2009).

Acute AIP poisoned cases presented clinically with nausea, vomiting, acute respiratory distress, severe hypotension, shock and coma. Various neurological changes like ataxia, tremors, and convulsions have been observed. Acute hypoxic encephalopathy has been reported after acute AIP exposure, which may lead to death as a result of complete depression of the central nervous system and the respiratory center (Dua and Gill, 2004).

Current medical management is to provide symptomatic treatment and supportive care for almost

all cases (Mehra and Sharma, 2016). In the absence of specific antidote, acute aluminum phosphide poisoning is considered a major public health problem (Mehrpour et al., 2012b and Nakhaee et al., 2017). There is an extremely high incidence of mortality in acute AIP poisoning, even when patients are in the intensive care unit (ICU). This incidence ranges from 30 to 100% (Mehrpour et al., 2012b).

Determination of prognosis of acutely poisoned patients is a major concern for clinical toxicologists. Moreover, prognostic factors facilitate appropriate disposition to limited ICU beds (Alizadeh et al., 2014).

Aim of the Work

The aim of this study was to determine the impact of clinical and laboratory findings for prediction of need for intensive care unit admission in acute aluminum phosphide poisoning.

Patients and Methods

I. Study design and ethical consideration:

This cross sectional study was carried out in Tanta University Poison Control Center (TUPCC) in the period from the start of May 2017 to the end of November 2019. This study was approved by medical

research ethical committee of Tanta Faculty of Medicine (approval code: 33137/05/19). Valid written informed consents were taken from adult conscious patients or legal guardians of unconscious patients. Confidentiality of patients' data was considered.

II. Patients:

During the study all admitted patients aged 18 years and more with acute AIP poisoning were included. Diagnosis of acute AIP poisoning was based on a history of taking AIP tablets and relevant clinical findings (symptoms or signs). Patients less than 18 years, ingestion of previously air-exposed AIP tablets, co-ingestion of other drugs or patients with missing data in their records, were excluded from the study.

All included patients were subjected to toxicological history taking including route, amount of AIP taken, mode of poisoning and time elapsed between AIP intake and hospital admission. Assessment of consciousness level was done by Glasgow Coma Scale (GCS). Complete physical examination consisting of chest examination, abdominal examination and regular monitoring of vital signs. Laboratory investigations at time of admission were done for all patients including arterial blood gases, serum electrolytes, random blood sugar, renal and liver functions. Assessment of outcome measures including need for ICU admission, need for vasopressors and mortality.

All patients were treated according to protocol of treatment in TUPCC (Emergency and supportive measurement, fluid therapy guided by central venous pressure (CVP), noradrenaline for refractory hypotension, anti-arrhythmic agents, DC cardioversion and temporary pacemaker, sodium bicarbonate if bicarbonate level less than 15mEq/L, magnesium sulfate: 3gm over 3 hours infusion followed by 6gm per 24 hour for 3 to 5 days if indicated, H₂ receptors antagonists and extensive gastric lavage with sodium bicarbonate solution, activated charcoal should be administered).

III. Statistical analysis:

Statistical analysis was performed using Statistical Package for Social Sciences (SPSS) version 26. For quantitative data, the Shapiro-Wilk test for normality was performed. For data that followed normal distribution, values were expressed as mean \pm standard deviation.

Comparisons between two unpaired groups were carried out using independent samples T-test. For data that did not follow normal distribution, median, interquartile range, and range (minimum -maximum values) were calculated; Mann-Whitney test was used to compare between two unpaired groups. For qualitative data, the variables were summarized as frequencies (count and percentage). Pearson's Chi square test for independence, Fisher's exact test or Fisher-Freeman-Halton exact test were used to examine association between two categorical variables as appropriate.

Binomial logistic regression analysis was conducted to evaluate potential predictors of ICU admission.

Results

The present study was carried out on 114 acute aluminum phosphide (AIP) poisoned patients admitted to TUPCC in the period from the first of May 2017 to the end of November 2019. The patients' data included their toxicological data, clinical evaluation, investigations and outcome.

Acute aluminum phosphide poisoned patients who required ICU admission represented 69.3% of cases (Figure 1). Criteria for ICU admission were cardiogenic shock, need for vasopressors, need for intubation and mechanical ventilation. The toxicological data of all patients are showed in (table 1). Mode, route of poisoning and delay time showed no significant difference between patients who needed and who didn't need ICU admission. However, a significant difference was revealed between both groups as regard ingested amount of aluminum phosphide tablets.

