PREDICTORS OF PROGNOSIS IN ACUTE ALUMINUM PHOSPHIDE POISONING

BY

Ahmad A. El-Ebiary, Fatma M. Elgazzar, Mohammed A.Soliman*, Osama M. Shouip*

Forensic Medicine and Clinical Toxicology Department, Faculty of Medicine, Tanta University, Tanta, Egypt,

**Internal Medicine Department, Alnahdah Hospital, Muscat, Oman.*

***Cardiology Department, Faculty of Medicine, Tanta University, Tanta, Egypt*

ABSTRACT

Aluminum phosphide (AP) is a highly toxic pesticide that is commonly used for grain preservation. Acute AP poisoning is a serious public health problem and a real challenge for physicians in developing countries because of the high rates of morbidity and mortality. This study was conducted to identify predictors that might be useful in determination of prognosis for cases of acute AP intoxication. All cases of aluminum phosphide poisoning that had been referred to Tanta University Poison Control Center from March 2013 to February 2015 were included. Patients were subjected to full history taking including demographic features, and their clinical examination data were reported. Electrocardiography, serum cardiac troponin, arterial blood gases, complete blood count were recorded, and their associations with the patients' outcomes were evaluated. Forty cases (11 males and 29 females) of acute AP poisoning were included in this study. Mortality rate was 67.5%. A statistically significant association was found between each of abnormal electrocardiography findings, elevated serum troponin level, low blood pH, serum bicarbonate, and serum glucose with the risk of mortality. Other alarming risk factors included suicidal ingestion, altered consciousness, hypokalemia, and leukocytosis. It could be concluded that, patients presenting with one or more of these findings are at higher risk of mortality. Early anticipation of these risks by the attending physician may help proper early intervention and might minimize mortality and improve outcome in these cases.

Keywords: *aluminum phosphide; fumigant; mortality; risk factors; severity.*

INTRODUCTION

Aluminum phosphide (AP) is one of the most commonly used grain fumigants because of its superior properties. It is highly potent and toxic to all stages of insects, does not affect seed viability, and leaves little residue on food grains (Mogh-

adamnia, 2012).

Because of its high toxicity, low cost, and easy accessibility; AP is one of the main causes of poisoning in developing countries. Patients commit suicide by ingesting tablets, which come in contact with aqueous solutions in the stomach and liberate phosphine gas immediately. The released gas is absorbed via the lungs causing cell hypoxia as a result of inhibition of oxidative phosphorylation (Louriz et al., 2009).

In acute AP poisoning, almost all organs are affected by the toxin leading to a variety of signs and symptoms. Early symptoms include nausea, vomiting, abdominal pain, dyspnea, anxiety, agitation, and garlic smell on the breath. Tachycardia, tachypnea, acidosis, marked hypotension, and shock, which is usually unresponsive to conventional treatment, are also observed. Patients remain mentally clear till cerebral anoxia due to shock supervenes resulting in drowsiness, delirium, and coma. Some common complications of acute AP poisoning include arrhythmias, acute renal failure, and disseminated intravascular coagulation. In addition, pulmonary edema, hepatitis, pericarditis, congestive heart failure, and acute gastrointestinal hemorrhage are occasionally noticed (Katira et al., 1990; Singh et al., 1996; Moghadamnia, 2012).

The specified fatal dose of AP is 0.15-0.5 gram. Most of the patients present with ingestion of three or more tablets, which invariably results in death. More than 90% of the patients die within 24 hours and the commonest cause of death in this group is heart dysrhythmia. However, the average time interval between ingestion of AP and death is three hours. Death after 24 hours is usually due to shock, acidosis, adult respiratory distress syndrome, and cardiac dysrhythmia. The mortality rate is highly variable ranging from 37-100% (Chugh et al., 1991; Wahab et al., 2009).

