Toxicology Reports 14 (2025) 101947

Contents lists available at ScienceDirect

Toxicology Reports

journal homepage: www.elsevier.com/locate/toxrep

Role of serum glucose/potassium ratio in assessing poisoning severity and adverse outcomes in patients with acute aluminum phosphide poisoning

Ghada N. El-Sarnagawy [1,2,*], Amal S.A.F. Hafez [2,3], Mona M. Ghonem [2,4]

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

ARTICLE INFO

Handling Editor: Prof. L.H. Lash

Keywords:
Aluminum phosphide poisoning
Outcomes
Mortality
Intensive care unit admission
Mechanical ventilation

ABSTRACT

Aluminum phosphide (ALP) poisoning is a crucial health problem owing to its easy availability, extreme potency, and absence of specific treatment modalities. This study aimed to assess the role of the glucose/potassium (Glu-K) ratio in predicting the severity and adverse outcomes of acute ALP poisoning. The current retrospective cohort study involved patients with acute ALP poisoning who were admitted to Tanta University Poison Control Center from June 2022 to June 2024. Sociodemographic and poisoning data, initial findings of clinical examination, Glu-K ratio, and calculation of Poisoning Severity Score (PSS), Acute Physiology and Chronic Health Evaluation II (APACHE II), and PGI score were documented. Patients were categorized into two groups according to mortality outcome. Out of 206 acute ALP-poisoned patients, we recorded 67.5 % fatalities. The median value of the Glu-K ratio was significantly higher in nonsurvivors than in survivors (44.8 versus 28.9; $p < 0.001$). The Glu-K positively correlated with PSS, APACHE II, and PGI scores ($p < 0.001$). The APACHE II score exhibited the highest performance for predicting mortality and the need for mechanical ventilation (AUC=0.876 and 0.853, respectively). *However*, the Glu-K ratio conveyed a comparable discriminatory power with other scoring systems (PSS and PGI) for anticipating all unfavorable outcomes. Patients with a Glu-K ratio ≥ 37.07 had significantly decreased survival duration than patients with a Glu-K ratio < 37.07 (0.38 *versus* 3 days; $p < 0.001$). Therefore, the initial Glu-K ratio is an easily accessible routine biomarker for assessing poisoning severity and outcomes probability of ALP-poisoned patients, particularly with limited healthcare facilities.

1. Introduction

Aluminum phosphide (ALP) is a commonly encountered pesticide known as a "rice tablet" or "wheat pill" and is used in agriculture and household settings in many developing countries [1]. Although it possesses the ideal properties for storing grains, it is considered one of the leading causes of poisoning deaths worldwide [2]. Accordingly, fatal outcomes range between 30 % and 100 %, even in highly specialized hospitals with advanced life support measures [3]. In Egypt, considerable rates of ALP poisoning with high mortality rates were observed in various poison centers [3–5].

The cardinal ALP toxicity mechanism is based on phosphine gas either by direct inhalation or liberation from the ingested ALP tablet after reacting with gastric hydrochloric acid [6]. At the cellular level, phosphine gas inhibits the mitochondrial cytochrome C oxidase enzymes, causing cellular energy depletion and tissue hypoxia [7]. Additionally, it induces oxidative stress by increasing the production of peroxide-free radicals and decreasing glutathione storage, causing multiorgan lipid peroxidation and protein denaturation [8].

Upon ALP exposure, the initial non-specific mild manifestations include nausea, vomiting, abdominal pain, dyspnea, and anxiety [9]. However, patients rapidly deteriorate over time, presenting with severe hemodynamic instability and respiratory failure, altered mental status arrhythmias, and cardiac arrest [10]. Death eventually occurs within the

* Corresponding author.
E-mail addresses: ghada.mahmoud1@med.tanta.edu.eg (G.N. El-Sarnagawy), amal.hafez@med.tanta.edu.eg (A.S.A.F. Hafez), mona.ghonaim@med.tanta.edu.eg (M.M. Ghonem).
[1] ORCID ID: 0000–0003-1671–0894
[2] Present/permanent address: Department of Forensic Medicine and Clinical Toxicology – 6th floor –Faculty of Medicine, Tanta University- Medical colleges complex – Al-Geish Street – Tanta, Gharbia, Egypt. Postal code: 31527.
[3] ORCID ID: 0000–0002-7463–8416
[4] ORCID ID: 0000–0003-1357–0777

https://doi.org/10.1016/j.toxrep.2025.101947
Received 27 November 2024; Received in revised form 30 January 2025; Accepted 31 January 2025
Available online 4 February 2025

first 48 hours due to primarily cardiac compromise, as well as multi-organ failure [11].

As a result of the absence of specific antidotes, the treatment strategies depend on intensive supportive measures for circulatory maintenance and administration of available antioxidants [12]. Consequently, many researchers are still searching for instantaneous outcome predictors to prompt emergent interventions for high-risk patients, especially in limited hospital settings.

The literature revealed considerable interest in identifying prognostic factors for acute ALP poisoning adverse outcomes, including different clinical and laboratory parameters, and validating conventional or proposed scoring systems [13–18]. Although the integration of more than one variable provides a tremendous predictive capacity, the time-consuming measurement and calculation hampered the prompt decision-making of ALP-poisoned patients at high risk for poor outcomes.

Clinically, serum glucose and potassium are routine blood biomarkers of metabolic status [19]. Substantially, glucose is a chief cellular energy source for performing essential functions [20]. Likewise, potassium is considered one of the crucial minerals for regulating heartbeats, muscle contractions, and neuronal conduction, as well as preserving the acid-base status and renal function stability [21].

Accordingly, the combined glucose-potassium (Glu-K) ratio has been previously investigated as a significant predictor of morbidity and mortality in many medical conditions, including pulmonary embolism [22], aneurysmal subarachnoid hemorrhage [23], thoracic trauma [24], acute traumatic spinal cord injury [25], and traumatic brain injury [26]. In clinical toxicology contexts, prior studies have demonstrated the correlation between the Glu-K ratio and the mushroom poisoning severity [27], as well as its prognostic significance in identifying intermediate syndrome in cholinesterase inhibitors poisoning [28], life-threatening events in acute theophylline poisoning [29] and delayed neurological sequels following carbon monoxide poisoning [30].

Since the relevance of the Glu-K ratio in acute ALP poisoning is still not defined, our goal was to find out the role of the Glu-K ratio in assessing poisoning severity and predicting outcomes, underscoring its correlation with previously validated predictive scoring systems such as Poisoning Severity Score (PSS), Acute Physiology and Chronic Health Evaluation II (APACHE II), and PGI score.

2. Patients and methods

2.1. Study design and setting

The current study was a two-year retrospective cohort study conducted on the medical records of ALP-poisoned patients admitted to Tanta University Poison Control Center (TUPCC) - Tanta Emergency Hospitals - Egypt, during the period between June 2022 and June 2024. The TUPCC was the only tertiary poison center in the middle of the delta, serving all countries in the Gharbia governorate.

2.2. Sample size calculation

The sample size was computed by the R Statistical language (version 4.4.1; R Core Team, 2024) using the package pROC (version 1.18.5; Robin X et al., 2011)[31]. One receiver operating characteristics (ROC) curve power calculation was performed, assuming an AUC of 0.7 or higher, an alpha level of 0.01, a power of 90 %, and a ratio of control to cases of 0.43 (3/7). The ratio was based on the incidence of mortality in AlP (70 % or more), as Anand et al. (2011)[32] reported. The minimal number was 162 cases. However, convenience sampling allowed us to recruit 206 patients who fulfilled the eligibility criteria to overcome incomplete and missing data.

2.3. Ethical consideration

This study was carried out according to the 1964 Declaration of Helsinki guidelines after receiving approval from Tanta University's Faculty of Medicine's Research Ethical Committee (REC) (approved number: 36264PR918/10/24). Due to the retrospective study design, the necessity of acquiring written informed permission from the patients was exempted. All patient data were managed anonymously, adhering to personal and clinical information confidentiality.

2.4. Eligibility criteria

2.4.1. Inclusion criteria

All acute ALP phosphide-poisoned patients over 18 years of both sexes were included in this study. The diagnostic criteria for enrolment entailed the history of exposure, the requested pesticide containers if available, suggestive clinical features, and the distinctive garlic odor of the breath. The diagnosis was endorsed by detecting phosphonic gas in the gastric aspirate using a silver nitrate test.

2.4.2. Exclusion criteria

The current study ruled out patients with comorbidities like diabetes, cardiovascular disorders, and hepatic or renal diseases, as well as those with a co-exposure history to other pharmaceutical and non-pharmaceutical preparations. We did not include patients with incomplete data or unknown poisoning, asymptomatic patients during the in-hospital follow-up period, and patients who were deceased on hospital arrival. Additionally, we excluded all patients taking drugs affecting serum glucose or potassium levels and those who received any medical intervention before admission.

2.5. Data collection

Data related to patients who fulfilled the eligibility criteria were gathered into a specially designed sheet, including socio-demographics (age, sex, and residence), as well as poisoning data (mode and route of poisoning, the amount of ingested AlP, and the delay time since exposure till admission). On admission findings of clinical assessment including Glasgow Coma Scale (GCS), vital signs measurements (systolic blood pressure [SBP], diastolic blood pressure [DBP], mean arterial pressure [MAP], pulse rate, respiratory rate [RR], and temperature) were also documented.

Furthermore, we recorded the initial laboratory investigation results, including arterial blood gas (ABG) consisting of blood pH, serum bicarbonate (HCO_3), partial arterial carbon dioxide pressure ($PaCO_2$), and oxygen (O_2) saturation. Likewise, the first reading of random blood sugar (RBS), serum electrolytes (sodium and potassium levels), liver enzymes including aspartate aminotransferase (AST) and alanine aminotransferase (ALT), and kidney function tests were also gathered. The serum glucose to potassium ratio was calculated by dividing serum glucose concentration (mmol/L) by serum potassium concentration (mmol/L) [19]. Electrocardiogram (ECG) findings were also reported on admission for every included patient.