As regard GCS, The highest score (15) represents fully conscious person while, the lowest GCS (3) is corresponding to deep coma. GCS from 13-14 is mild, GCS from 9-12 is moderate and GCS less than 9 is considered severe. A significant difference was found between acute aluminum phosphide poisoned patients who didn't need (higher score) and those who needed ICU admission (lower score). While, no significant difference was revealed between both groups as regard respiratory manifestations and gastrointestinal manifestation (Table 2).

As regard vital data, table 3 demonstrated a statistically significant difference between patients who needed and those who didn't need ICU admission as regard heart rate, blood pressure and respiratory rate. Heart rate was significantly higher in acute aluminum phosphide poisoned patients who didn't need ICU admission compared to patients who needed ICU, while blood pressure was significantly lower in acute aluminum phosphide poisoned patients who required ICU admission than patients who didn't require. Moreover, Respiratory rate was significantly higher in acute aluminum phosphide poisoned patients who needed ICU admission than who didn't need. On the other hand, no significant difference was revealed between both groups regarding temperature.

Table 4 showed the results of laboratory investigations. A statistically significant difference was observed between patients who needed and those who didn't need ICU admission as regard RBS, O₂ saturation, pH, HCO₃, serum sodium and potassium level. On the other hand, no significant difference was revealed between acute aluminum phosphide poisoned patients who didn't need and those who needed ICU admission as regard PCO₂, renal and liver functions (Table 4).

As regard outcome of acute aluminum phosphide poisoned cases, all cases who needed ICU were in need for vasopressors as shown in figure 2 and 91.1% of them died (Figure 3).

Backward, stepwise, binomial logistic regression analysis was conducted and showed that respiratory rate, O₂ saturation, serum Na and systolic blood pressure were potential factors that may affect need for ICU admission. The choice of risk factors entered into the regression model was based on clinical relevance and the p value in univariate analysis (variables were p value less than 0.2 were considered) (Table 5).

Increase in respiratory rate by one unit, when the other variables were adjusted, was associated with increased probability of need to ICU admission by 1.185 times ($p = 0.007$). Increase in O₂ saturation and systolic blood pressure by one unit, was associated with a decreased probability of need to ICU admission by 0.784 and 0.930 times, respectively ($p = 0.006$ and 0.008 respectively) (Table 5).

Table (1): Association between toxicological history and patients' need for ICU admission in acute aluminum phosphide poisoned patients (N=114)

		Need for ICU				Test of significance	
		Yes (n = 79)		No (n=35)		Test statistic	p
		n	%	n	%		
Mode	Suicidal	79	100.0%	35	100.0%	N/A	N/A
Route	Oral	79	100.0%	35	100.0%	N/A	N/A
Amount (tablets)	Range	0.25 - 3.00		0.25 - 3.00		Z = 2.419	0.016*
	Median	1.00		1.00			
	IQR	1.00 - 1.00		0.50 - 1.00			
	Mean rank	57.8		44.8			
Delay (hours)	Range	0.5 - 9.0		0.1 - 34.0		Z = 0.911	0.362
	Median	2.0		3.0			
	IQR	1.0 - 4.0		1.0 - 4.5			
	Mean rank	53.1		59.0			

IQR: interquartile range, N/A: non-applicable, Z: Mann-Whitney test *significant at $p < 0.05$.

Table (2): Association between clinical manifestations on admission and need for ICU admission in acute aluminum phosphide poisoned patients (N=114)

		Need for ICU				Test of significance	
		Yes (n = 79)		No (n=35)		Test statistic	p
		N	%	N	%		
Respiratory manifestations	No	78	98.7%	35	100.0%	FE	1.000
	Yes	1	1.3%	0	0.0%		
GIT manifestations	No	34	43.0%	11	31.4%	$X^2_{\text{chs}} = 1.368$	0.242
	Yes	45	57.0%	24	68.6%		
Neurological manifestations (GCS)	Normal (15)	64	81.0% ^s	35	100.0% ^s	$X^2_{\text{FFH}} = 7.496$	0.031*
	Mild (13-14)	10	12.7%	0	0.0%		
	Moderate (9-13)	1	1.3%	0	0.0%		
	Severe (< 9)	4	5.1%	0	0.0%		
GCS	Range	4 - 15		15 - 15		Z = 2.746	0.006*
	Median	15		15			
	IQR	15 - 15		15 - 15			
	Mean rank	54.2		65.0			

*significant at $p < 0.05$, FE: Fisher's exact test Z: Mann-Whitney test, X^2_{FFH} : Fisher-Freeman-Halton exact test.