In order to provide the best possible care, any health care system should be capable of evaluation of the patients' outcomes. In medicine, an increased interest in measuring the severity of illness and predicting outcome in critically ill patients has been observed (Henderson, 2004). Owing to presence of many factors that may affect the outcome of patients with acute AP poisoning, besides the lack of specific antidote for this dangerous toxicity, physicians are in need for recognizing risk factors and predicting outcome in those patients soon after presentation. This allows more intensive treatment and monitoring, and may thereby decrease morbidity and mortality. Besides, acute AP toxicity is a big problem with major consequences especially in Egypt and other developing countries, and unfortunately there is a noticeable lack of studies regarding predic-

tors of acute AP toxicity outcomes. Hence the aim of the present study was to identify predictors to support determination of prognosis for cases of acute AP toxicity.

SUBJECTS AND METHODS

This analytical cross sectional study got the approval of the local Research Ethics Committee. An informed written consent was obtained from each patient or patient's close relatives for participation. All individual information was securely protected and available to investigators only.

Patients :

All cases with history of exposure and/or clinical manifestations of acute AP poisoning presenting to Tanta University Poison Control Center, from the beginning of March 2013 to the end of February 2015 were included. Diagnosis of acute AP poisoning based on history (self-report or by attendants) and clinical manifestations. Patients with history of cardiovascular disease and those who coingested cardiotoxic substances such as digitalis, cyclic antidepressants, antipsychotics, class I antiarrythmics and anticholinergics were excluded.

Methods :

All patients were subjected to full history taking with special regard to demographic features, circumstances of exposure, compound involved, time elapsed between exposure and admission to the hospital, and underlying diseases. In addition, vital signs, level of consciousness according to Glasgow Coma Scale, gastrointestinal, neurological, respiratory, and cardiovascular findings were recorded. Investigations included electrocardiography (ECG), serum cardiac troponin, arterial blood gases (ABG), complete blood count (CBC), and routine blood biochemistry were done. Each blood sample was collected under complete aseptic technique with a disposable syringe. Arterial samples were collected in heparinized syringes and used for arterial blood gas (ABG) analysis, while venous samples were used for other measurements. ECG analysis included the rate, rhythm, ST/T abnormalities, conduction defects, and measurement of PR and QT intervals. The QT interval was corrected (QTc) according to the formula of Bazett (Bazett, 1920). QTc was considered prolonged when it was longer than 0.440 second (Crotti et al., 2008).

The main outcome of patients either survival or death was recorded.

Statistical analysis :

Continuous variables were expressed as means and standard deviations and categorical variables as numbers and percentages. For comparisons between patient groups (survivors and non survivors) Chi-square test was used for categorical variables. The normal distribution of quantita-

tive variables was tested by Kolmogorov–Smirnov test. The statistical comparison was done with independent Student t-test and Mann Whitney U-test for parametric and nonparametric variables respectively. Correlations of some variables with mortality were assessed using Pearson and Spearman rank correlation coefficient. P values of 0.05 or less were considered statistically significant. All analyses were performed using SPSS 15.0 for Windows (SPSS Inc., Chicago, Illinois, USA).

RESULTS

During the study period, according to inclusion criteria, forty patients with acute AP poisoning (11 males, 29 females), of age ranging from 1 to 54 years, with a mean age of 20.67 ± 12.93 years were recruited. Most (67.50%) of the patients were in the age group "<20 years". In most (88%) of the patients, toxicity occurred by oral ingestion with the majority (72.50%) of them alleged intentional suicide. Average time elapsed between poisoning and admission at the hospital was 5.70 ± 5.90 hours with the highest percentage (82.50 %) of patients came to hospital with a delay of ≤ 6 hours. The majority (80%) of the studied patients were hypotensive, while about two thirds (65%) presented with tachycardia and 70% presented with tachypnea. Most (80%) of the patients had vomiting, whereas 60% of them presented to hospital with impaired consciousness. Additionally, ECG changes were found in 32 patients (80%) at the time of admission. Eleven (27.5%) cases had sinus tachycardia. ECG showed atrial fibrillation, elevated ST segment, depressed ST segment, right bundle branch block, and prolonged QTc in 10%, 20%, 5%, 10%, and 7.5% of the studied patients respectively. In addition, each of inverted T wave, premature ventricular contractions, and cardiac asystole were detected in only one case (3.75%) (Table 1).