2.6. Calculation of various scoring systems

2.6.1. Poisoning Severity Score (PSS)

To categorize the degree of the severity of the poisoning, we utilized the Poisoning Severity Score (PSS) according to the International Programme on Chemical Safety (IPCS). PSS stratified the patients according to the most severe presentations into four groups: grade 0 (no symptoms), grade 1 (mild transient with spontaneously resolving symptoms), grade 2 (pronounced or prolonged symptoms), and grade 3 (severe or life-threatening symptoms) [33].

2.6.2. APACHE II score

The APACHE II score incorporates routine physiological variables, with the total score ranging from 1 to 71 points. It includes twelve parameters, i.e., pulse rate, respiratory rate, mean arterial pressure, oxygen saturation, temperature, arterial pH, serum sodium, serum potassium, serum creatinine, leukocyte count, hematocrit, and GCS. Each parameter is assigned from 0 (normal) to 4 (worst) points. Afterward, the total point values were added to adjusting marks of age chronic health problems (severe organ insufficiency or immune-compromised patients) [34,35].

2.6.3. PGI score

The PGI score calculation was based on clinical and metabolic characteristics stratifying the risk of ALP-poisoned patients for poor outcomes. It includes pH < 7.25, GCS < 13, or SBP$<$90 mmHg. Each variable scores with a single point, and the total score ranges from 0 to 3, where the highest score value refers to severe toxidrome [36].

2.7. Treatment and patient outcomes

All patients received the ordinary treatment according to the TUPCC regimen adopted from the international guidelines. Since there is no specific antidote till now, supportive treatment is the cornerstone of ALP therapy. The supportive treatment includes ensuring a patent airway, concomitant intravenous fluid administration with vasopressors to maintain the hemodynamic status, and correcting metabolic acidosis with sodium bicarbonate. Additionally, according to recent guidelines recommendations, gastric decontamination was performed using paraffin oil. All patients with unstable hemodynamics were admitted to the intensive care unit (ICU), and mechanical ventilation (MV) therapy was applied in patients with respiratory failure.

Finally, patients were categorized according to the primary outcomes (mortality) into survivors and nonsurvivors. Additionally, the predictive role of the Glu-K ratio was investigated in requiring ICU admission and MV.

2.8. Statistical analysis

Analyses were conducted by the R Statistical language (version 4.4.1; R Core Team, 2024) using the packages rstatix (version 0.7.2; Kassambara A, 2023)[37], pROC (version 1.18.5; Robin X et al., 2011)[31], gtsummary (version 2.0.0; Sjoberg D et al., 2021)[38], and ggsurvfit (version 1.1.0; Sjoberg D et al., 2024)[39], and survival (version 3.7.0; Therneau T, 2024)[40]. Continuous quantitative variables were assessed for normality of distribution using the Shapiro-Wilk test and Q-Q plots. As all variables did not follow the normal distribution, they were presented using the median and interquartile range (IQR; 25th – 75th percentiles), and comparisons were carried out using the Wilcoxon rank sum test. Spearman's rank-order correlation was used to assess the relationship between the Glu-K ratio and scoring systems (PSS, APACHE II, and PGI). Categorical variables were summarized as counts and frequencies, and their association with studied groups was evaluated using either Pearson's Chi-square test for independence of observations or Fisher's exact test. The ROC curve was analyzed to determine the diagnostic performance of relevant predictors for the studied outcomes, with reporting of area under the curve (AUC) as well as the optimal cutoff value with its associated sensitivity, specificity, predictive values, and overall accuracy. Pairwise comparisons of AUCs of tested predictors were conducted using DeLong's test for two correlated ROC curves. Survival analysis was performed by constructing Kaplan-Meier curves for assessing the time till death and the potential relationship with the Glu-K ratio. A log-rank test was performed. A p-value < 0.05 was selected for interpreting the results of significance tests.

3. Results

Out of the included patients (206), the mortality rate was 67.5 %. More than half of the studied patients were females ($n = 122$; 59 %) with a median age [IQR] of 19 [18.0–27.0]. The nonsurvivors had significantly younger ages than survivors, with predominance among males ($p = 0.014$ and < 0.001, respectively). Although most of the nonsurvivors were from rural regions and alleged suicidal intentions, these observed findings did not reach statistical significance compared to survivors ($p>0.05$). The number of ingested ALP tables significantly affected the outcomes ($p=0.044$). Although there was no significant difference between groups regarding the delay time till hospital admission, the nonsurvivors had a significantly shorter hospitalization period than survivors ($p<0.001$). All of the nonsurvivors were admitted to the ICU, and most of them required MV (81 %), with a significant difference compared to survivors ($p<0.001$ for each), as demonstrated in Table 1.

Table 2 depicts a summarized overview of the initial clinical assessment of the studied patients. The nonsurvivors had significantly lower SBP, DBP, MAP, and pulse rates than survivors ($p<0.001$). Conversely, the respiratory rate of nonsurvivors was substantially higher than survivors ($p<0.001$). All patients with disturbed consciousness (30 %) and agitation (7.2 %) belonged to the nonsurvivor group with significant differences compared to survivors ($p<0.001$ and 0.032, respectively). Additionally, most nonsurvivors had abnormal ECG findings compared to survivors ($p<0.001$). Accordingly, there was a significant association between mortality and PSS ($p<0.001$), where the majority of nonsurvivors belonged to grade 3 compared to survivors (88 % versus 28 %). Compared to survivors, the medians of APACHE II (12 *versus* 6; $p<0.001$) and PGI scores (1 *versus* 0, $p<0.001$) in nonsurvivors were significantly higher.

The results of initial laboratory investigations are depicted in Table 3. Regarding ABG parameters, the nonsurvivors had significantly lower median values of pH, HCO_3, and O_2 saturation than survivors ($p < 0.001$). Although the median serum sodium level was significantly higher in nonsurvivors, the serum K level was considerably lower than in survivors ($p < 0.001$ and 0.017, respectively). Compared to survivors, the median values of serum ALT, blood urea, and serum creatinine were significantly higher in nonsurvivors ($p= 0.011$, < 0.001, and < 0.001, respectively). Likewise, RBS and Glu-K medians were substantially higher in nonsurvivors than survivors (150 mg/dl *versus* 105 mg/dl and 44.8 *versus* 28.9, respectively). Significant positive correlations were observed between Glu-K and each of PSS ($r = 0.318$, $p< 0.001$), APACHE II score ($r = 0.372$, $p< 0.001$), and PGI score ($r = 0.264$, $p < 0.001$), as revealed in Fig. 1.

The predictive ROC curve analyses of the PSS, APACHE II, PGI score, and Glu-K ratio of potential outcomes with pairwise comparisons were demonstrated in Table 4 and Figs. 2- 4. The APACHE II score had a substantially highest performance for predicting the likelihood of mortality and the need for MV (AUC=0.876 and 0.853, respectively) compared to other scoring systems and the Glu-K ratio ($p< 0.05$). Additionally, PSS exhibited the highest discriminatory power (AUC = 0.833) in expecting the need for ICU admission with no significant differences from other studied scores and the Glu-K ratio. However, PGI at cutoff ≥ 1 had fair to good discrimination in predicting all outcomes without substantial differences from PSS and Glu-K ratio ($.p > 0.05$). Although the Glu-K ratio conveyed fair, acceptable performance (0.743, 0.701, and 0.782) for anticipating all unfavorable outcomes, no significant difference was observed between the Glu-K ratio and scoring systems (PSS and PGI), highlighting the comparable discriminatory power between them ($p> 0.5$).

Analysis of overall survival by Kaplin-Meier curve analysis revealed that the median time to death or discharge in the studied patients was 0.5 days (95 % CI: 0.38, 0.54). At the end of one week from admission, the survival probability was only 32 % (95 % CI: 26 %, 39), as shown in Table 5 and Fig. 5. Survival in subgroups was assessed based on the

Table 1
Socio-demographics, toxicological data, and outcomes of acute aluminum phosphide-poisoned patients ($n = 206$).

Characteristics	Outcome			
	Overall n = 206	Survived n = 67	Not survived n = 139	*p* value*[1]
Age (years)				0.014*[2]
Median	19.0	20.0	18.0	
[IQR]	[18.0–27.0]	[18.0–30.0]	[18.0–21.0]	
Range	18.0–55.0	18.0–50.0	18.0–55.0	
Sex, n (%)				< 0.001*[3]
Male	84 (41 %)	16 (24 %)	68 (49 %)	
Female	122 (59 %)	51 (76 %)	71 (51 %)	
Residence, n (%)				0.247[4]
Rural	203 (99 %)	65 (97 %)	138 (99 %)	
Urban	3 (1.5 %)	2 (3.0 %)	1 (0.7 %)	
Mode, n (%)				> 0.999[4]
Suicidal	206 (100 %)	67 (100 %)	139 (100 %)	
Accidental	0 (0 %)	0 (0 %)	0 (0 %)	
Route, n (%)				> 0.999[4]
Oral	206 (100 %)	67 (100 %)	139 (100 %)	
Inhalation	0 (0 %)	0 (0 %)	0 (0 %)	
Amount (tablet), n (%)				
Median [IQR]	1.0 [0.5–1.0]	1.0 [0.5–1.0]	1.0 [1.0–1.0]	< 0.001*[3]
Range	0.3–3.0	0.3–2.0	0.3–3.0	
1/4	25 (12.1 %)	14 (20.9 %)	11 (7.91 %)	0.044*[5]
1/2	39 (18.9 %)	18 (26.9 %)	21 (15.1 %)	
3/4	1 (0.49 %)	0 (0.0 %)	1 (0.72 %)	
1	127 (61.7 %)	34 (50.7 %)	93 (66.9 %)	
2	8 (3.88 %)	1 (1.49 %)	7 (5.04 %)	
3	6 (2.91 %)	0 (0.0 %)	6 (4.32 %)	
Delay (hours)				0.050[2]
Median [IQR]	2.0 [1.5–3.5]	2.5 [2.0–4.0]	2.0 [1.5–3.0]	
Range	0.5–6.0	0.5–6.0	0.5–6.0	
Length of hospital stay (hours)				< 0.001*[2]
Median [IQR]	69.0 [48.0–96.0]	12.0 [6.0–48.0]	7.0 [4.0–12.0]	
Range	2.5–324.0	8.0–324.0	2.5–72.0	
ICU admission, n (%)				< 0.001*[3]
Yes	166 (80.6 %)	27 (40.3 %)	139 (100.0 %)	
No	40 (19.4 %)	40 (59.7 %)	0 (0 %)	
Mechanical ventilation, n (%)				< 0.001*[3]
Yes	116 (56 %)	4 (6.0 %)	112 (81 %)	
No	90 (44 %)	63 (94 %)	27 (19 %)	