Table (3) Independent samples T-test to compare acute aluminum phosphide poisoned patients who needed and those who didn't need ICU admission as regard vital signs on admission (N=114)

		Need for ICU		t	p
		Yes (n=79)	No (n=35)		
Pulse (beats/min)	Range	40.0 - 186.0	60.0 - 165.0	2.245	0.027*
	Mean \pm SD	89.0 \pm 22.9	99.6 \pm 23.5		
Systolic blood pressure (mmHg)	Range	40.0 - 110.0	60.0 - 150.0	6.429	<0.001*
	Mean \pm SD	78.9 \pm 14.3	102.4 \pm 18.7		
Diastolic blood pressure (mmHg)	Range	30.0 - 80.0	30.0 - 100.0	4.676	<0.001*
	Mean \pm SD	47.2 \pm 11.6	61.2 \pm 15.4		
Mean blood pressure	Range	33.0 - 153.0	40.0 - 203.0	3.813	<0.001*
	Mean \pm SD	59.1 \pm 17.9	82.8 \pm 32.4		
Respiratory rate (Cycles/min)	Range	8.0 - 48.0	16.0 - 40.0	4.196	<0.001*
	Mean \pm SD	26.4 \pm 6.8	21.6 \pm 5.1		
Temperature ($^{\circ}$ C)	Range	36.0 - 37.5	36.5 - 37.5	0.922	0.358
	Mean \pm SD	36.9 \pm 0.3	36.9 \pm 0.3		

t: Independent samples T-test; *significant at $p < 0.05$.

Table (4): Comparison between acute aluminum phosphide poisoned patients who needed and those who didn't need ICU admission as regard laboratory investigations on admission (N=114)

		Need for ICU		Test of significance	
		Yes (n=79)	No (n=35)	Test statistic	P
Random blood sugar (mg/dl)	Range	25.0 - 441.0	46.0 - 375.0	Z = 2.774	0.006*
	Median	135.0	114.0		
	IQR	107.0 - 200.0	91.0 - 130.0		
	Mean rank	63.2	44.6		
Oxygen saturation (%)	Range	45.0 - 100.0	79.0 - 100.0	t= 6.697	<0.001*
	Mean \pm SD	85.2 \pm 10.9	94.9 \pm 4.5		
pH	Range	6.7 - 7.6	7.1 - 7.6	t= 4.383	<0.001*
	Mean \pm SD	7.3 \pm 0.1	7.4 \pm 0.1		
HCO ₃ (mEq/L)	Range	2.6 - 23.0	7.2 - 23.8	t= 3.646	<0.001*
	Mean \pm SD	13.0 \pm 4.5	16.1 \pm 3.7		
PCO ₂	Range	8.0 - 72.0	12.0 - 38.6	t= 1.282	0.203
	Mean \pm SD	27.0 \pm 10.6	25.1 \pm 5.6		
Serum sodium (mEq/L)	Range	131.0 - 159.0	130.0 - 147.0	t= 2.295	0.024*
	Mean \pm SD	143.4 \pm 5.6	140.9 \pm 4.1		
Serum potassium (mEq/L)	Range	1.1 - 6.8	2.5 - 5.3	t= 2.116	0.037*
	Mean \pm SD	3.6 \pm 0.7	3.8 \pm 0.6		
BUN (mg/dl)	Range	12.0 - 72.0	15.0 - 71.0	t= 0.656	0.513
	Mean \pm SD	29.6 \pm 10.4	31.0 \pm 11.1		
Serum Creatinine (mg/dl)	Range	0.6 - 1.9	0.4 - 2.2	t= 1.658	0.100
	Mean \pm SD	1.1 \pm 0.3	1.0 \pm 0.3		
ALT (IU/L)	Range	7.0 - 161.0	7.0 - 120.0	Z = 1.516	0.130
	Median	22.0	18.0		
	IQR	15.0 - 30.0	12.0 - 29.0		
	Mean rank	60.6	50.5		
AST (IU/L)	Range	8.0 - 151.0	8.0 - 88.0	Z = 0.519	0.603
	Median	24.0	26.0		
	IQR	17.0 - 33.0	15.0 - 39.0		
	Mean rank	56.4	59.9		

IQR: interquartile range; t: Independent samples T-test; Z: Mann-Whitney test; *significant at $p < 0.05$.

Table (5): Backward stepwise binomial logistic regression for assessing factors affecting the need for ICU admission in acute aluminum phosphide poisoned patients

	B	SE	Wald	p	OR	95% CI for OR	
O ₂ saturation (%)	-0.243	0.089	7.548	0.006*	0.784	0.659	0.933
RR (Cycle/min)	0.170	0.063	7.219	0.007*	1.185	1.047	1.341
Na (mEq/L)	0.121	0.073	2.772	0.096	1.128	0.979	1.301
Systolic bl.pr (mmHg)	-0.072	0.027	7.042	0.008*	0.930	0.882	0.981
Constant	8.219	13.140	0.391	0.532	3709.612		

