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Fatma Shaban Kandeel

Safaa Abdelzaher Amin

Ahmad Abdelsattar El-Ebiary

Ahmed Kamal Elfeky

Soha Hamid Abd El-khalek

ORIGINAL STUDY

Predictors of Acute Aluminum Phosphide Intoxication Outcome

Fatma S. Kandeel [a], Safaa A. Amin [a], Ahmad A. El-Ebiary [b], Ahmed K. Elfeky [a], Soha H.A. El-khalek [a,*]

[a] Department of Forensic Medicine, Faculty of Medicine, Menoufia University, Shebin El-Kom, Menoufia, Egypt
[b] Department of Forensic Medicine, Faculty of Medicine, Tanta University, Tanta, Gharbia, Egypt

Abstract

Objectives: **To evaluate the predictive factors of acute aluminum phosphide (AlP) poisoning outcome.**

Background: **Acute toxicity with the lethal aluminum phosphide has become a major and frequent problem in many poison control centers.**

Methods: **It is a prospective cohort study that was conducted over 1 year on acute AlP intoxicated patients. The patients were allocated into two groups; died and survived. The socio-demographic, toxicological data, and clinical evaluation were reported for each patient. Patients were subjected to laboratory investigations, electrocardiogram (ECG), echocardiogram (Echo), and oxidative biomarkers evaluation. All data were studied to estimate their associations with the patients' outcomes. A three-point prognostic scoring system for acute AlP was used as a predictive value in determining differences between dead and survived patients.**

Results: **The total number of cases was 96 patients who fulfilled the inclusion criteria, 18 patients died and 78 survived. There were no significant differences between the survived and dead groups regarding the following variables: age, manner and route of exposure, gastrointestinal manifestations, baseline liver enzymes, serum electrolytes, and oxidative stress biomarkers levels. Lowered Glasgow Coma Scale (score <13), decreased systolic and diastolic blood pressure, tachycardia, and tachypnea, had significant associations with mortality (all $P < 0.001$). Metabolic acidosis was a significant predictive laboratory finding for outcome ($P < 0.001$). ECG arrhythmias and decreased ejection fraction were significantly reported in the dead group ($P < 0.001$).**

Conclusion: **Being aware of these risk factors of mortality is helpful in early, proper intervention and improving the AlP poisoning outcome.**

Keywords: Acute aluminum phosphide, Aluminum phosphide outcomes, Aluminum phosphide, Intoxication outcome, Prognostic factors

1. Introduction

Aluminum phosphide (AlP) intoxication is a widespread and rising public health challenge. This is because the AlP tablet is inexpensive, broadly available, and has no definite antidote with a high mortality rate [1]. With the easy availability of AlP tablet as a household rodenticide and knowing its lethal action, it has become one of the commonly used agents for self-poisoning [2]. Cytotoxic phosphine gas released from AlP tablets after contact with water or gastric acid is rapidly distributed in systemic circulation [3]. It causes oxidative stress due to the inhibition of 70% oxidative phosphorylation by blocking mitochondrial cytochrome C oxidase and reducing glutathione with subsequent lipid peroxidation [4]. Symptoms of toxicity appear within minutes of exposure, starting with gastrointestinal manifestation, then circulatory collapse, acidosis, agitation, multi-organ failure, and death within 24−48 h [5,6]. Different studies evaluated the magnitude of AlP poisoning in their communities

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* Corresponding author at: Shebin Elkom, Menoufia, 32511, Egypt.
E-mail address: d_sh_2007@yahoo.com (S.H.A. El-khalek).

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both in Egypt [7,8] and in several African and Asian countries, which found that the frequency of acute AlP poisoning is increasing every year with a high fatality rate [5,9,10]. Predicting the outcomes of AlP-intoxicated patients is required for appropriate triaging of the patients, guiding clinical decision-making, and evaluation of therapeutic interventions [11].

This study aimed to evaluate the personal, toxicological, clinical, and laboratory variables that predict the outcome and mortality of AlP-poisoned patients.