[1] * Significant at $p<0.05$;
[2] Wilcoxon rank sum test;
[3] Pearson's Chi-squared test;
[4] Fisher's exact test;
[5] Chi-squared Test for Trend in Proportions; n: Number; IQR: Interquartile range; ICU: Intensive care unit

studied predictor's cutoff values. We demonstrated a significant decrease in survival probability and time to death or discharge in patients with a Glu-K ratio ≥ 37.07 compared to those with a ratio < 37.07 (0.38 versus 3 days; $p < 0.001$), as depicted in Table 5 and Fig. 6.

4. Discussion

Acute ALP poisoning is particularly prevalent in various low-income countries and constitutes one of the most challenging medical emergencies [41]. Given the steadily increasing incidences of related morbidity and mortality, the prognostic stratification of patients intoxicated with ALP is advisable to triage patients and allocate resources appropriately for prompt initiation of therapeutic interventions that could contribute to better outcomes [13,42].

We aimed to assess the Glu-K ratio's predictive potential for unfavorable outcomes (mortality, ICU admission, and MV) and the power of its correlation with the severity of ALP poisoning and with other conventional scores such as APACHE II and PGI. The Glu-K ratio performs comparably to PSS and PGI in assessing poisoning severity and forecasting various outcomes, suggesting its practical ability for clinical decision-making in resource-limited emergencies.

As revealed in the current study, 67.5 % of the patients did not survive. The higher mortality incidence in our study could be explained by potent ALP poisoning, which is in line with El-Sarnagawy et al. (2024)[16], Khalaf et al.(2023), [3], and Elhosary and Hodeib (2020) [43] who recorded 70 %, 72 %, and 73.9 % ALP fatalities, respectively. However, lower mortality rates (39 % and 43 %) were reported by Anbalagan et al. (2023)[42] and Sheta et al. (2019)[44], respectively, due to discrepancies in poisoning-related factors, poisoning severity degree, and early patient presentation, permitting rapid initiating treatment [45].

In the current study, mortality was significantly observed at a young age, with males predominance compared to survivors ($p = 0.014$ and < 0.001, respectively). Accordingly, Saravani et al. (2024) [46] demonstrated that the highest number of ALP-poisoned patients (35 % and 33 %) belonged to two age groups (11−20) and (21−30), respectively. However, Sheta et al. (2019) [44], Bogale et al. (2021)[47], and Deraz et al. (2022) [48] observed a significant association between ALP mortality and young females. Furthermore, we detected suicidal ALP ingestion in all patients without substantial differences between groups. The young age group usually experiences anxiety resulting from stressful life conditions, especially family problems, failure in love, and education difficulties, making them a vulnerable group to commit suicide [49,50]. Additionally, the rural residents' predominance in our study could be explained by the broad farming region in our locality, as well as affordable prices and easy ALP accessibility without strict regulation of

Table 2
Initial clinical characteristics, scoring systems, and electrocardiograms of acute aluminum phosphide-poisoned patients ($n = 206$).

Characteristics	Outcome			
	Overall n = 206	Survived n = 67	Not survived n = 139	*p* value*[a]
Consciousness, n (%)				<0.001*[b]
Normal	164 (80 %)	67 (100 %)	97 (70 %)	
Disturbed	42 (20 %)	0 (0 %)	42 (30 %)	
Grading of GCS, n (%)				< 0.001*[d]
Normal	164 (80 %)	67 (100 %)	97 (70 %)	
Mild	25 (12 %)	0 (0 %)	25 (18 %)	
Moderate	8 (3.9 %)	0 (0 %)	8 (5.8 %)	
Severe	9 (4.4 %)	0 (0 %)	9 (6.5 %)	
Agitation, n (%)				0.032*[e]
Yes	10 (4.9 %)	0 (0 %)	10 (7.2 %)	
No	196 (95 %)	67 (100 %)	129 (93 %)	
Vomiting, n (%)				0.541[b]
Yes	120 (58 %)	37 (55 %)	83 (60 %)	
No	86 (42 %)	30 (45 %)	56 (40 %)	
Systolic blood pressure (mmHg)				< 0.001*[c]
Med	80.0	100.0	60.0	
[IQR]	[40.0–100.0]	[90.0–110.0]	[40.0–80.0]	
Range	40.0–160.0	40.0–160.0	40.0–120.0	
Diastolic blood pressure (mmHg)				< 0.001*[c]
Median	40.0	60.0	30.0	
[IQR]	[20.0–60.0]	[50.0–70.0]	[20.0–50.0]	
Range	20.0–100.0	20.0–100.0	20.0–90.0	
Mean arterial pressure (mmHg)				< 0.001*[c]
Median	56.7	73.3	46.7	
[IQR]	[26.7–70.0]	[63.3–80.0]	[26.7–56.7]	
Range	26.7–120.0	26.7–120.0	26.7–100.0	
Pulse rate (beats/minute)				< 0.001*[c]
Median	90.0	96.0	86.0	
[IQR]	[76.0–110.0]	[89.0–120.0]	[72.0–106.0]	
Range	36.0–148.0	67.0–148.0	36.0–140.0	
Respiratory rate (cycles/minute)				< 0.001*[c]
Median	24.0	20.0	26.0	
[IQR]	[20.0–30.0]	[19.0–24.0]	[22.0–31.0]	
Range	14.0–48.0	14.0–40.0	16.0–48.0	
Temperature (Celsius)				0.062[c]
Median	37.0	37.0	37.0	
[IQR]	[37.0–37.0]	[37.0–37.0]	[37.0–37.0]	
Range	36.0–39.0	36.5–39.0	36.0–38.5	
ECG findings, n (%)				< 0.001*[b]
Normal	80 (39 %)	47 (70 %)	33 (24 %)	
Abnormal	126 (61 %)	20 (30 %)	106 (76 %)	
PSS categories, n (%)				< 0.001*[b]
1	22 (11 %)	21 (31 %)	1 (0.7 %)	
2	42 (20 %)	27 (40 %)	15 (11 %)	
3	142 (69 %)	19 (28 %)	123 (88 %)	
APACHE II score				< 0.001*[c]
Median	10.0	6.0	12.0	
[IQR]	[6.0–15.0]	[4.0–8.0]	[10.0–16.0]	
Range	3.0–37.0	3.0–19.0	4.0–37.0	
PGI Score				< 0.001*[c]
Median	1.0	0.0	1.0	
[IQR]	[0.0–1.0]	[0.0–0.0]	[1.0–2.0]	
Range	0.0–3.0	0.0–2.0	0.0–3.0	

[a] * Significance at $p<0.05$;
[b] : Pearson's Chi-squared test;
[c] : Wilcoxon rank sum test;
[d] : Chi-squared Test for Trend in Proportions;
[e] : Fisher's exact test; n: Number; IQR: Interquartile range; GCS: Glasgow coma scale; ECG: Electrocardiography; PSS: Poisoning Severity Score; APACHE II: Acute Physiology and Chronic Health Evaluation II

its sale [51].

Regarding the amount of ingested ALP, the current study showed a significant association between mortality and the number of tablets, aligning with Navabi et al. (2018) [11]. The amount of ingested dose was considered the most predominant poisoning-related factor affecting severity grade and outcomes in ALP poisoning [42].

Consistent with Farzaneh et al. (2018)[52] and Vijay Kumar et al. (2024)[53], our study revealed impaired hemodynamic status in non-survivors, which was represented by lower median values of SBP, DBP, pulse, and MAP compared to survivors. The occurrence of ALP-associated hypovolemia is a well-recognized causative mechanism resulting from dehydration and substantial intravascular fluid loss consequential to increased capillary permeability and adrenal damage inducing peripheral vasodilatation. Additionally, direct phosphine-inducing myocardial suppression is another postulated cause [54].

Similarly, we observed a significant association between morality and impaired consciousness, including lower GCS and agitations. All

Table 3
Initial laboratory investigations of acute aluminum phosphide-poisoned patients ($n = 206$).