Table (2) shows that only 40% of the studied patients were positive for cardiac troponin qualitative assay. The evaluation of ABG showed that the majority of the patients had metabolic acidosis at the time of admission. Furthermore, it was found that 27.5% of the patients had pH ≤ 7.15, while 50% of them had pH more than 7.15 and less than 7.35. Nearly half (45%) of cases were hypokalemic, 42.5% of them showed abnormal serum sodium levels, and 47.5% showed hyperglycemia. Leukocytosis and elevated serum creatinine, AST, and ALT levels were reported in 35%, 55%, 37.5%, and 55% of the patients respectively.

Table (3) shows that 27 patients died with an overall mortality rate of about 67.5%. Neither gender nor age of the studied patients showed a statistically significant difference between survivors and non survivors. On the other hand, a statistical-

ly significant difference between both groups was observed as regards the route of intoxication, the manner of toxicity whether intended suicidal or accidental exposure, and the time interval between exposure and hospital admission. In addition, impaired consciousness at time of admission showed a statistically significant association with the outcome where it was present in 81.5% of non survivors. Conversely, history of vomiting did not show a statistically significant association with mortality. This work revealed also a statistically significant association between ECG findings and occurrence of death where the majority (92.6%) of non survivors showed ECG abnormalities at the time of admission. Specifically, the presence of ST segment abnormalities either elevation or depression was significantly associated with mortality and showed also positive correlation with it ($r = 0.398$, $p = 0.011$). In addition, a statistically significant association was present between serum cardiac troponin and outcome where 59.3% of non survivors showed a positive result. Furthermore, a significant positive correlation between positive serum cardiac troponin assay and mortality was detected ($r = 0.567$, $p = 0.000$).

The means of Glasgow Coma Score (GCS) and vital signs including the systolic blood pressure (SBP) and respiratory rate in survivors (100.00 ± 16.83 and 19.30 ± 500) and non survivors (67.40 ± 14.83 and 32.44 ± 9.07) were significantly different. Furthermore, a significant reverse correlation was found between SBP and mortality ($r = -0.721$, $p = 0.000$). Regarding laboratory investigations, a statistically significant difference was detected between survivor and non-survivor groups regarding each of the following: blood pH, serum bicarbonate level, random blood sugar, serum potassium, and total leukocytic count (Table 4).

Table (1) : Demographic data, characters of toxic exposure, clinical findings, and ECG findings of the studied patients (n=40).

Characteristic parameters	n	%
Age (years)		
< 20	27	67.50
20–40	8	20.0
> 40	5	12.50
Sex		
Male	11	27.50
Female	29	72.50
Route of toxicity		
Oral	32	88.0
Inhalation	8	20.0
Manner of toxicity		
Alleged intentional	29	72.50
Alleged accidental	11	27.50
Prehospitalization period		
≤ 6 hours delay	33	82.50
> 6 hours delay	7	17.50
Vital signs		
Hypotension	32	80.0
Tachycardia	26	65.0
Bradycardia	3	7.50
Tachypnea	28	70.0
Bradypnea	1	2.50
Gastrointestinal manifestations		
Vomiting	32	80.0
Colic	10	25.0
CNS manifestations		
Impaired consciousness	24	60.0
ECG changes		
Normal sinus rhythm	25	62.50
Sinus tachycardia	11	27.50
Atrial fibrillation	4	10.0
Elevated ST segment	8	20.0
Depressed ST segment	2	5.0
Right bundle branch block	4	10.0
Prolonged QTc	2	7.50
Inverted T wave	1	3.75
Premature ventricular contractions	1	3.75
Asystole	1	3.75

n : number, CNS: central nervous system, ECG: electrocardiogram, QTc: corrected QT interval.

Table (2) : Baseline laboratory findings of the studied patients (n=40).