2. Methods

This is a prospective cohort study conducted on patients with acute AlP poisoning who arrived at Menoufia Poison Control and Dependence Center, between September 2020 and October 2021. Ethical approval was obtained from the Ethics Committee of the Faculty of Medicine, Menoufia University, Egypt (IRB no.: 19519FORE19). Informed written consents were collected from the patients or their guardians. Eligibility criteria were patients greater than or equal to 12 years of both sexes, with a diagnosis of acute AlP poisoning. Diagnosis was established by a history of exposure to well-identified AlP tablets either by container or its typical odor and known clinical presentation. Silver nitrate test was used to confirm in patients with oral intake. Exclusion criteria: Patients aged less than 12 years old, lactating and pregnant females, patients who suffered from chronic diseases, patients who ingested air-exposed AlP tablets or co-ingestion of other drugs or poisons, patients presented more than 8 h after ingestion or immediately died after arrival.

Full history was taken for all patients with emphasis on gender, age, manner of poisoning (suicidal or accidental), route and amount of exposure prehospital period, and referral data. A complete physical examination was done for all participants, including general examination, evaluation of consciousness level by Glasgow coma score coma scale (GCS), and vital signs. Blood samples were collected for routine laboratory investigation, including arterial blood gases, serum electrolytes (serum sodium, potassium, and magnesium), serum glutamic pyruvic transaminase (SGPT), serum glutamic-oxaloacetic transaminase levels (SGOT), and blood urea and serum creatinine. Echocardiogram and electrocardiogram were evaluated.

Oxidative stress biomarkers were estimated at the time of arrival, including malondialdehyde (MDA) and total antioxidant capacity (TAC), using commercial kits supplied by Biodiagnostics Corp., Giza, Egypt. Patients were classified using a three-point scoring system for low pH (P) less than 7.25, low GCS score less than 13 (G), and impaired or low systolic blood pressure (SBP) less than 87 mm Hg (I), known as PGI score. Each of the three predictors was given a score value of 1 and a maximum score of three [12].

All Patients were divided into two groups according to the main outcome either died or survived.

2.1. Statistical analysis

The collected data were statistically analyzed using the Statistical Package for Social Sciences (SPSS), version 22 (Armonk, NY: IBM Corp, 2013). Qualitative data were expressed as numbers and percentages and were compared using the χ^2 test. Quantitative data were expressed as means and standard deviations or ranges, and they were compared using the Student *t*-test or Mann–Whitney test as appropriate. A *P* value less than 0.05 was considered statistically significant.

Sample size was calculated using the G power 3.1.9.4 software program using the study of El-Sarnagawy (2017) [4] confidence interval of 95% and power of study of 80% to be a minimum 96%.

3. Results

There was an increasing rate of AlP poisoning cases at MPCC in the years 2017, 2018, 2019, and 2020 (3.3, 5.8, 9.9, and 23.5%, respectively) of total poisoned cases.

According to morbidity and mortality, committee reports of Menoufia University Hospital, Aluminum phosphide dead cases represent 67, 83, 83, and 89% of total deaths in the years 2017, 2018, 2019, and 2020 (Fig. 1).

The present study revealed that there were 1326 poisoned patients admitted to the center with 159 (12%) deaths in over a year 2020. Out of all cases, 311 acute AlP-poisoned patients were assessed for eligibility with 142 (54.7%) deaths.

The 96 patients were included in the study and divided regarding the primary outcome into two groups, died (18 patients) and survived (78 patients).