Characteristics	Outcome			
	Overall n = 206	Survived n = 67	Not survived n = 139	*p*value*[a]
pH				< 0.001*[b]
Median [IQR]	7.3 [7.3–7.4]	7.4 [7.4–7.5]	7.3 [7.2–7.4]	
Range	6.9–7.6	7.1–7.5	6.9–7.6	
Serum HCO_3 (mEq/L)				< 0.001*[b]
Median [IQR]	14.0 [10.9–18.0]	18.0 [14.8–20.0]	12.0 [9.9–16.5]	
Range	3.9–31.0	7.2–31.0	3.9–26.0	
PCO_2 (mmHg)				0.071[b]
Median [IQR]	26.0 [20.0–32.0]	26.5 [22.3–32.0]	24.8 [19.0–31.5]	
Range	7.0–53.0	12.7–44.8	7.0–53.0	
O_2 saturation (%)				< 0.001*[b]
Median [IQR]	92.0 [83.0–96.0]	96.0 [94.0–98.0]	87.0 [79.0–93.0]	
Range	35.0–100.0	67.0–100.0	35.0–100.0	
Serum Na level (mmol/L)				< 0.001*[b]
Median [IQR]	142.0 [138.0–146.0]	139.0 [136.0–143.0]	144.0 [140.0–146.0]	
Range	120.0–160.0	132.5–152.0	120.0–160.0	
Serum K level (mmol/L)				0.017*[b]
Median [IQR]	3.5 [3.1–3.9]	3.7 [3.3–4.0]	3.5 [3.1–3.8]	
Range	2.1–5.2	2.1–5.2	2.2–4.4	
Serum RBS level (mg/dL)				< 0.001*[b]
Median [IQR]	129.0 [100.0–171.0]	105.0 [91.0–129.0]	150.0 [114.0–196.0]	
Range	42.0–441.0	42.0–348.0	42.0–441.0	
AST (U/L)				0.538[b]
Median [IQR]	24.0 [17.5–32.0]	24.0 [18.0–29.0]	24.0 [15.0–35.0]	
Range	6.0–151.0	10.0–45.0	6.0–151.0	
ALT (U/L)				0.011*[b]
Median [IQR]	18.0 [14.0–27.0]	18.0 [13.0–21.0]	19.0 [15.0–30.0]	
Range	5.0–161.0	5.0–44.0	8.0–161.0	
Blood urea (mg/dL)				< 0.001*[b]
Median [IQR]	32.0 [27.0–38.0]	30.0 [23.0–35.0]	35.0 [28.0–40.0]	
Range	16.0–65.0	17.0–51.0	16.0–65.0	
Serum creatinine (mg/dL)				< 0.001*[b]
Median [IQR]	1.1 [0.9–1.6]	0.9 [0.7–1.0]	1.3 [1.0–1.7]	
Range	0.4–2.4	0.4–1.6	0.6–2.4	
Glucose potassium ratio				< 0.001*[b]
Median [IQR]	36.9 [27.1–51.6]	28.9 [24.6–36.7]	44.8 [31.0–54.4]	
Range	11.1–113.6	14.0–94.1	11.1–113.6	

[a] *Significance at $p < 0.05$;
[b] : Wilcoxon rank sum test; n: Number; IQR: Interquartile range; HCO_3: Bicarbonate; $PaCO_2$: Partial arterial carbon dioxide pressure; O_2: Oxygen; Na: Sodium; K: Potassium; RBS: Random blood sugar; AST: Aspartate aminotransferase, ALT: Alanine aminotransferase

these neurological presentations can be explained by cerebral anoxia resulting from phosphine-inducing hypoxia and systemic hypoperfusion [8].

Additionally, the current study conveyed some laboratory parameters as significant indicators for mortality in acute ALP poisoning, including lower pH and HCO_3, referring to metabolic acidosis status. Previous studies have documented that metabolic acidosis is a significant prognostic indicator of mortality [9,44,52]. The ALP-inducing metabolic acidosis is explained by lactic acid accumulation resulting from blockage of oxidative phosphorylation and poor tissue perfusion [55]. Accordingly, we detected the respiratory compensation mechanism for metabolic acidosis in nonsurvivors by significantly increasing

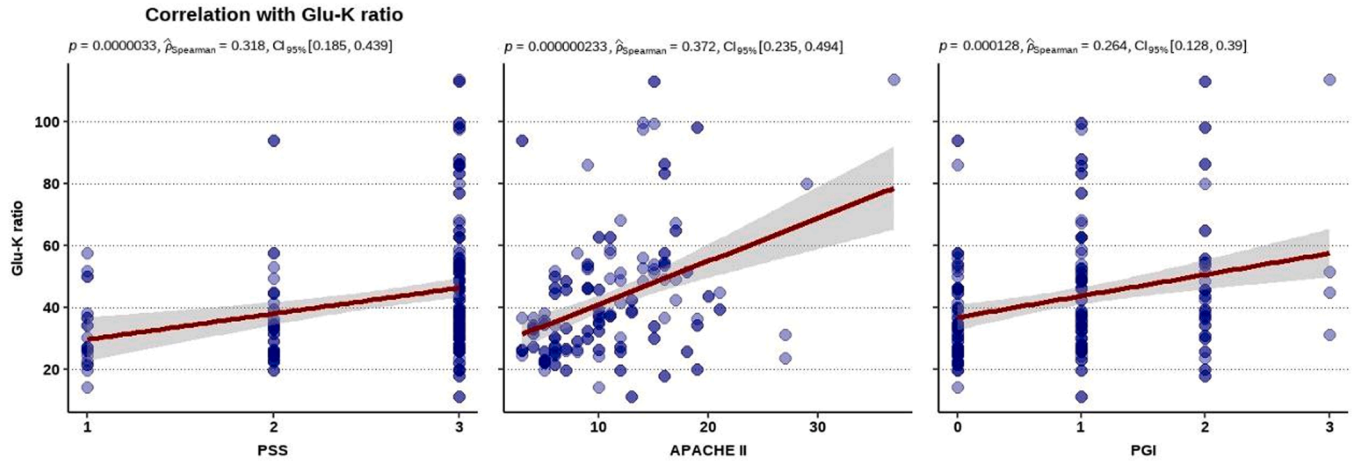

Fig. 1. Correlation between glucose-potassium (Glu- K) ratio and scoring systems (PSS: Poisoning Severity Score, APACHE II: Acute Physiology and Chronic Health Evaluation II, and PGI score) in acute aluminum phosphide poisoned patients.

Table 4
The receiver operating characteristic (ROC) curve analysis of scoring systems and glucose potassium ratio for predicting outcomes in acute aluminum phosphide poisoned patients (n = 206).

Predictors	AUC* (95 % CI)	Cutoff value	TP (N)	FP (N)	TN (N)	FN (N)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)
Mortality											
PSS	0.816 (0.756–0.877)	≥ 3	123	19	48	16	88.49	71.64	86.62	75.00	83.01
APACHE II	0.876 (0.822–0.930)	≥ 8.5	98	14	53	17	85.22	79.10	87.50	75.71	82.97
PGI score	0.815 (0.759–0.870)	≥ 1	113	16	51	26	81.29	76.12	87.60	66.23	79.61
Glu-K ratio	0.743 (0.671–0.814)	≥ 37.07	88	14	53	51	63.31	79.10	86.27	50.96	68.45
Mechanical ventilation											
PSS	0.779 (0.723–0.835)	≥ 3	108	34	56	8	93.10	62.22	76.06	87.50	79.61
APACHE II	0.853 (0.795–0.910)	≥ 9.5	64	19	79	20	76.19	80.61	77.11	79.80	78.57
PGI Score	0.763 (0.702–0.823)	≥ 1	95	34	56	21	81.90	62.22	73.64	72.73	73.30
Glu-K ratio	0.701 (0.629–0.773)	≥ 36.90	76	27	63	40	65.52	70.00	73.79	61.17	67.48
ICU admission											
PSS	0.833 (0.757–0.910)	≥ 3	133	9	31	33	80.12	77.50	93.66	48.44	79.61
APACHE II	0.819 (0.744–0.893)	≥ 8.5	106	6	34	36	74.65	85.00	94.64	48.57	76.92
PGI Score	0.775 (0.705–0.845)	≥ 1	121	8	32	45	72.89	80.00	93.80	41.56	74.27
Glu-K ratio	0.782 (0.712–0.852)	≥ 37.07	98	4	36	68	59.04	90.00	96.08	34.62	65.05

AUC: Area under the curve; CI: Confidence interval; FN: False negative; FP: False positive; NPV: Negative predictive value; PPV: Positive predictive value; TN: True negative; TP: True positive; PSS: Poisoning severity score; Glu-K: Glucose potassium ratio; ICU: intensive care unit; APACHE II: Acute Physiology and Chronic Health Evaluation II.

* Pairwise testing of AUCs using Delong's test for paired ROC curves: PSS vs. Glu-K ratio ($p = 0.101$ for predicting mortality, 0.068 for predicting mechanical ventilation, and 0.349 for predicting ICU); PSS vs. APACHE II ($p = 0.038$ for predicting mortality, 0.031 for predicting mechanical ventilation, and 0.822 for predicting ICU admission); PSS vs. PGI ($p = 0.969$ for predicting mortality, 0.600 for predicting mechanical ventilation, and 0.083 for predicting ICU); Glu-K ratio vs. APACHE II ($p = 0.004$ for predicting mortality, 0.001for predicting mechanical ventilation, and 0.476 for predicting ICU); Glu-K ratio vs. PGI ($p = 0.121$ for predicting mortality, 0.171for predicting mechanical ventilation, and 0.881 for predicting ICU); APACHE II vs. PGI ($p = 0.010$ for predicting mortality, 0.001for predicting mechanical ventilation, and 0.051 for predicting ICU).