Laboratory findings	n	%
Serum cardiac troponin		
Positive	16	40.00
Negative	24	60.00
Serum potassium level (mEq/L)		
Normal (3.5 – 5.5)	22	55.00
Hypokalemia (< 3.5)	18	45.00
Serum sodium level (mEq/L)		
Normal (135 – 145)	23	57.50
Hyponatremia (<135)	7	17.50
Hypernatremia (>145)	10	25.00
Random blood sugar (mg/dl)		
Normal (70–140)	21	52.50
Hyperglycemia (> 140)	19	47.50
Blood pH		
7.35–7.45	8	20.00
> 7.45	1	2.50
< 7.35 and > 7.15	20	50.00
≤ 7.15	11	27.50
Total leukocytic count (cell/mm^3)		
Normal (7000 – 11000)	26	65.00
Leukocytosis (> 11000)	14	35.00
Serum creatinine level (mg/dl)		
Normal (0.8-1.2)	18	45.00
Elevated (> 1.2)	22	55.00
Serum AST level (U/L)		
Normal (8-20)	25	62.50
Elevated (>20)	15	37.50
Serum ALT level (U/L)		
Normal (8-20)	18	45.00
Elevated (>20)	22	55.00

n : number, AST: aspartate aminotransferase, ALT: alanine aminotransferase.

Table (3) : Association between demographic data, characters of toxic exposure, clinical findings, and ECG findings of the studied patients and the outcome.

Parameter	All patients (n= 40)		Survivors (n= 13)		Non survivors (n= 27)		*Chi square test* *P value*
	N	%	n	%	n	%	
Gender							
Male	11	27.50	6	46.2	5	18.5	0.146
Female	29	72.50	7	53.8	22	81.5	
Age (years)							
<20	27	67.5	10	76.92	17	62.92	0.665
20-40	8	20.0	2	15.38	6	22.22	
>40	5	12.5	1	7.69	4	14.81	
Route							
Oral	32	88	7	53.8	25	92.59	0.014*
Inhalation	8	12	6	46.2	2	7.41	
Manner							
Alleged suicidal	29		6	46.2	23	85.2	0.027*
Alleged accidental	11		7	53.8	4	14.8	
Delay hours							
≤ 6 hours delay	33	82.50	7	53.84	26	96.29	0.004*
> 6 hours delay	7	17.50	6	46.15	1	3.70	
Vomiting							
Yes	32	80.0	13	100.0	19	70.4	0.076
No	8	20.0	0	0.00	8	29.6	
Consciousness							
Normal	16	40.0	11	84.6	5	18.5	0.000*
Impaired	24	60.0	2	15.4	22	81.5	
ECG findings							
Normal	8	20.0	6	46.2	2	7.4	0.014*
Abnormal	32	80.0	7	53.8	25	92.6	
ST segment							
Elevation	8	20.0	0	0.00	8	29.6	0.040*
Depression	2	5.0	0	0.00	2	7.4	
Serum cardiac troponin							
Positive	16	40.0	0	0.00	16	59.3	0.000*
Negative	24	60.0	13	100.0	11	40.7	

n : number, ECG: electrocardiogram., *significant ($p < 0.05$).

Table (4) : Differences between survivor and non-survivor groups as regards vital signs, GCS, and routine laboratory investigations.

Parameter	All patients (n= 40)		Survivors (n= 13)	Non survivors (n= 27)	*Independent - t test P value*
	Mean ± SD	Range	Mean ± SD	Mean ± SD	
Systolic blood pressure	78.00±21.74	30-130	100.00±16.83	67.40±14.83	0.000*
Respiratory rate	28.17±10.07	15-48	19.30±500	32.44±9.07	0.000*
GCS	11.70±4.05	3-15	14.84±.37	10.18±4.15	0.000*
Random blood sugar (mg/dl)	173.85±81.96	79-461	123.15±58.01	198.25±81.36	0.005*
Total leukocytic count (cell/mm^3)	9482.50±3445.83	4000-16000	7661.53±2596.32	10359.25±3499.03	0.018*
Serum AST(U/L)	51.65±72.87	10-290	57.61±84.57	48.77±68.10	0.724
Serum ALT(U/L)	56.82±101.03	12-500	89.84±159.42	40.92±52.66	0.300
Parameter	**Mean ± SD**	**Range**	**Mean rank**	**Mean rank**	***Mann Whitney U test P value***
Pulse rate	112.27±32.62	0-180	22	19.78	0.572
Blood pH	7.24±.10	7.13-7.48	32.08	14.93	0.000*
Serum bicarbonate (mEq/L)	11.75±5.82	5.3-26.3	32.46	14.74	0.000*
Serum potassium (mEq/L)	3.40±.55	2.4-4.4	27.46	17.15	0.009*
Serum sodium (mEq/L)	140.99±.8.28	125.6-153.1	27.54	17.11	0.570
Serum creatinine (mg/dl)	1.38±.49	0.6-2.9	15.92	22.70	0.084