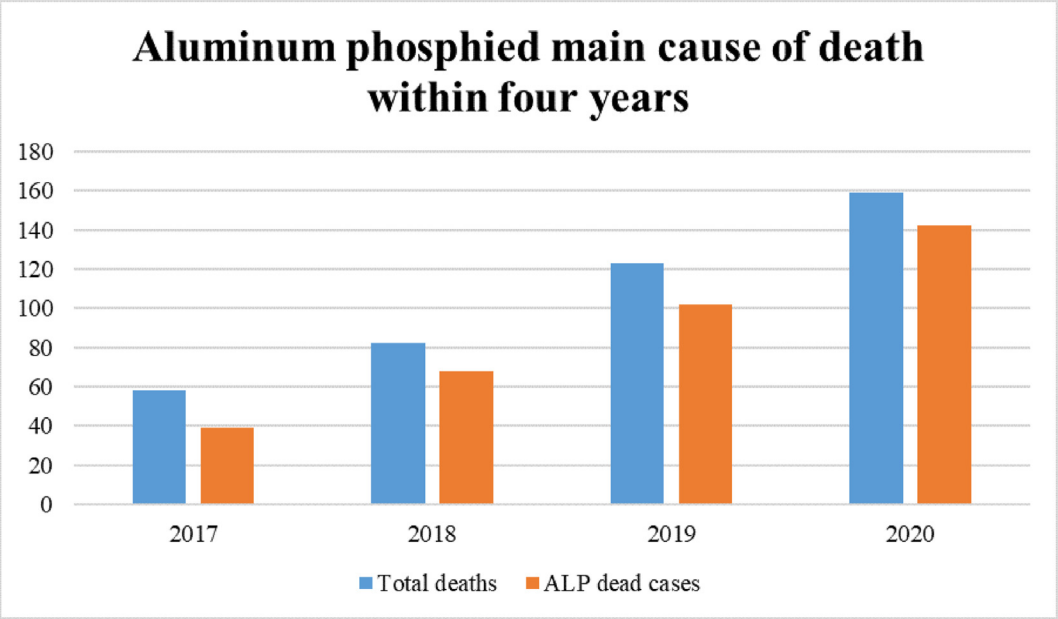

Fig. 1. Number of deaths from aluminum phosphide in relation to total deaths of acute toxicity within 4 years at MPCC Menoufia University Hospital. ALP, aluminum phosphide.

In the present study, the mean age of acute aluminum phosphide-poisoned patients was 23.6 ± 9.7 years. Most patients committed suicide (91.7%) and 52.1% were females.

We found no statistically significant association between the outcome and patients age, manner, and route of exposure. However, sex shows a significant difference between the two groups. There was a statistically significant association between amount of phosphide and mortality ($P = 0.043$), there was (88.9%) of nonsurvival ingested more than one AlP tablet. Regarding received treatment outside hospital, intake of AlP with water had a significant association with poor patients' outcome in our study ($P < 0.001$). In contrast, patients who received gastric lavage with paraffin oil survived. There was a statistically significant difference between both groups regarding the mean of prehospital period, it was longer in dead cases ($P = 0.05$) (Table 1).

The died group showed significantly lower conscious level (GCS < 13), SBP and diastolic blood pressure (DBP) measurement, but higher pulse and respiratory rate compared with survived group (all $P < 0.001$). There was no significant association could be observed between GIT manifestations and patients' outcome. Regarding PGI score, 61.1% of dead patients had a score value 3 (bad prognostic factor for mortality). Meanwhile, 76.9% of survivors had a score of 0 and the difference was significant ($P < 0.001$) (Table 2).

There were no significant differences between the two groups regarding their liver enzymes, serum electrolytes and oxidative stress (serum MDA and TAC) parameters, while there were significantly higher mean serum creatinine and blood urea levels, and lower serum PH and HCO_3 in non-survivors compared with the survivors (all $P < 0.05$). There was a significant decrease the mean values of partial oxygen pressure (PO_2) and oxygen saturation (SO_2) in non survivors in comparison to survivors ($P < 0.001$ and 0.002, respectively) (Table 3).

Regarding ECG changes, there were significantly higher percentages of dead cases who had arrhythmia, whether sinus tachycardia (44.4%) or other types (55.6%) as atrial fibrillation (AF) (Fig. 2).

ST-segment changes, compared with survived cases who had 33.4% and 5.1%, respectively ($P < 0.001$). There was a significant association of ejection fraction and outcome, there was 88.9% of dead cases, had decreased ejection fraction compared with 6.4% of survived cases ($P < 0.001$) (Table 4).