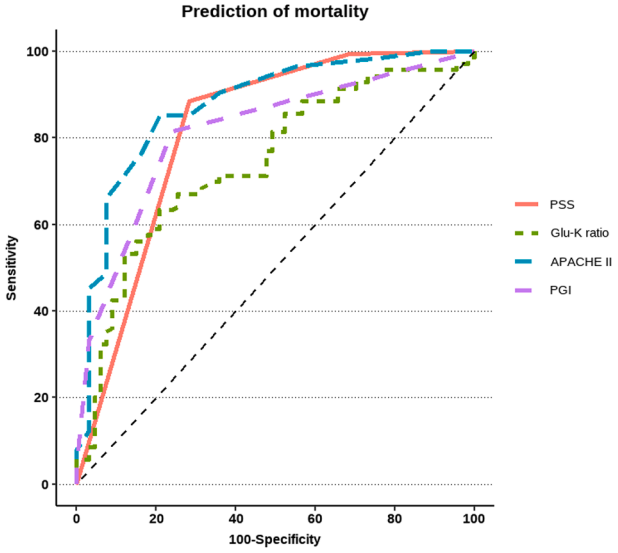

Fig. 2. Receiver operating characteristics (ROC) curve of Poisoning Severity Score (PSS), Acute Physiology and Chronic Health Evaluation II (APACHE II), PGI score and glucose potassium ratio (Glu-K) for predicting mortality in acute aluminum phosphide poisoned patients.

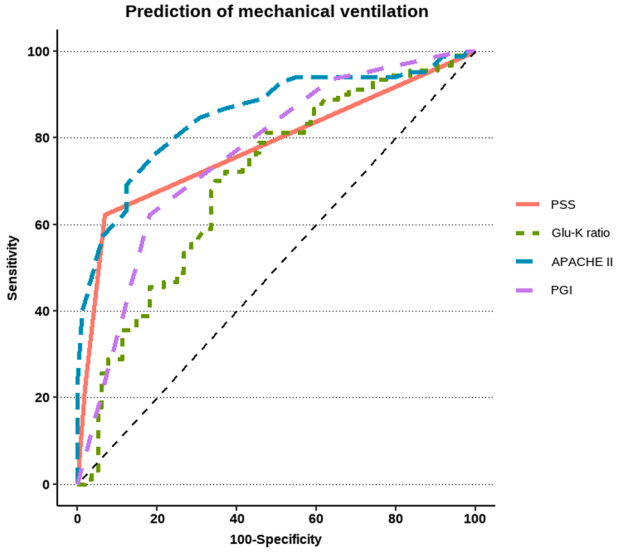

Fig. 3. Receiver operating characteristics (ROC) curve of Poisoning Severity Score (PSS), Acute Physiology and Chronic Health Evaluation II (APACHE II), PGI score and glucose potassium ratio (Glu-K) for predicting the need for mechanical ventilation in acute aluminum phosphide poisoned patients.

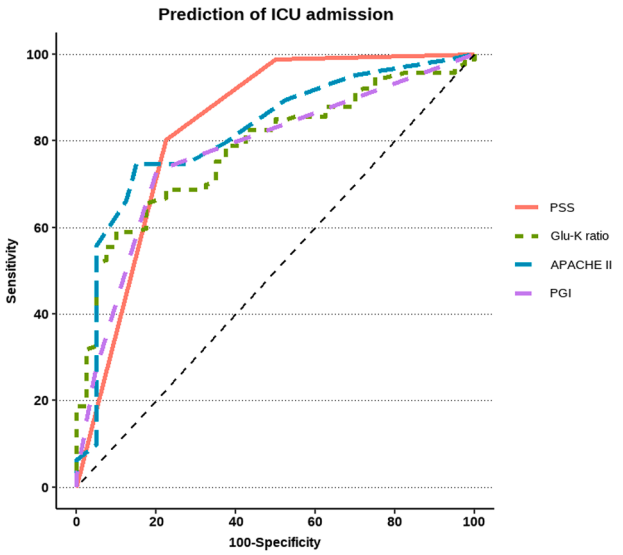

Fig. 4. Receiver operating characteristics (ROC) curve of Poisoning Severity Score (PSS), Acute Physiology and Chronic Health Evaluation II (APACHE II), PGI score and glucose potassium ratio (Glu- K) for predicting the intensive care unit admission requirement in acute aluminum phosphide poisoned patients.

the RR.

Compared to the survival group, liver enzymes, renal function tests, and total leucocytic count were significantly higher in the mortality group. Although liver damage manifestations are less severe, transient elevation of liver enzymes is a common finding [56]. Furthermore, hypoxia and shock-associated ALP poisoning commonly predispose to renal impairment [57]. Additively, Fayyaz. [58] has documented that infections, inflammation, stress, metabolic imbalance, cardiac infarction, and acute renal failure are the commonly assumed mechanisms for ALP-inducing leucocytosis, which is supported by previous studies [18, 55].

We noted that all the nonsurvivors were admitted to the ICU, with a considerable portion requiring MV (81 %). A noticeable increase in the percentage of non-survived 123 (88 %) in severe poisoning grade explains these higher incidences of poor outcomes. Likewise, higher percentages of nonsurvivors were admitted to the ICU (94.6 % and 86.4 %) and mechanically ventilated (86.4 % and 43.6 %) in ALP-poisoned patients as reported by El-Sarnagawy et al. (2024) [16] and Zaki et al. (2024) [4], respectively.

In the current study, the median value of RBS was significantly higher in nonsurvivors than in survivors (150 mg/dL versus 105 md/dL). Likewise, Sharma et al. (2018) [9] recorded a significant elevation of mean blood glucose level (159.7 ± 92.5 mg/dL versus 119.9 ± 35.7 mg/dL) and frequency of blood glucose alteration (42% versus 5%) in nonsurvivors compared to survivors, respectively. Furthermore, Mehrpour et al. (2008) [59] and Kadamati et al. [60] observed a substantial increase in blood glucose levels in fatal ALP cases compared to survived patients (222.6 ± 20 mg/dL versus 143.4 ± 13.7 mg/dL and 223.1 mg/dL versus 142.5 mg dL, respectively).

Subsequently, Lionate et al. [61] revealed a significant association between multiple glycemic parameters and in-hospital adverse outcomes in acutely poisoned patients. Notably, Tawfik. [62] concluded that hyperglycemia was one of the crucial determinants for predicting mortality in acute AlP poisoning using logistic regression analysis. Furthermore, Mehrpour et al. [59] and Kadamati et al. [60] demonstrated that the existence of hyperglycemia increased the risk for ALP fatalities by 5.7 times after adjusting other significant predictors (age, gender, ingested amount, pH, and HCO_3 concentration).

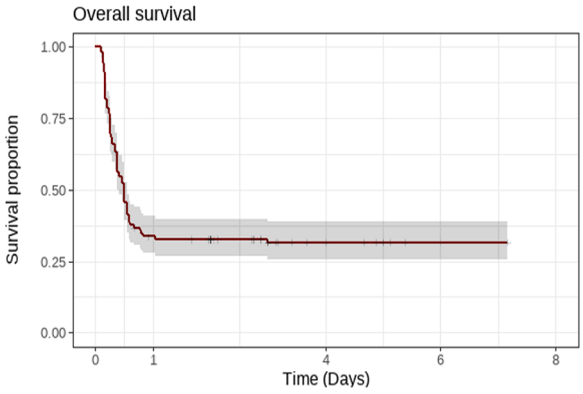

Fig. 5. Kaplan–Meier overall survival analysis curve of aluminum phosphide poisoned patients.

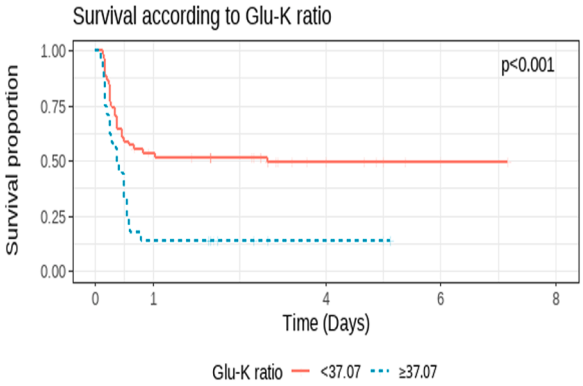

Fig. 6. Kaplan–Meier analysis of the survival probability of acute aluminum phosphide poisoned patients stratified by glucose-potassium ratio (Glu-K) cut-off level.

Table 5
Kaplan–Meier curve analysis for overall survival duration and survival probability according to glucose-potassium ratio in acute aluminum phosphide-poisoned patients.

Characteristics	OS% (95 % CI)			Median survival (days)	
	1 Day(s)	3 Day(s)	7 Day(s)	Median (95 % CI)	p-value[1]
Overall	34 % (28 %, 41 %)	32 % (26 %, 39 %)	32 % (26 %, 39 %)	0.50 (0.38, 0.54)	
Glu-K ratio					<0.001*
<37.07	54 % (45 %, 64 %)	50 % (40 %, 61 %)	50 % (40 %, 61 %)	3.0 (0.50, 3.0)	
≥ 37.07	14 % (8.4 %, 22 %)	14 % (8.4 %, 22 %)	Not reached	0.38 (0.29, 0.50)	

[1] :Log-rank test;
* significant at $p < 0.05$; CI: confidence interval; Glu-K: Glucose potassium ratio

Various hypothesized pathways are postulated for how ALP causes hyperglycemia. Noticeably, ALP induces cellular oxidative phosphorylation impairment, causing a decrease in energy production and ATP levels [59]. Another suggested mechanism is ALP-associated acute pancreatitis either directly or via oxidative stress-mediated generalized inflammatory response, triggering insulin resistance, pancreatic β-cell dysfunction, and glucose tolerance impairment. As always happens in cases of acute poisoning, ALP-linked stress reactions promote sympathetic overstimulation, cause catecholamine release, and induce glucose production by glycogenolysis and gluconeogenesis activation [61,63]. Many previous experimental and human studies support all these theories through decreased ATP production, increased cortisol levels, and degenerative alteration in both adrenal glands and pancreas of fatal ALP poisoning [60,63,64].