n : number, GCS: Glasgow Coma Score, AST: aspartate aminotransferase, ALT: alanine aminotransferase, *significant ($p < 0.05$).

DISCUSSION

Aluminum phosphide is a cheap, solid, and highly toxic pesticide that is commonly used for grain preservation. Acute AP poisoning is characterized by high risks of major morbidity and mortality (Yatendra et al., 2014). Therefore, there is an increasing need for improved knowledge of these risks with an emphasis on recognition, management, and prevention.

This study was undertaken to make use of readily available, easily obtained data from clinical examination and routine laboratory investigations in predicting risks of mortality in patients with acute AP poisoning. Early anticipation of these risks by the attending physician may help proper, early intervention and could minimize

high mortality rates in these cases. In this regard, the important prognostic factors in this study were; the route of intoxication, the manner of toxicity whether intentional, suicidal, or accidental, and the time interval between exposure and hospital admission, in addition to the presence of low GCS, low SBP, abnormal ECG findings, high serum cardiac troponin, metabolic acidosis, hypokalemia, hyperglycemia, and leukocytosis at the time of admission.

In this study, despite early administration of vasoactive drugs and standard supportive treatment 27 out of 40 patients died with an overall mortality rate of about 67.5%. The reported mortality rate of AP poisoning is highly variable, ranging from 37 to 100%, and can reach more than 60% even in experienced and well-equipped hospitals (Moghadamnia, 2012).

The route of intoxication showed a statistically significant association with mortality. Most (92.59%) non survivor exposure to AP was by oral ingestion. Six out of eight cases that reported exposure by inhalation while placing AP tablets on stored grains survived. Aluminum phosphide tablets on contact with air moisture or hydrochloric acid in the stomach release phosphine gas, which is then absorbed by the gastrointestinal and respiratory tracts (Valmas et al., 2008). The foul and irritating odor of phosphine gas could lead to short term exposure with subsequent poisoning with low severity and hence better outcome (Shadnia et al., 2008).

In this study, the majority (72.50%) of patients alleged intentional poisoning. The manner of toxicity whether intentional, suicidal, or accidental, showed a statistically significant association with outcome. Most (85.2%) non survivors alleged intentional exposure. Similarly, the use of AP to commit suicide was reported by other relevant studies (Ferrer et al., 2009). The increased use in agricultural purposes, wide availability, low cost, and free unrestricted use in the market may direct persons to use AP tablets for suicide (Mehrpour et al., 2012). The amount of the ingested poison, the severity of symptoms and the necessary treatment may differ substantially between unintentional and intentional poisonings (Lauterbach et al., 2005).

In this study, the time interval between exposure to AP and hospital admission showed a statistically significant association with mortality. The important role of providing supportive care early after exposure and its impact on improving survival of AP poisoning is emphasized also by Singh et al. (1997). However, reports from other studies (Nejad et al., 2012) found insignificant differences between improved and expired patients as regards time interval between the poison ingestion and start of medical interventions. The characteris-

tic fast progression to life threatening symptoms and the short time interval between ingestion of AP and death, may explain this conflicting finding (Yatendra et al., 2014).

Regarding clinical manifestations, our work revealed a statistically significant association between altered consciousness at the time of admission and outcome. In addition, the mean SBP and respiratory rate in survivors and non survivors were significantly different. Soltaninejad et al. (2012) concluded that SBP is a key risk factor that can be assessed at admission to the hospital to predict mortality from AP poisoning. The mechanism of shock in these cases is multifactorial. Clinical, biological, and electrical observations suggest that myocardial lesion is the main cause of hemodynamic failure (Siddaiah et al., 2009).