Table 1. The association between personal and toxicological data, and patients' outcome.

Variables	Died ($n = 18$)	Survived ($n = 78$)	Total ($n = 96$)	Test of significance	P value
Age, years, mean ± SD	27.4 ± 15.5	22.9 ± 7.5	23.6 ± 9.7	$U = 0.64$	0.637
Sex, n (%)					
Males	4 (22.2)	42 (53.8)	46 (47.9)	$\chi^2 = 5.86$	0.015[a]
Females	14 (77.8)	36 (46.2)	50 (52.1)		
Manner of poisoning, n (%)					
Suicidal	18 (100.0)	70 (89.7)	48 (91.7)	$\chi^2 = 2.01$	0.156
Accidental	0	8 (10.3)	8 (8.3)		
Route of exposure, n (%)					
Ingestion	18 (100.0)	76 (97.4)	94 (97.9)	$\chi^2 = 0.47$	0.492
Inhalation	0	2 (2.6)	2 (2.1)		
Amount, tablet, n (%)					
1/4	2 (11.1)	10 (12.8)	12 (12.5)	$\chi^2 = 6.291$	0.043[a]
1/2	0	20 (25.6)	20 (20.8)		
≥ 1	16 (88.9)	48 (61.5)	62 (64.6)		
Intake with water, n (%)					
Yes	18 (100.0)	34 (43.6)	44 (45.8)	$\chi^2 = 18.75$	<0.001[a]
No	0	44 (56.4)	52 (54.2)		
Referral, n (%)					
Yes	6 (33.3)	28 (35.9)	34 (35.4)	$\chi^2 = 0.04$	0.838
No	12 (66.7)	50 (64.1)	62 (64.6)		
Treatment received outside, n (%)					
No	14 (77.8)	52 (66.7)	66 (68.8)	$\chi^2 = 10.47$	0.005[a]
Gastric lavage	4 (22.2)	4 (5.1)	8 (8.3)		
Gastric lavage with paraffin oil	0	22 (28.2)	22 (22.9)		
Pre-hospital period, hours					
Mean ± SD	2.61 ± 1.43	2.13 ± 2.44	2.2 ± 2.3	$U = 1.96$	0.05[a]

n, number; SD, standard deviation; U, Mann–Whitney test; χ^2 $_{Ch's}$, Pearson's Chi square test for association.
[a] P value of less than 0.05 was statistically significant.

Table 2. Clinical predictive factors for mortality.

Clinical data	Died ($n = 18$)	Survived ($n = 78$)	Test of significance	P value
Consciousness level, n (%)				
Conscious	6 (33.3)	72 (92.3)	$\chi^2 = 33.39$	<0.001[a]
Grade 1-drawzy	12 (66.7)	6 (7.7)		
GCS, n (%)				
<13	14 (77.8)	14 (17.9)	$\chi^2 = 25.34$	<0.001[a]
15	4 (22.2)	64 (82.1)		
Pulse, beat/min, mean ± SD	121.67 ± 5.94	98.92 ± 14.35	$t = 6.57$	<0.001[a]
Systolic blood pressure				
Mean ± SD	78.89 ± 6.76	102.05 ± 9.58	$t = 9.69$	<0.001[a]
Diastolic blood pressure				
Mean ± SD	55.56 ± 7.05	70.64 ± 7.99	$t = 7.37$	<0.001[a]
Respiratory rate				
Mean ± SD	22.67 ± 4.58	18.38 ± 3.37	$t = 4.52$	<0.001[a]
PGI score, n (%)				
0	0	60 (76.9)	$\chi^2 = 66.66$	<0.001[a]
1	3 (16.7)	14 (17.9)		
2	4 (22.2)	4 (5.1)		
3	11 (61.1)	0		
Vomiting, n (%)				
No	6 (12.5)	6 (12.5)	–	–
Yes	42 (87.5)	42 (87.5)		
Abdominal pain, n (%)				
No	8 (16.7)	10 (20.8)	$\chi^2 = 0.27$	0.601
Yes	40 (83.3)	38 (79.2)		
Temperature, C°, n (%)				
Mean ± SD	35.23 ± 0.51	36.74 ± 0.60	$t = 3.28$	0.001[a]