The interconnection of hyperglycemia with electrolyte disturbances, reduced immune response, and increased risks of infectious complications may explain hyperglycemia's substantial association with poor outcomes [61]. Noticeably, hyperglycemia was associated with neurological adverse outcomes in CO poisoning [30], the intermediate syndrome in organophosphorus poisoning [65], methanol mortality [66], and poisoning severity of calcium channel blockers [67].

Although hyperglycemia is frequently observed in acute ALP poisoning, mild hypoglycemia has also been reported in acute ALP, though severe hypoglycemia is relatively rare [68]. Hypoglycemia is attributed to hepatic injury inhibiting gluconeogenesis and glycogenolysis, as well as adrenal cortex damage associated with decreased cortisol, glucagon, and epinephrine production [9]. Additionally, ALP-induced vomiting may reduce the patient's appetite and exacerbate the effect of hypoglycemia. Other suggested mechanisms contributing to hypoglycemic status include failure of glucagon secretion and epinephrine synthesis and the release of insulin-like growth factors in response to severe shock [69].

The existence of vomiting as an initial GIT presentation and transcellular serum K shifting caused by stress-mediated released catecholamines are advocated mechanisms for hypokalemia in acute ALP-poisoned patients [70]. Accordingly, we noted a substantial reduction in serum K in nonsurvivors compared to survivors, which aligns with previous studies [16,55].

The relation between alteration in potassium levels and adverse cardiac outcomes is well known in the medical field and poisoning contexts. In hypokalemic patients, Kjeldsen [71] declared that the risk of ventricular fibrillation incidence and mortality rates were found to be increased up to five and 10-fold, respectively [71]. Additionally, Liu et al. [72] and Chang et al. [73] revealed that hypokalemia is an independent predictor of mortality in acute paraquat-poisoned patients [72,73]. Basant et al. [74] concluded that remarkable hypokalemia is an alarming sign of adverse outcomes in organophosphoruspoisoned patients secondary to potentially aggravated cardiac arrhythmias and respiratory muscle paralysis [74].

As a result of the reciprocal connection between RBS and K level, incorporating the two parameters into the Glu-K ratio represents a better indicator for excessive catecholamine release than individual measurements. Previously, an increased Glu-K ratio has been investigated as an indicator of clinical outcomes of cerebral ischemic and traumatic injuries [26,75]. Although the Glu-K ratio has attained considerable interest from clinical toxicologists, assessing its predictive performance in poisoning severity and outcomes in acutely ALP-poisoned patients has not been addressed yet. Owing to a substantial increase in RBS and a decrease in serum K between the two groups, there is a significant elevation of the Glu-K median values among nonsurvivors rather than survivors (44.8 *versus* 28.9).

Our study recorded that the APACHE II score exhibited the best discriminatory power in predicting the likelihood of mortality and the need for mechanical ventilation (AUC=0.876 and 0.853, respectively). Similarly, Farzaneh et al. [52], Dorooshi et al. [76], and Zaki et al. [4] have demonstrated good to excellent performance of APACHE II score for mortality prediction in ALP poisoning (AUC = 0.939, 0.779, and 0.969, respectively).

Additionally, we observed that PSS had the highest performance (AUC = 0.833) in predicting the need for ICU admission with no significant differences from other studied scores and the Glu-K ratio. However, El-Sarnagawy et al. [17] recorded a substantial increase in discriminatory performance in PSS versus APACHE II for predicting ICU admission in ALP-poisoned patients (AUC=0.987 *versus* 0.907; $p < 0.001$).

Our study recorded that the PGI score had moderate performance comparably to PSS and Glu-K ratio in adverse outcomes prediction. Similarly, Pannu et al. [77] demonstrated a strong positive correlation between PGI score and other conventional scores (APACHE II, SOFA, and SAPS-II) on admission and 24 hours later in ALP-poisoned patients. Although El-Sarnagawy et al.[16] illustrated that the PGI score at cutoff > 1 displayed good discriminatory ability in outcome prediction, Sakr et al.[78] verified that the PGI score had excellent discrimination of mortality prediction (AUC = 0.951) in ALP-poisoned patients.

Furthermore, our study revealed that the Glu-K ratio exhibited fair discriminatory power (0.743, 0.701, and 0.782) for anticipating mortality, the MV, and ICU admission requirements, respectively. Likewise, Demirtaş et al. [30] and Yalçın et al. [79] reported fair predictive ability (0.791 and 0.796, respectively) of Glu-K ratio for the likelihood of delayed neuropsychiatric syndrome following acute carbon monoxide poisoning [30,79]. However, Sharif and Fayed [28] and Sharif et al. [29] revealed that the Glu-K ratio had an excellent predictive ability for anticipating intermediate syndrome after acute anticholinesterase intoxication (AUC = 0.971) and life-threatening events of acute methylxanthine intoxication (AUC = 0.906), in the same order [28,29].

Additionally, we revealed that the Glu-K ratio had a significant positive correlation with PSS, PGI, and APACHE II scores ($p< 0.001$) and exhibited comparable prognostic ability with PSS and PGI for predicting adverse outcomes in ALP-poisoned patients ($p > 0.05$). However, incorporating several complex clinical and laboratory parameters in calculating PSS and APACHE II scores limits their clinical utility for timely emergency decision-making [80,81]. Furthermore, a recent study by El-Sarnagawy et al. [16] defined the PGI shortcomings that depend on certain cutoff levels (blood pH < 7.25, GCS score < 13, and SBP$<$ 87 mmHg) underestimating high-risk ALP-poisoned patients with initially higher physiological variables over PGI cutoff levels. Consequently, given the abovementioned results, the Glu-K ratio provides a reliable, simple, objective bedside marker for assessing ALP poisoning severity and outcomes probability.

Furthermore, this study investigated the impact of the Glu-K ratio cutoff on the survival duration of ALP-poisoned patients using Kaplan-Meier curve analysis. Our study demonstrated that patients with a Glu-K ratio ≥ 37.07 should be rapidly allocated to emergent treatment resources as they have a significantly lower median survival time (0.38 days) than patients whose Glu-K ratio was < 37.07 as they had prolonged survival duration (3 days). This finding adds weight to the previous results, highlighting the importance of early Glu-K ratio detection for triaging the high-risk ALP for intensive supportive management.

In ALP-inducing circulatory shock, there is concomitant impairment in myocardial glucose uptake and metabolism, resulting in further myocardial suppression [82]. Accordingly, rational glucose-insulin-potassium infusion (GLK) therapy depends on improving cardiac inotropic and hemodynamic status by increasing myocardial glucose uptake, promoting energy production, and restoring calcium flux [83,84]. This protocol may also refine ALP-inducing metabolic acidosis by endorsing lactic acid metabolism. Furthermore, insulin may improve peripheral vascular resistance by activating the adrenergic receptors and causing vasoconstriction that limits vasopressor use [85].

In this context, our findings could highlight researchers' interest in evaluating the role of GLK regimen in acute ALP poisoning. Hassanian-Moghaddam and Zamani (2016) revealed that the GIK protocol

improved ALP-poisoned patient outcomes and survival [86]. Likewise, Pannu et al. [82] observed a 26.6 % reduction in mortality rate and improved survival and hemodynamics with GIK therapy without altering metabolic acidosis [82]. Furthermore, Adel et al. [85] documented that insulin-euglycemia therapy is safe and effective and associated with a substantial improvement in primary and secondary outcomes in ALP-poisoned patients [85]. Correspondingly, Ullah et al. [84] reported incorporating GIK therapy with the standard treatment reduces 54 % of ALP fatalities and enhances overall survival rates [84]. Subsequently, a systematic review by Shukla et al. [10] concluded that effective insulin therapy reduced the mortality rates of AlP poisoning [10].

5. Limitations

Although this study's results offered a preliminary, easily objective parameter for assessing poisoning severity and unfavorable outcomes in ALP-poisoned patients, being a single-center study on a considerably small sample size may limit the result's generalizability and validity. Therefore, we recommended further multicenter studies on a large patient scale to validate our study's clinical applicability. Additionally, advanced studies should assess stress-related factors, including catecholamines and cortisol levels, to support the earlier theories.

Despite the silver nitrate test being used to confirm phosphine poisoning, false negative and false positive results challenged the test's sensitivity. Noticeably, a false negative result may occur in patients receiving oxygen as phosphine may be converted to phosphorus pentoxide. On the other hand, a false positive result may ensue if hydrogen sulfide is in the air [2]. These misleading results account for the lower sensitivity of testing the breath with silver nitrate-impregnated strips (50 %). However, the silver nitrate test was conducted on gastric aspirate samples in the current study since all included cases were poisoned by suicidal ingestion of ALP tablets. Despite the test demonstrating a sensitivity of 100 % on gastric aspirate, sulfide derivatives may interfere, resulting in false positive outcomes. To differentiate, applying ammonium molybdate on dark filter paper induces a bluish discoloration solely in the presence of phosphine gas [87,88].

6. Conclusion

ALP is a well-known fatal pesticide poisoning with substantial mortalities and adverse outcomes. The Glu-K ratio exhibited a significant correlation and a comparable outcome prediction to PSS and PGI. Additionally, assessing the on-admission Glu-K ratio indicates the survival duration of ALP-poisoned patients. Accordingly, our study recommends adopting the initial Glu-K ratio as a reliable routine biomarker for assessing poisoning severity and outcomes probability to stratify the high-risk ALP-poisoned patients for appropriate intensive management, especially with limited hospital resources.

Funding

This research did not receive a specific grant from public, commercial, or not-for-profit funding agencies.

Author statement

We the undersigned declare that this manuscript is original, has not been published before, and is not currently being considered for publication elsewhere.