The current study showed significant differences between survivors and non-survivors regarding the presence of abnormal ECG findings. Furthermore, the presence of ST segment abnormalities, either elevation or depression was significantly associated with mortality and showed also positive correlation with it. This finding is in concordance with previous research work (Gupta and Ahlawat, 1995; Soltaninejad et al., 2012). However, an earlier study (Chugh et al., 1991) reported absence of association between ECG abnormalities and mortality in AP intoxicated cases. The pathogenesis of ECG abnormalities including cardiac dysrhythmia and ST changes can be explained by the action of phosphine gas on the heart. It causes inhibition of mitochondrial cytochrome c oxidase resulting in myocardial energy depletion similar to what occurs with ischemia (Singh et al., 2006). In addition, phosphine enhances the generation of reactive oxygen species causing lipid peroxidation with focal areas of myocardial necrosis (Akkaoui et al., 2007). These effects can cause alterations in cardiac transmembrane action potentials leading to dysrhythmia, and ischemia-like effect on ECG.

In the current study, a statistically significant association was present between serum cardiac troponin and outcome. Further, it showed a significant positive correlation with mortality. This finding is supported by Soltaninejad et al. (2012) who concluded that specific cardiac enzymes such as troponin are reliable prognostic markers of cardiac injury in these cases. This work revealed also that non-survivors of AP poisoning had a statistically significant tendency towards hyperglycemia when compared with survivors. This is supported by an earlier study (Mehrpour et al., 2008), which reported an increased risk of death in AP poisoning patients with glucose levels greater than 140 mg/dl at admission. Suggested mech-

anisms for this hyperglycemia include impaired oxidative phosphorylation and glucose utilization with involvement of the adrenal axis and/or pancreas (Singh et al., 2006; Verma et al., 2007). In contrast, hypoglycemia has been also reported following AP ingestion in other studies (Singh et al., 1994; Chugh et al., 2000).

In this study non-survivors have more metabolic acidosis and total leukocytic count than survivors. This finding is in accordance with (Louriz et al., 2009; Mathai and Bhanu, 2010) who concluded that the prognostic factors associated with mortality from AP poisoning included low serum pH and bicarbonate levels and high leukocytic count. All these effects of AP poisoning could be explained by the pathologic action of phosphine, which involves inhibition of cytochrome oxidase associated with free radical damage of tissues. Additionally, a statistically significant low potassium level was detected in non-survivor compared to survivor groups. This finding is supported by Kochar et al. (2000) who reported that successful management of hypokalemia and related conduction disorders saved a case of acute AP poisoning.

CONCLUSION

According to our results, it seems that AP is a remarkably toxic compound with a very high mortality rate. The variables at admission, which could be used to detect patients at greater risk of mortality from AP were hypotension, abnormal ECG findings, low blood pH, low serum bicarbonate, and hyperglycemia. Other alarming risk factors included suicidal ingestion, altered consciousness, high troponin level, hypokalemia, and leukocytosis. Because of unavailability of a specific antidote, early anticipation of these risk factors and prompt supportive care might improve outcome. In addition, efforts to set strict rules for AP use and sales are recommended. However, this research work was performed on a small sample size with regard to the long duration of the study. Further studies with larger sample size are encouraged.

REFERENCES

Akkaoui, M.; Achour, S.; Abidi, K.; et al. (2007): "Reversible myocardial injury associated with aluminum phosphide poisoning". Clin.Toxicol., 45: 728-731.

Bazett, H.C. (1920): "An analysis of the time-relations of electrocardiograms". Heart 7: 353-370.

Chugh, S.N.; Chugh, K.; Ram, S. and Malhotra, K.C. (1991): "Electrocardiographic abnormalities in aluminum phosphide poisoning with special reference to its incidence, pathogenesis, mortality and histopathology". J. Indian Med. Assoc.,

89:32-35.