GCS, Glassco coma scale; PGI score, low PH less than 7.25 low GCS less than 13, and Impaired SBP less than 87 mm Hg Simplified predictive score for AlP poisoning mortality (Pannu et al., 2020); t, Student's t-test; U, Mann–Whitney test; χ^2 $_{Ch's}$, Pearson's Chi square test for association.
[a] P value of less than 0.001 was statistically significant.

Table 3. Baseline laboratory values and their association with patients' outcome.

Laboratory data	Died ($n = 18$) mean ± SD	Survived ($n = 78$) mean ± SD	Test of significance	P value
SGPT, U/l	21.44 ± 6.20	21.49 ± 8.66	$U = 0.23$	0.821
SGOT, U/l	28.78 ± 16.07	28.72 ± 13.60	$U = 0.68$	0.498
INR	1.04 ± 0.05	1.07 ± 0.14	$t = 0.94$	0.350
Creatinine, mg/dl	0.99 ± 0.28	0.81 ± 0.26	$t = 2.59$	0.011[a]
Urea, mg/dl	27.78 ± 11.02	22.85 ± 7.38	$U = 2.44$	0.015[a]
Serum sodium, mmol/l	140.02 ± 5.93	139.51 ± 4.23	$t = 0.43$	0.672
Serum potassium, mmol/l	4.12 ± 0.63	4.20 ± 0.66	$t = 0.46$	0.650
Serum magnesium, mmol/l	2.47 ± 0.58	2.35 ± 0.52	$t = 0.86$	0.393
PH	7.08 ± 0.16	7.36 ± 0.06	$t = 12.26$	<0.001[a]
PCO_2, mmHg	34.89 ± 8.50	36.37 ± 5.21	$t = 0.95$	0.344
PO_2, mmHg	43.24 ± 19.71	62.32 ± 15.85	$U = 4.27$	<0.001[a]
SO_2, %	65.11 ± 30.04	87.72 ± 8.56	$U = 3.16$	0.002[a]
HCO_3, mmol/l	11.73 ± 3.75	18.77 ± 3.42	$t = 7.73$	<0.001[a]
Silver nitrate test:	N (%)	N (%)		
Positive	18 (100.0)	58 (74.4)	$\chi^2 = 5.83$	0.015[a]
Negative	0	20 (25.6)		
MDA, nmol/ml	14.74 ± 12.13	12.19 ± 7.94	$U = 0.46$	0.499
TAC, mM/l	0.58 ± 0.11	0.59 ± 0.16	$t = 0.21$	0.836

SD: standard deviation; U = Mann–Whitney test; t: Student's t-test; MDA: malondialdehyde; TAC: total antioxidant capacity; χ^2 Ch's: Pearson's Chi square test for association.
[a] P value of less than 0.05 was statistically significant.