CRediT authorship contribution statement

Ghada N. El-Sarnagawy: Writing – review & editing, Validation, Supervision, Methodology, Formal analysis, Data curation, Conceptualization. **Amal S. A. F. Hafez:** Writing – review & editing, Validation, Supervision, Methodology, Formal analysis, Data curation, Conceptualization. **Mona M. Ghonem:** Writing – review & editing, Validation, Supervision, Methodology, Formal analysis, Data curation, Conceptualization.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

All authors thank the TUPCC staff members for their valuable support and assistance in this study.

Data availability

Data will be made available on request.

References

[1] S. Ali, I. Ahmad, Ashraf, Z. Islam, S.K.A. Shah, N. Ali, The effects of wheat pill poisoning (aluminum phosphide) dosage on arterial blood gases and their clinical outcome, J. Pak. Med. Assoc. 73 (2023) 1–12, https://doi.org/10.47391/jpma.8116.

[2] D. Yadav, R. Bhattacharyya, D. Banerjee, Acute aluminum phosphide poisoning: the menace of phosphine exposure, Clin. Chim. Acta 520 (2021) 34–42, https://doi.org/10.1016/j.cca.2021.05.026.

[3] M. Khalaf, F. Amin, A. Hishmat, Outcomes of acute aluminium phosphide poisoning and role of magnesium sulfate as an antidote in Minia poisoning control center, Minia governorate, Egypt, Egypt. J. Forensic Sci. Appl. Toxicol. 23 (2023) 135–148, https://doi.org/10.21608/ejfsat.2024.231351.1301.

[4] F.F. Zaki, Construction of a Crucial Score to predict the Outcome of patients with aluminum phosphide poisoning at the Poison Control Center - Ain Shams University Hospitals, Ain Shams J. Forensic Med. Clin. Toxicol. 43 (2024) 107–120, https://doi.org/10.21608/ajfm.2024.292130.1136.

[5] E.E.-S. Khayal, I. Abd El Rady, A. Morsi, Trends and acute poisoning profiles: 2023 annual report of zagazig university poisoning treatment unit, Egypt. Soc. Clin. Toxicol. J. 12 (2024) 45–59, https://doi.org/10.21608/esctj.2024.278873.1051.

[6] F. Bagherian, N. Kalani, F. Rahmanian, S. Abiri, N. Hatami, M. Foroughian, N. J. Mehramiz, B. Shahi, Aluminum phosphide poisoning mortality rate in Iran; a Systematic Review and Meta-Analysis, Arch. Acad. Emerg. Med. 9 (2021) e66, https://doi.org/10.22037/aaem.v9i1.1396.

[7] H.O. Shaker, O.E.S. Rageh, M. Alnajar, N.F. Alshamaly, W.A. Abdelmaged, M. Abd-ElGawad, Efficacy of intravenous N acetylcysteine as an adjuvant therapy in the treatment of acute aluminum phosphide Poisoning: a systematic review and meta-analysis, BMC Pharmacol. Toxicol. 24 (2023) 1–10, https://doi.org/10.1186/s40360-023-00699-2.

[8] S. Katwal, K. Malbul, S.K. Mandal, S. KC, M.Z. Alam, P. Karki, C. Pant, Successfully managed aluminum phosphide poisoning: a case report, Ann. Med. Surg. 70 (2021) 102868, https://doi.org/10.1016/j.amsu.2021.102868.

[9] A. Sharma, P. Balasubramanian, K.D. Gill, A. Bhalla, Prognostic significance of blood glucose levels and alterations among patients with aluminium phosphide poisoning, Sultan Qaboos Univ. Med. J. 18 (2018) e299–e303, https://doi.org/10.18295/squmj.2018.18.03.006.

[10] R. Shukla, K. Lamichhane, D. Pandey, C.K. Gupta, S. Shukla, Insulin in aluminum phosphide poisoning: A systematic review of the current literature, Medicine 103 (2024) e40066, https://doi.org/10.1097/MD.0000000000040066.

[11] S.M. Navabi, J. Navabi, A. Aghaei, Z. Shaahmadi, R. Heydari, Mortality from aluminum phosphide poisoning in Kermanshah Province, Iran: characteristics and predictive factors, Epidemiol. Health 40 (2018) e2018022, https://doi.org/10.4178/epih.e2018022.

[12] H. Niknahad, R. Heidari, A. Jangjou, V. Asghari, F.M. Niknahad, F. Goudarzi, N. Tavakoli, M. Rahimi, A.M. Niknahad, M. Rashedinia, The therapeutic effect of a novel parenteral formulation of dihydroxyacetone in aluminum phosphide-intoxicated patients, Heliyon 9 (2023) e22165, https://doi.org/10.1016/j.heliyon.2023.e22165.

[13] N. Ahmed, I. El-Mehallawi, M. Abo Elnoor, A. Hodeib, Potential clinical and laboratory prognostic factors for prediction of need for icu admission in acute aluminum phosphide poisoning, Ain Shams J. Forensic Med. Clin. Toxicol. 37 (2021) 98–106, https://doi.org/10.21608/ajfm.2021.182111.

[14] M. Majidi, M. Jamalpour, N. Solmaz, Research paper: prognostic factors of aluminum phosphide poisoning in urmia: a-five-years cross-sectional study, 32663, Int. J. Med. Toxicol. Forensic Med. 11 (2021) 32663, https://doi.org/10.32598/ijmtfm.v11i2.32663.

[15] M.M. Ghonem, A.A. Abdelnoor, A.A. Hodeib, Shock and modified shock indices in predicting poisoning severity and outcomes in acute aluminum phosphide