Chugh, S. N.; Kishore, K.; Aggarwal, N. and Attri, S. (2000): "Hypoglycaemia in acute aluminium phosphide poisoning". J. Assoc. Physicians India, 48:855-856.

Crotti, L.; Celano,. G; Dagradi, F. and Schwartz, P.J. (2008): "Congenital long QT syndrome". Orphanet J. Rare Dis., 3: 18.

Ferrer, M. I.; Alvarez, L. F.; Cepero; R.A.; et al. (2009): "Suicide by ingestion of aluminum phosphide: a case report" Emergencies, 21:228–231.

Gupta, S. and Ahlawat, S.K. (1995): "Aluminum phosphide poisoning review". J. Toxicol. Clin. Toxicol., 33:19-24.

Henderson, W. (2004): "APACHE: An evaluation". Paedagogica Hist., 40: 435-453.

Katira, R.; Elhence, G. P.; Mehrotra, M. L.; et al. (1990): "A study of aluminum phosphide poisoning with special reference to electrocardiographic changes". J. Assoc. Physicians India, 38(7): 471-473.

Kochar, D. K.; Shubhakaran; Jain, N.; et al. (2000): "Successful management of hypokalaemia related conduction disturbances in acute aluminium phosphide poisoning". J. Indian Med. Assoc., 98 (8): 461-462.

Lauterbach, M.; Solak, E.; Kaes, J.; et al. (2005): "Epidemiology of hydrogen phosphide exposures in humans reported to the poison center in Mainz, Germany, 1983-2003". Clin. Toxicol. (Phila), 43 (6): 575-581.

Louriz, M.; Dendane, T.; Abidi, K.; et al. (2009): "Prognostic factors of acute aluminum phosphide poisoning". Indian J. Med. Sci., 63 (6): 227-234.

Mathai, A. and Bhanu, M.S. (2010): "Acute aluminium phosphide poisoning: Can we predict mortality"? Indian J. Anaesth., 54 (4): 302-307.

Mehrpour, O.; Alfred, S.; Shadnia, S.; et al. (2008): "Hyperglycemia in acute aluminum phosphide poisoning as a potential prognostic factor". Hum. Exp. Toxicol., 27 (7): 591-595.

Mehrpour, O.; Jafarzadeh, M. and Abdollahi, M. (2012): "A systematic review of aluminium phosphide poisoning". Arh. Hig. Rada. Toksikol., 63: 61-73.

Moghadamnia, A. A. (2012): "An update on toxicology of aluminum phosphide". Daru., 20(1): 25.

Nejad, F. T.; Mohammadi, A. B.; Behnoush, B.; et al. (2012): "Predictors of Poor Prognosis in Aluminum Phosphide Intoxication". Iranian J. Toxicol., 6 (16):

610-614.

Shadnia, S.; Mehrpour, O. and Abdollahi, M. (2008): "Unintentional poisoning by phosphine released from aluminum phosphide". Hum. Exp. Toxicol., 27: 87-89.

Siddaiah, L.; Adhyapak, S.; Jaydev, S.; et al. (2009): "Intra-aortic balloon pump in toxic myocarditis due to aluminum phosphide poisoning". J. Med .Toxicol., 5: 80-83.

Singh, B.; Gupta, S.; Minocha, S. K. and Aggarwal, N. M. (1994): "Hypoglycaemia in aluminium phosphide poisoning". J. Assoc. Physic. India, 42 (8): 663.

Singh, S.; Bhalla, A.; Verma, S. K.; et al. (2006): "Cytochrome-c oxidase inhibition in 26 aluminum phosphide poisoned patients". Clin. Toxicol. (Phila), 44 (2): 155-158.

Singh, S.; Singh, D.; Wig, N.; et al. (1996): "Aluminum phosphide ingestion--a clinico-pathologic study". J. Toxicol. Clin. Toxicol., 34 (6): 703-706.

Singh, U.K.; Chakraborty, B. and Prasad, R. (1997): "Aluminium phosphide poisoning: a growing concern in pediatric population". Indian Pediatr., 34 (7): 650-651.