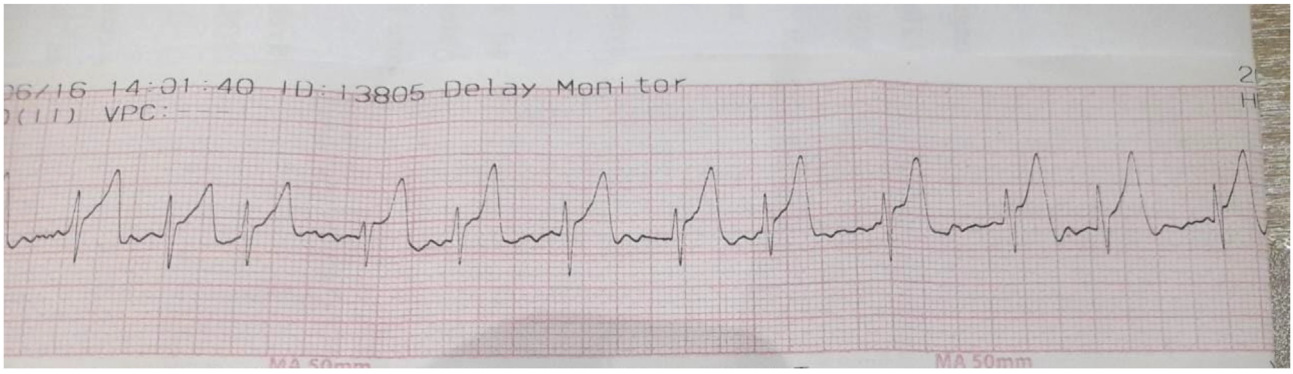

Fig. 2. ECG shows atrial fibrillation: heart rate 180/min for female patient 24 years old suicidal ingestion of aluminum phosphide tablet.

Table 4. Electrocardiogram and echocardiogram findings as predictive factors for mortality.

Investigations	Died ($n = 18$)	Survived ($n = 78$)	χ^2	P value
ECG, n (%)				
Normal	0	48 (61.5)	37.1	<0.001[a]
Sinus tachycardia	8 (44.4)	26 (33.4)		
Other arrhythmia	10 (55.6)	4 (5.1)		
EF/Echo, n (%)				
Normal, 50–70%	1 (5.55)	66 (84.6)	58.8	<0.001[a]
Borderline, 41–49%	1 (5.55)	7 (9.0)		
Decreased, ≤40%	16 (88.9)	5 (6.4)		

ECG, Electrocardiogram; Echo: Echocardiogram; EF, Ejection Fraction; χ^2_{ChS}, Pearson's Chi square test for association.
[a] P value of less than 0.001 was highly statistically significant.

4. Discussion

AlP is a fumigant rodenticide that has a high fatality rate between 30 and 100% in cases of acute poisoning [13].

On reviewing the magnitude of acute intoxication with AlP for 4 years from 2017 to 2020, there was a steadily increasing arrival and death percentage. So, we were in need of sensitizing on evaluate the prognostic factors for death as a step to improve management and patients' outcomes.

In the present study, the mean age of acute aluminum phosphide-poisoned patients was 23.6 ± 9.7 years. Patients at this age are more

susceptible to socioeconomic stress and depression so become more vulnerable to committing suicide [14].

Most of the studied cases were self-poisoned (91.7%) and 52.1% of them were females. This was parallel to the study of El-Sarnagawy [7] which explained that females are more likely to commit suicide due to psychological issues in their families, as well as emotional and educational challenges. Same results were shown in other studies that most acute AlP-poisoned patients were between 20 and 30 years but with dominance of males [15].

The current study demonstrated that 88.9% of dead patients had ingested one or more AlP tablets. This finding was parallel to studies, which reported that nearly half of nonsurvivors of AlP poisoning ingested greater than 1 tablet [16,17]. There was a significant association between the ingested amount and mortality, due to the high dose of liberated toxic phosphine gas. The available form of AlP tablet is 3 gm, which releases 1 gm of phosphine gas on exposure to moisture, which is 10 times the lethal dose for adults [18]. In the current study, intake of water with AlP tablet and absence of prehospital first aid management had a significant association with poor patients' outcomes. As water increases phosphine release and absorption causes multi-organ damage and death. This result agrees with El-Sarnagawy [7].

The studied patients who received gastric lavage with paraffin oil were survivors. So, early decontamination by paraffin oil can limit the liberation of phosphine with subsequent better prognosis. This agreed with the study of Abdelkader *et al.* [19], who stated that there was a significant increase in the percentage of surviving patients decontaminated by paraffin oil compared with other methods.

The more time elapsed without appropriate treatment, the more absorption of phosphine and result in rapid onset of toxicity symptoms and complications [20]. This finding can explain the significant association of bad prognosis of poisoned patients with delayed presentation and intervention, in the current study. Other studies revealed similar results [17,21].