Variable(s) entered on step 1: Pulse (beats/min), O₂ saturation (%), RR (Cycle/min), RBS (mg/dl), pH, HCO₃ (mEq/L), Na (mEq/L), K (mEq/L), Creatinine (mg/dl), Systolic bl.pr (mmHg)._a

CI: confidence interval; OR: odds ratio; SE: standard error; * significant at $p < 0.05$

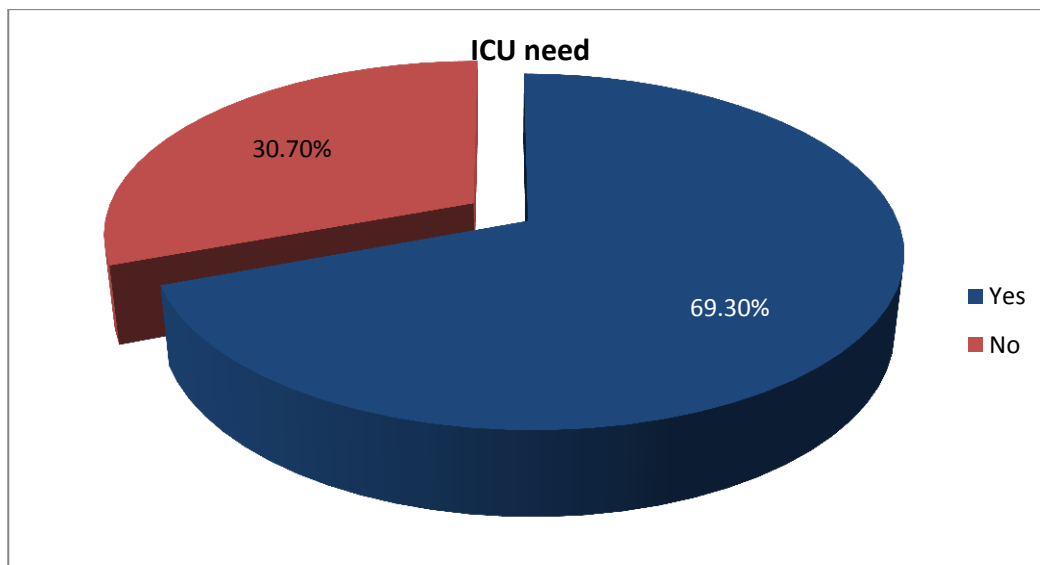


Figure (1): Frequency of acute aluminum phosphide poisoned patients according to need of ICU admission (N=114).

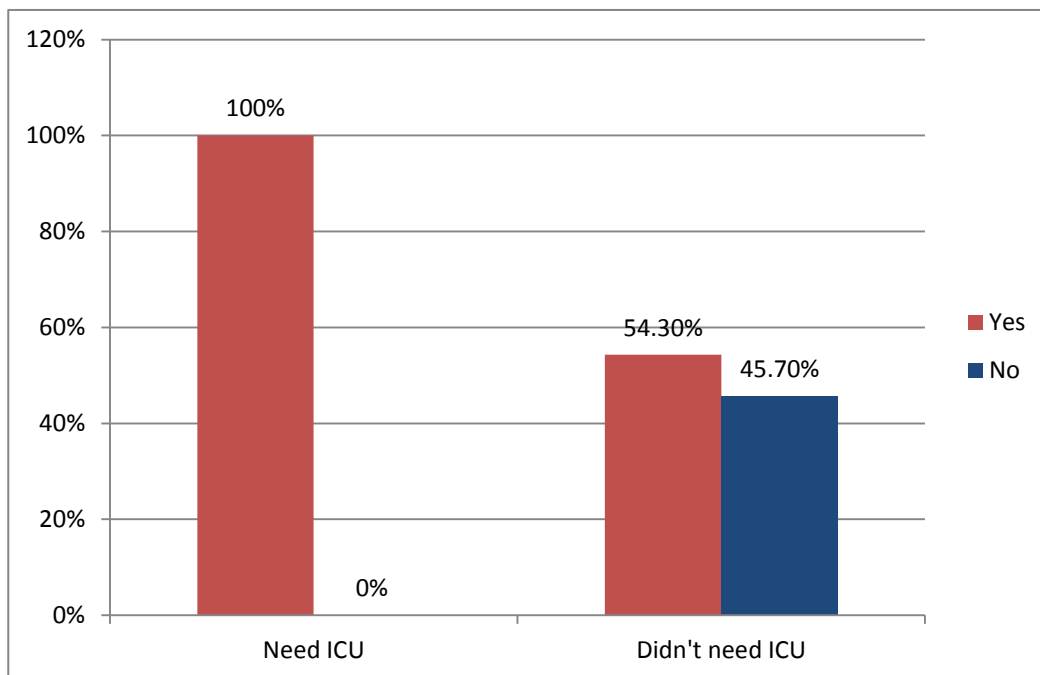


Figure (2): Association between need for vasopressors and need for ICU admission in acute aluminum phosphide poisoned patients (N=114)