Soltaninejad, K.; Beyranvand, M. R.; Momenzadeh, S. A. and Shadnia, S. (2012): "Electrocardiographic findings and cardiac manifestations in acute aluminum phosphide poisoning". J. Forensic Legal Med., 19: 291-293.

Valmas, N.; Zuryn, S. and Ebert, P.R. (2008): "Mitochondrial uncouplers act synergistically with the fumigant phosphine to disrupt mitochondrial membrane potential and cause cell death". Toxicol., 252: 33–39.

Verma, S. K.; Ahmad, S.; Shirazi, N.; et al. (2007): "Acute pancreatitis: a lesser-known complication of aluminum phosphide poisoning". Hum. Exp. Toxicol., 26 (12): 979-981.

Wahab, A.; Rabbani, M. U.; Wahab, S. and Khan, R. A. (2009): "Spontaneous self-ignition in a case of acute aluminium phosphide poisoning". Am. J. Emerg. Med., 27 (6): 752.e5-6.

Yatendra, S.; Subhash, C. J.; Vivekanand, S. and Abhisek, G. (2014): "Acute aluminium phosphide poisoning, what is new"? Egypt. Soc. Int. Med., 26: 99–103.

مؤشرات التنبؤ بالمسار المرضي في التسمم الحاد بفوسفيد الألومنيوم

المشتركون فى البحث

أحمد عبد الستار الابيارى فاطمة محمد الجزار
محمد عبد الرحيم سليمان* أسامه ممدوح شعيب**

من قسم الطب الشرعي والسموم الإكلينيكية – جامعة طنطا وقسم الباطنة العامة – مستشفى النهضة – مسقط – سلطنة عمان*
وقسم القلب والأوعية الدموية كلية الطب – جامعة طنطا**

يعد فوسفيد الألومنيوم من المبيدات شديدة السمية التي تستخدم عادة لحفظ الحبوب،ويمثل التسمم الحاد بفوسفيد الألمونيوم مشكلة صحية عامة خطيرة وتحديا حقيقيا للأطباء في البلدان النامية بسبب المعدلات العالية لحدوث المضاعفات والوفيات، ولقد أجريت هذه الدراسة لتحديد عوامل التنبؤ التي قد تكون مفيدة في تحديد المسار المرضى لحالات التسمم الحاد بفوسفيد الألومونيوم، وقد أدرجت جميع حالات التسمم بفوسفيد الألومنيوم التي أحيلت إلى مركز علاج التسمم بجامعة طنطا خلال الفترة من شهر مارس ٢٠١٣ إلى فبراير ٢٠١٥، ولقد تم أخذ التاريخ المرضى بما فى ذلك البيانات الديموجرافية، وتم تسجيل نتائج الفحص السريرى للمرضى، كما سجلت نتائج الرسم الكهربي للقلب، ومستوى التروبونين في الدم، وغازات الدم بالإضافة إلى صورة الدم الكاملة، حيث تم تقييم علاقتها ومدى ارتباطها بالمصير المرضى للمرضى محل الدراسة، ولقد أدرجت أربعون حالة (منهم ١١ من الذكور) من حالات التسمم الحاد بفوسفيد الألومنيوم في هذه الدراسة، وكان معدل الوفيات ٦٧,٥٪ ولقد تبين وجود ارتباط ذي دلالة إحصائية بين كل من اختلال الرسم الكهربي للقلب، وارتفاع مستوى التروبونين في الدم، وانخفاض درجة الحموضة ومستوى البيكربونات وإرتفاع الجلوكوز بالدم وحدوث الوفاة، وكذلك تبين وجود عوامل أخرى تنذر بخطورة الحالة مثل التسمم الانتحاري، ووجود تغير بدرجة الوعي، ونقص البوتاسيوم بالدم، وزيادة عدد الكريات البيضاء، ويمكن أن نخلص إلى أن المرضى الذين يعانون من واحد أو أكثر من هذه المتغيرات هم أكثر عرضة للوفاة، كما أن توقع هذه المخاطر من قبل الطبيب المعالج في وقت مبكر ومن ثم التدخل السليم ربما يقلل الوفيات ويحسن من نتائج هذه الحالات.