In the current study, most of the nonsurvivors had lower GCS (77.8%) in comparison to survivors (17.9%). Altered consciousness level in AlP intoxicated patients is only a terminal event [22]. Patients remain mentally clear till cerebral anoxia due to shock, which results in drowsiness, delirium, and coma [23]. This is parallel to the study of Sheta *et al.* [17]. So, physicians should consider the low consciousness level of poisoned patients as a bad sign of prognosis.

There was a highly significant association in the current study between patients' outcome and clinical parameters as: pulse, blood pressure, respiratory rate, and temperature. This is in harmony with the results of studies evaluating the predictive factors of mortality of acute AlP poisoning as well [7,17].

Hypotension and tachycardia, which are resistant to fluid therapy and inotropes, were mainly detected in died group in the current study, explained by AlP-induced toxic myocarditis that eventually cause death [13]. Tachypnea was a common presentation among non-survivors (88.9%) and could be a compensatory response to metabolic acidosis [24].

There was a significant difference between died and survived groups regarding PGI score, where most of the dead cases had the worst score (score 3). This agrees with the study of Pannu *et al.* [12] that reported 96.4% died patients had a score of 3. So, these parameters (GCS, SBP, and PH) are essential predictors of mortality rate in acute AlP poisoning [25].

There was a statistically significant association between metabolic acidosis and mortality as present study revealed that all the non-survivors developed lower pH and HCO_3 values, acidosis leads to depression of myocardium contractility and aid in the progression of shock and so increases the mortality rate. This finding was in accordance with other studies [7,17].

The current study revealed that hypoxia is significantly associated with a high mortality rate. Phosphine gas inhibits mitochondrial oxidative respiration, resulting in histotoxic hypoxia and organ damage [4]. Same finding was reported in Abd Elghany *et al.* study [26].

There was a significantly lower level of creatinine among dead cases, which was attributed to the presence of shock accompanied by certain factors such as disseminated intravascular coagulation and acute tubular necrosis, which can eventually result in renal failure and death [24].

Regarding oxidative stress biomarkers (MDA and TAC) in the current study, at the time of presentation, the mean serum MDA level (14.74 ± 12.13 nmol/ml) for dead cases was higher than survived ones (12.19 ± 7.94 nmol/ml), without significant difference. This was in contrast with the study of Emam *et al.*, which studied larger sample size and concluded that both blood MDA and TAC were predictors of poor outcomes [27].

The current study revealed a significant association between mortality and abnormal ECG changes. More than 50% of nonsurvivors had AF and ST-segment changes that would cause circulatory

collapse and death. These changes could be attributed to oxidative stress of phosphine in myocardial tissue with subsequent focal necrosis and ischemic-like effects on ECG, this is because generation of oxygen free radicals and electrolyte disturbances [28]. This agreed with the findings of other studies [7,17].

Analysis of Echo findings in the present study demonstrated that most of the dead cases had decreased ejection fraction due to toxic myocarditis, cardiac impairment, and arrest. So, the Echo study was a good predictor of mortality for acute AlP-poisoned cases. The same results were found in Elgazzar *et al.*, a study which stated that echocardiographic abnormalities were detected in most non-survivors and echocardiography was a valuable diagnostic tool to assess cardiac function and was superior to ECG changes in terms of accuracy for the prediction of mortality [29].

4.1. Conclusion and recommendations

The mortality incidence of acute AlP poisoning is increasing steadily in our locality. Therefore, there is an increasing need for recognition of predictive factors of mortality to improve patients' outcomes. At admission, altered mental state, shock, presence of abnormal ECG, decreased EF, elevated urea and creatinine levels, metabolic acidosis, and hypoxia could be used as alarming predictive factors of mortality. Early management of these risk factors via intensive hemodynamic monitoring and proper coordination between clinical toxicologists, cardiologists, and ICU physicians for effective management is essential. Rapid referral to poison control centers is recommended to decrease the prehospital period. Emergency stabilization and early GIT decontamination with paraffin oil should be done with a documented complete referral report.

Ethics information

None.

Finding

None.

Conflicts of interest

There are no conflicts of interest.

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