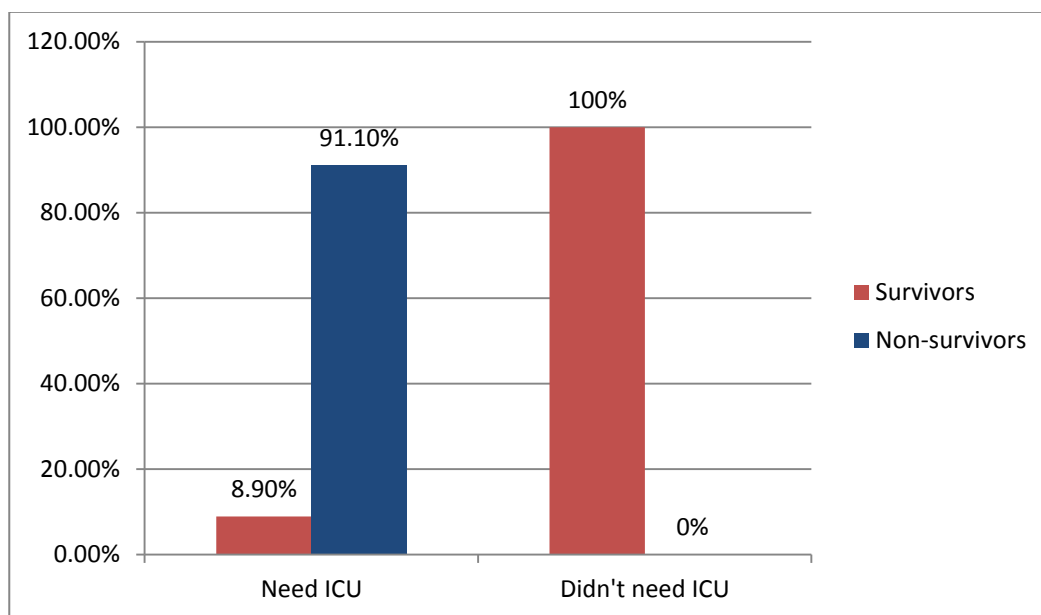


Figure (3): Association between mortality and need for ICU admission in acute aluminum phosphide poisoned patients (N=114)

Discussion

Acute aluminum phosphide poisoning is considered a fatal poisoning especially in absence of specific antidote and management only by supportive measures. So it is important to predict outcome of acute aluminum phosphide poisoned patients at admission for appropriate management and use of hospital resources. It is advisable to select patients who will get benefit from the available resources in order to improve their outcome (Ghonem et al., 2020).

Criteria for intensive care unit (ICU) admission of patients vary widely across countries. In some countries, ICU admission was firmly limited to severe and life-threatening poisoning while, other countries habitually admit all poisoned patients to the ICU, irrespective to the severity of symptoms on the time of admission. Decision of ICU admission is affected by different factors such as availability of resources and physicians prognostication (Taghaddosinejad et al., 2012).

In poisoned patients, intensive care unit admission requires rapid diagnosis and supportive care. So accurate detection of ICU admission predictors can help clinicians to make decisions like whether and when patients may benefit from ICU admission (Assaf et al., 2019).

In the current study, no significant difference was detected between acute aluminum phosphide poisoned patients who needed ICU admission and those who didn't need ICU admission as regard mode and route of poisoning, respiratory and gastrointestinal manifestations and temperature. Moreover, PCO₂, liver and renal functions showed no significant difference between both groups.

A significant difference was found between acute aluminum phosphide poisoned patients who didn't need and those who needed ICU admission as regard ingested amount of aluminum phosphide tablets. The amount ranged from quarter tablet to 3 tablets in

both groups. In accordance to this result Shadnia et al. (2010) found that the amount of ingested aluminum phosphide tablets in acute aluminum phosphide poisoned patients who admitted to ICU ranges from quarter tablet to 4 tablets. Each tablet (3 grams) contains 56% of aluminum phosphide and releases 1 gram phosphine gas. The fatal dose of aluminum phosphide by ingestion in an adult is 150–500 mg and this could explain the toxicity from relative small amount of aluminum phosphide (Singh et al., 2014).

As regard GCS, a significant difference was found between acute aluminum phosphide poisoned patients who didn't need and those who needed intensive care unit (ICU) admission. In patients who needed ICU admission, it ranged from 4-15. This coincides with Shadnia et al. (2018) who reported that GCS ranged from 3-15 in acute aluminum phosphide poisoned patients who admitted to ICU in their studies. Neurological manifestations caused by acute aluminum phosphide poisoning may be due to hypotension which results in brain anoxia (Garg, 2020).

Heart rate was significantly higher in acute aluminum phosphide poisoned patients who didn't need ICU admission compared to patients who needed ICU. In patients who needed ICU admission, heart rate ranged from 40 to 186 beats/min. In accordance with this result, Erfantalab et al. (2017) demonstrated that heart rate ranged from 49 to 144 beats/min. Tachycardia in aluminum phosphide poisoning could be explained by sympathetic over activity or as a reflex due to hypotension (Singh and Bhalla., 2015).

Blood pressure was significantly lower in acute aluminum phosphide poisoned patients who required ICU admission than patients who didn't require ICU admission. The majority of cases who needed ICU admission had low blood pressure. In agreement with this result, Shadnia et al. (2018) reported that the majority of acute aluminum phosphide poisoned

patients who were admitted to ICU presented with hypotension. Intractable shock in acute aluminum phosphide poisoning may be due to arrhythmia, myocardial damage and depression, small vessel injury and peripheral vasodilatation (Anand et al., 2011).

Respiratory rate was significantly higher in acute aluminum phosphide poisoned patients who needed ICU admission than those who didn't need ICU admission. Respiratory rate ranged from 8 to 48 cycles/min. Similarly, Shadnia et al. (2010) found that respiratory rate ranged from 0 to 55 cycles/min in acute aluminum phosphide poisoned patients who were admitted to ICU. Tachypnea could be a sign of metabolic acidosis which was the most common type of acid base disturbance in the current study (Abd Elghany et al., 2018).

Random blood sugar (RBS) of patients who needed ICU admission, ranged from 25 to 441 mg/dl. It was significantly higher in acute aluminum phosphide poisoned patients who needed ICU admission compared to those who didn't need it. This partially agreed with Erfantalab et al. (2017) who demonstrated that RBS ranged from 25 to 418 mg/dl in acute aluminum phosphide poisoned patients who were admitted to ICU. Hyperglycemia may be attributed to insulin synthesis inhibition with stimulation of glucagon, cortisol and adrenaline secretion. On the other hand, inhibition of gluconeogenesis and glycogenolysis, liver and adrenal cortex damage may contribute to decreased synthesis of adrenaline and glucagon and finally hypoglycemia (Mehrpour et al., 2008 and Mehrpour et al., 2012a).

There was a significant difference between acute aluminum phosphide poisoned patients who didn't need and who needed ICU admission as regard arterial blood gases. HCO_3^- ranged from 2.6 to 23 and pH ranged from 6.7 to 7.6 in acute aluminum phosphide poisoned patients in the current study. In harmony with this result, Mathai and Bhanu, (2010) found that acute aluminum phosphide poisoned patients who were admitted to ICU showed HCO_3^- ranged from 3.9 to 29 and pH ranged from 6.80 to 7.51. Metabolic acidosis in acute aluminum phosphide poisoned cases is attributed to hypoperfusion of tissues and inhibition of oxidative phosphorylation which lead to accumulation of lactic acid (Berry et al., 2015).

A significant difference was recorded between those who needed ICU admission and those who didn't need admission as regard serum sodium and potassium level. In acute aluminum phosphide poisoned patients who needed ICU admission, serum sodium level ranged from 131 to 159 mEq/L and serum potassium level ranged from 1.1 to 6.8 mEq/L. This coincides with Louriz et al. (2009) who demonstrated that serum sodium level ranged from 125 to 151 mEq/L and serum potassium level ranged from 3.20 – 6.6 mEq/L in acute aluminum phosphide poisoned patients who were admitted to ICU. Hypokalemia may be attributed to repeated vomiting following aluminum phosphide ingestion.

As regard outcome of acute aluminum phosphide poisoned cases, all cases that needed ICU

were in need for vasopressors and 91.1% of them died. This could be explained by the highly cardiotoxic property of aluminum phosphide without specific antidote (Abd Elghany et al., 2018). Moreover, aluminum phosphide leads to refractory hypotension which doesn't respond to crystalloid administration and the second step in management is administration of vasopressors (Farahani et al., 2016).

Backward, stepwise, binomial logistic regression analysis was conducted and showed that respiratory rate, O_2 saturation, serum Na and systolic blood pressure were potential factors that may affect need for ICU admission.

Few studies were conducted to evaluate ICU need in acute aluminum phosphide poisoning and this could be attributed to considering acute aluminum phosphide poisoning a severe poisoning from the start and admit the patients to ICU immediately after arrival in some countries.

Conclusion

This study concluded that respiratory rate, O_2 saturation, systolic blood pressure and serum Na can predict need for ICU admission in acute aluminum phosphide poisoned patients.

References

- Abd Elghany S, Heshmat M, Oreby M and Elsarnagawy G (2018): Evaluation of various scoring systems in prediction of acute aluminum phosphide (alp) poisoning outcome. *Ain Shams Journal of Forensic Medicine and Clinical Toxicology*, 30 (1): 117-127.
- Alizadeh A M, Hassanian-Moghaddam H, Shadnia S, Zamani N and Mehrpour O (2014): Simplified acute physiology score II /acute physiology and chronic health evaluation II and prediction of the mortality and later development of complications in poisoned patients admitted to intensive care unit. *Basic and Clinical Pharmacology and Toxicology*, 115 (3): 297-300.
- Anand R, Binukumar B K and Gill K D (2011): Aluminum phosphide poisoning: an unsolved riddle. *Journal of Applied Toxicology*, 31(6): 499-505.
- Assaf A, Abd El Kareem M and Hasb Elnabi M (2019): Outcome prediction in acutely intoxicated patients admitted to intensive care unit. *Ain Shams Journal of Forensic Medicine and Clinical Toxicology*, 33 (2): 16-23.
- Berry A, Singh G, Kaur S J and Bala K (2015): Aluminium phosphide: toxicity mechanism and credible treatments. *World Journal of Pharmacy and Pharmaceutical Sciences*, 4 (10): 2276-2293.
- Dua R and Gill K D (2004): Effect of aluminium phosphide exposure on kinetic properties of cytochrome oxidase and mitochondrial energy metabolism in rat brain. *Biochimica Et Biophysica Acta (BBA)-General Subjects*, 1674 (1): 4-11.

- Erfantalab P, Soltaninejad K, Shadnia S, Zamani N, Hassanian-Moghaddam H, Mahdavinejad A and Damaneh B H (2017): Trend of blood lactate level in acute aluminum phosphide poisoning. *World Journal of Emergency Medicine*, 8 (2): 116-120.
- Farahani M V, Soroosh D and Marashi S M (2016): Thoughts on the current management of acute aluminum phosphide toxicity and proposals for therapy: An evidence-based review. *Indian Journal of Critical Care Medicine: (Peer-Reviewed, Official Publication of Indian Society of Critical Care Medicine)*, 20 (12): 724-730.
- Fayyaz A F (2015): The relationship between rice tablet consumption and pathological signs leading to death: a study in Tehran-Iran. *Annals of Military and Health Sciences Research*, 13(1): 21-25.
- Garg K K (2020): Review of aluminum phosphide poisoning. *International Journal of Medical Science and Public Health*, 9 (7): 392-400.
- Ghonem M M, El Sharkawy S I and Lashin H I (2020): Predictive variables of acute aluminum phosphide poisoning outcome: a new proposed model. *The Egyptian Journal of Forensic Sciences and Applied Toxicology*, 20 (2): 45-60.
- Khan M K, Ahmad F, Mahmood A and Azmat J (2020): Aluminum phosphide poisoning and blast in gastric tube a rare phenomenon-a case report. *Asia Pacific Journal of Medical Toxicology*, 4 (2): 60-62.m
- Louriz M, Dendane T, Abidi K, Madani N, Abouqal R, and Zeggwagh A A (2009): Prognostic factors of acute aluminum phosphide poisoning. *Indian Journal of Medical Sciences*, 63 (6): 227-234.
- Mathai A and Bhanu M S (2010): Acute aluminium phosphide poisoning: Can we predict mortality?. *Indian Journal of Anaesthesia*, 54 (4): 302-307.
- Mehra A and Sharma N (2016): ECMO: A ray of hope for young suicide victims with acute aluminum phosphide poisoning and shock. *Indian Heart Journal*, 68 (3): 256-257.
- Mehrpour O, Aghabiklooei A, Abdollahi M and Singh S (2012a): Severe hypoglycemia following acute aluminum phosphide (rice tablet) poisoning; a case report and review of the literature. *Acta Medica Iranica*, 50 (8): 568-571.
- Mehrpour O, Alfred S, Shadnia S, Keyler D E, Soltaninejad K, Chalaki N and Sedaghat M (2008): Hyperglycemia in acute aluminum phosphide poisoning as a potential prognostic factor. *Human & Experimental Toxicology*, 27 (7): 591-595.
- Mehrpour O, Jafarzadeh M and Abdollahi M (2012b): A systematic review of aluminium phosphide poisoning. *Archives of Industrial Hygiene and Toxicology*, 63 (1): 61-73.
- Nakhaee S, Mehrpour O and Balali-Mood M (2017): Does N-acetyl cysteine have protective effects in acute aluminum phosphide poisoning?. *Indian Journal of Critical Care Medicine*, 21 (8):65-67.
- Proudfoot A T (2009): Aluminium and zinc phosphide poisoning. *Clinical Toxicology*, 47 (2): 89-100.
- Rigobello M P, Scutari G, Boscolo R and Bindoli A (2002): Induction of mitochondrial permeability transition by auranofin, a gold (I)-phosphine derivative. *British Journal of Pharmacology*, 136 (8): 1162-1168.
- Shadnia S, Mehrpour O and Soltaninejad K (2010): A simplified acute physiology score in the prediction of acute aluminum phosphide poisoning outcome. *Indian Journal of Medical Sciences*, 64 (12): 532-539.
- Shadnia S, Zamani N, Hassanian-Moghaddam H, Shafaroodi H, Padandar M and Rezaeizadeh M H (2018): Prognostic value of cortisol and thyroid function tests in poisoned patients admitted to toxicology ICU. *World Journal of Emergency Medicine*, 9 (1): 51-55.
- Singh S and Bhalla A (2015): Aluminum phosphide poisoning. *Journal of Mahatma Gandhi Institute of Medical Sciences*, 20 (1): 15-19.
- Singh S, Bhalla A, Verma S K, Kaur A and Gill K (2006): Cytochrome-c oxidase inhibition in 26 aluminum phosphide poisoned patients. *Clinical Toxicology*, 44 (2): 155-158.
- Singh Y, Joshi S C, Satyawali V and Gupta A (2014): Acute aluminium phosphide poisoning, what is new?. *The Egyptian Journal of Internal Medicine*, 26 (3): 99-103.
- Taghaddosinejad F, Sheikhezadi A, Yaghmaei A, Mehrpour O and Schwake L (2012): Epidemiology and treatment of severe poisoning in the intensive care unit: lessons from a one-year prospective observational study. *Journal of Clinical Toxicology*, 5 (1): 2161-0495.

الملخص العربي

العوامل السريرية والمعملية المحتملة للتنبؤ بالحاجة إلى دخول وحدة العناية المركزة في حالات التسمم الحاد بفوسفيد الألومنيوم

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المقدمة: يعتبر فوسفيد الألومنيوم نوع من أنواع التبخير التي أصبحت عاملاً رئيسياً للتسمم الانتحاري في البلدان النامية. في عدم وجود ترياق محدد، يعتبر التسمم الحاد بفوسفيد الألومنيوم مشكلة صحية عامة كبرى. و أصبح تحديد المصير المرضي هو مصدر قلق كبير لعلماء السموم الاكلينيكية. تسهل العوامل التنبؤية التصرف المناسب في الأسرة المحدودة لوحدة العناية المركزة.

الهدف: تهدف هذه الدراسة الى تحديد العوامل السريرية والمعملية المحتملة للتنبؤ بالحاجة إلى دخول وحدة العناية المركزة في حالات التسمم الحاد بفوسفيد الألومنيوم.

الطريقة المستخدمة في البحث: أجريت هذه الدراسة على ١١٤ مريضاً مصاباً بالتسمم الحاد بفوسفيد الألومنيوم تم دخولهم مركز جامعه طنطا لعلاج حالات التسمم من مايو ٢٠١٧ إلى نوفمبر ٢٠١٩. بالنسبة لهؤلاء المرضى ، تم تسجيل الفحص الاكلينيكي والتحليل المعملية والمصير المرضي الخاص بهم.

النتائج: مثل المرضى المصابين بالتسمم الحاد بفوسفيد الألومنيوم والذين احتاجوا لدخول وحدة العناية المركزة ٦٩.٣٪ من الحالات وتوفي ٩١.١٪ من جميع الحالات. تم تسجيل فرق ذو دلالة احصائية بين الحالات الذين كانوا بحاجة الى دخول وحدة العناية المركزة والذين لم يكونوا بحاجة الى دخولها وذلك من حيث مقياس الغلاسكو، النبض، سكر الدم العشوائي، بيكربونات و مستوى البوتاسيوم في الدم. وعلاوة على ذلك، فقد وجد أن معدل التنفس، تشبع الدم بالأكسجين، ضغط الدم الانقباضي و مستوى الصوديوم في الدم عوامل جيدة للتنبؤ بالحاجة الى دخول وحدة العناية المركزة في المرضى المصابين بالتسمم الحاد بفوسفيد الألومنيوم.

الاستنتاج: يمكن لمعدل التنفس، وتشبع الدم بالأكسجين و ضغط الدم الانقباضي و الصوديوم التنبؤ بالحاجة الى دخول وحدة العناية المركزة في المرضى المصابين بالتسمم الحاد بفوسفيد الألومنيوم.