poisoned patients, Toxicol. Res. 13 (2024), https://doi.org/10.1093/toxres/tfad124.
[16] G.N. El-Sarnagawy, A.A. Abdelnoor, M.M. Ghonem, Performance assessment of new Poisoning Mortality Score and PGI score for predicting mortality in patients with acute aluminum phosphide poisoning, Https://Doi.Org/10.1177/09603271241302208 43 (2024), https://doi.org/10.1177/09603271241302208.
[17] G.N. El-Sarnagawy, A.A. Abdelnoor, A.A. Abuelfadl, I.H. El-Mehallawi, Comparison between various scoring systems in predicting the need for intensive care unit admission of acute pesticide-poisoned patients, Environ. Sci. Pollut. Res. 29 (2022) 33999–34009, https://doi.org/10.1007/s11356-021-17790-y.
[18] M. Ghonem, S. El Sharkawy, H. Lashin, Predictive variables of acute aluminum phosphide poisoning outcome: a new proposed model, Egypt. J. Forensic Sci. Appl. Toxicol. 20 (2020) 45–60, https://doi.org/10.21608/ejfsat.2020.18651.1106.
[19] X.Y. Wu, Y.K. Zhuang, Y. Cai, X.Q. Dong, K.Y. Wang, Q. Du, W.H. Yu, Serum glucose and potassium ratio as a predictive factor for prognosis of acute intracerebral hemorrhage, J. Int. Med. Res. 49 (2021) 03000605211009689, https://doi.org/10.1177/03000605211009689.
[20] X. Remesar, M. Alemany, Dietary energy partition: the central role of glucose, Int. J. Mol. Sci. 21 (2020) 1–38, https://doi.org/10.3390/ijms21207729.
[21] P. Russo, F. Lauria, A. Siani, Regulation of potassium homeostasis, in: Encycl. Endocr. Dis., Elsevier, 2018, pp. 552–557, https://doi.org/10.1016/B978-0-12-801238-3.65419-3.
[22] F. Boyuk, The Predictor potential role of the glucose to potassium ratio in the diagnostic differentiation of massive and non-massive pulmonary embolism, Clin. Appl. Thromb. 28 (2022), https://doi.org/10.1177/10760296221076146.
[23] J. Wang, Q. Feng, Y. Zhang, W. Qiu, H. Gao, Elevated glucose-potassium ratio predicts preoperative rebleeding in patients with aneurysmal subarachnoid hemorrhage, Front. Neurol. 12 (2022), https://doi.org/10.3389/fneur.2021.795376.
[24] D. Acar, E. Kadıoğlu, N. Kenan, E. Doğan, A.M. Çap, Y. Yılmaz, The Glucose/Potassium ratio exhibits a predictive role that is both earlier and more efficacious compared to the inflammatory response in the context of isolated thoracic trauma, Genel Tıp Derg. 34 (2024) 358–364, https://doi.org/10.54005/geneltip.1428167.
[25] W. Zhou, Y. Liu, Z. Wang, Z. Mao, M. Li, Serum glucose/potassium ratio as a clinical risk factor for predicting the severity and prognosis of acute traumatic spinal cord injury, BMC Musculoskelet. Disord. 24 (2023), https://doi.org/10.1186/s12891-023-07013-5.
[26] M. Joaquín Ignacio, M.E. Sein, The role of the glucose potassium ratio in the management of traumatic brain injury, Korean J. Neurotrauma 19 (2023) 82–89, https://doi.org/10.13004/kjnt.2023.19.e11.
[27] Y. Tekin, E. Demirtaş, İ. Korkmaz, Y. Demirel, G. Tekin, N. Nur, Correlation between serum glucose/potassium ratio and the severity of mushroom poisoning at the time of admission to the emergency departments, Cumhur. Med. J. 44 (2022) 104–109, https://doi.org/10.7197/cmj.1068418.
[28] A.F. Sharif, M.M. Fayed, Assessment of the serum glucose/potassium GLU/K ratio as a predictor of intermediate syndrome following acute anticholinesterase exposure, Neurotoxicology 89 (2022) 161–173, https://doi.org/10.1016/j.neuro.2022.02.001.
[29] A.F. Sharif, Z.A. Kasemy, H.A. Mabrouk, O. Shoeib, M.M. Fayed, Could the serum glucose/potassium ratio offer an early reliable predictor of life-threatening events in acute methylxanthine intoxication? Toxicol. Res. 12 (2023) 310–320, https://doi.org/10.1093/toxres/tfad023.
[30] E. Demirtaş, I. Korkmaz, Y.K. Tekin, E. Demirtaş, I. Çaltekin, Assessment of serum glucose/potassium ratio as a predictor for delayed neuropsychiatric syndrome of carbon monoxide poisoning, Hum. Exp. Toxicol. 40 (2021) 207–213, https://doi.org/10.1177/0960327120945773.
[31] X. Robin, N. Turck, A. Hainard, N. Tiberti, F. Lisacek, J.C. Sanchez, M. Müller, pROC: An open-source package for R and S+ to analyze and compare ROC curves, BMC Bioinforma. 12 (2011) 1–8, https://doi.org/10.1186/1471-2105-12-77.
[32] R. Anand, B.K. Binukumar, K.D. Gill, Aluminum phosphide poisoning: an unsolved riddle, J. Appl. Toxicol. 31 (2011) 499–505, https://doi.org/10.1002/jat.1692.
[33] H.E. Persson, G.K. Sjöberg, J.A. Haines, J.P. De Garbino, Poisoning severity score. Grading of acute poisoning, J. Toxicol. - Clin. Toxicol. 36 (1998) 205–213, https://doi.org/10.3109/15563659809028940.
[34] W.A. Knaus, E.A. Draper, D.P. Wagner, J.E. Zimmerman, APACHE II: a severity of disease classification system, Crit. Care Med. 13 (1985) 818–829, https://doi.org/10.1097/00003246-198510000-00009.
[35] H. Sungurtekin, E. Gürses, C. Balci, Evaluation of several clinical scoring tools in organophosphate poisoned patients, Clin. Toxicol. 44 (2006) 121–126, https://doi.org/10.1080/15563650500514350.
[36] A.K. Pannu, A. Bhalla, A. Sharma, N. Sharma, PGI Score": a simplified three-point prognostic score for acute aluminum phosphide poisoning, Indian J. Crit. Care Med. 24 (2020) 790–793, https://doi.org/10.5005/jp-journals-10071-23555.
[37] A. Kassambara, Pipe-Friendly Framework for Basic Statistical Tests [R package rstatix version 0.7. 2]. Comprehensive R Archive Network (CRAN), (2023).
[38] D.D. Sjoberg, K. Whiting, M. Curry, J.A. Lavery, J. Larmarange, Reproducible summary tables with the gtsummary package, R. J. 13 (2021) 570–580, https://doi.org/10.32614/rj-2021-053.
[39] T.T. Sjoberg D, Baillie M., Fruechtenicht C., Haesendonckx S., Authors and Citation • biomod2, 2004. (n.d.). ⟨https://www.danieldsjoberg.com/ggsurvfit/authors.html⟩ (accessed November 25, 2024).
[40] T.M. Therneau, Survival Analysis [R package survival version 3.7-0, CRAN Contrib. Packag. (2024), https://doi.org/10.32614/cran.package.survival.
[41] G. El-Sarnagawy, Predictive factors of mortality in acute aluminum phosphide poisoning: 5 years retrospective study in tanta poison control unit, Ain Shams J. Forensic Med. Clin. Toxicol. 29 (2017) 70–79, https://doi.org/10.21608/ajfm.2017.18211.
[42] L.C. Anbalagan, A.K. Pannu, A. Bhalla, D.P. Dhibar, N. Sharma, Prognostic significance of poison-related factors and consumption patterns in acute aluminum phosphide poisoning, Turk. J. Emerg. Med. 23 (2023) 88–95, https://doi.org/10.4103/tjem.tjem_253_22.
[43] N. Elhosary, A. Hodeib, Blood lactate and lactate pyruvate ratio as prognostic biomarkers of outcome in acute aluminum phosphide poisoning, Egypt. J. Forensic Sci. Appl. Toxicol. 20 (2020) 15–30, https://doi.org/10.21608/ejfsat.2020.22995.1124.
[44] A.A. Sheta, A.S. El-Banna, R.A. Elmeguid, H.E. Mohamed, N.H. Gad, A study of the predictive factors of mortality in acute poisoning with aluminum phosphide with special reference to echocardiography and SOFA score, Environ. Sci. Pollut. Res. 26 (2019) 33135–33145, https://doi.org/10.1007/s11356-019-06457-4.
[45] S.S. Vijay Kumar, S. Shekhli, A. Jose, Examining the effectiveness of prognostic scoring systems and mortality predictors in aluminum phosphide poisoning: insights from emergency department settings, Emerg. Care J. (2024), https://doi.org/10.4081/ecj.2024.12557.
[46] K. Saravani, A.A. Ghamsari, B. Dadpour, A.G. Toussi, S.R. Mousavi, F. Pouramiri, A review on complications and management of aluminium phosphide (Rice Pill) and Zinc Phosphide Poisoning, Nov. Biomed. 12 (2024), https://doi.org/10.22037/NBM.V12I4.44690.
[47] D.E. Bogale, B.D. Ejigu, T.A. Muche, Clinical profile and treatment outcome of aluminum phosphide poisoning in felege hiwot referral hospital, northwest ethiopia: a retrospective study, Open Access Emerg. Med. 13 (2021) 239–248, https://doi.org/10.2147/OAEM.S313181.
[48] R. Deraz, D. Elrafey, D. Mesallam, Acute aluminium phosphide poisoning in east Delta, Egypt: a growing public health problem over the last five years, Egypt. Soc. Clin. Toxicol. J. 10 (2022) 49–61, https://doi.org/10.21608/esctj.2022.140273.1009.
[49] G. El-Sarnagawy, A. Hafez, R. Amer, Characteristics of suicidal poisoned patients admitted to tertiary care center during COVID-19 pandemic, Egypt. J. Neurol. Psychiatry Neurosurg. 58 (2022), https://doi.org/10.1186/S41983-022-00577-4.
[50] F. Kandeel, R. Azab, Prognostic value of blood lactate and C-reactive protein levels in acute Phosphides poisoned patients admitted to Menoufia University Poison control Center, Mansoura J. Forensic Med. Clin. Toxicol. 31 (2023) 47–60, https://doi.org/10.21608/mjfmct.2023.200085.1060.
[51] S. Abd Elghany, M. Heshmat, M. Oreby, G. Elsarnagawy, Evaluation of various scoring systems in prediction of acute aluminum phosphide (ALP) poisoning outcome, Ain Shams J. Forensic Med. Clin. Toxicol. 30 (2018) 117–127, https://doi.org/10.21608/ajfm.2018.18202.
[52] E. Farzaneh, H. Ghobadi, M. Akbarifard, S. Nakhaee, A. Amirabadizadeh, G. Akhavanakbari, D.E. Keyler, O. Mehrpour, Prognostic factors in acute aluminium phosphide poisoning: a risk-prediction nomogram approach, Basic Clin. Pharmacol. Toxicol. 123 (2018) 347–355, https://doi.org/10.1111/bcpt.13005.
[53] S.S.V. Kumar, S. Shekhli, A. Jose, Examining the effectiveness of prognostic scoring systems and mortality predictors in aluminum phosphide poisoning: insights from emergency department settings, Emerg. Care J. 20 (2024) 12557, https://doi.org/10.4081/ecj.2024.12557.
[54] G.N. El-Sarnagawy, A.E. Shaban, H.I. Lashin, Validation of corrected and dispersed qt as predictors of adverse outcomes in acute cardiotoxicities, Cardiovasc. Toxicol. 22 (2022) 1–13, https://doi.org/10.1007/s12012-020-09629-8.
[55] A. Wahdan, H. Khalifa, Clinical Data, Laboratory Investigations and electrocardiographic changes as predictors of mortality in acute aluminum phosphide poisoning, Mansoura J. Forensic Med. Clin. Toxicol. 28 (2020) 111–123, https://doi.org/10.21608/MJFMCT.2020.21290.1009.
[56] M. Abd-Allah, A. Abdalla, N. Mohamed, M. Rady, A. Farrag, K. Salama, G. Rakha, Y. Elfakhrany, Updates on toxicology of Aluminum Phosphide and different management protocols, Zagazig Univ. Med. J. 28 (2022) 1176–1183, https://doi.org/10.21608/zumj.2022.125913.2491.
[57] Q. Saif, K. Ruhi, S. Aparna, Aluminium phosphide induced acute kidney injury, Egypt. J. Intern. Med. 27 (2015) 115–117, https://doi.org/10.4103/1110-7782.157999.
[58] F. Fayyaz, Changes in some hematology parameters in poisoning with rice tablet (Aluminum Phosphide), Med. Lab. J. 9 (2015) 43–46, https://doi.org/10.18869/acadpub.mlj.9.4.43.
[59] O. Mehrpour, S. Alfred, S. Shadnia, D.E. Keyler, K. Soltaninejad, N. Chalaki, M. Sedaghat, Hyperglycemia in acute aluminum phosphide poisoning as a potential prognostic factor, Hum. Exp. Toxicol. 27 (2008) 591–595, https://doi.org/10.1177/0960327108096382.
[60] S. Kadamati1, P.B. Potharlanka, R.K. Palaparthi, Lalitha Palaparthi, Acute aluminium phosphide poisoning and hyperglycemia: a possible warning sign for, 14 (2024) 379–382. https://doi.org/10.5530/ijmedph.2024.2.74.
[61] C. Lionte, C. Bologa, I. Agafiti, V. Sorodoc, O.R. Petris, E. Jaba, L. Sorodoc, Association of multiple glycemic parameters at hospital admission with mortality and short-term outcomes in acutely poisoned patients, Diagnostics 11 (2021) 361, https://doi.org/10.3390/diagnostics11020361.
[62] H. Tawfik, Study of the causes of mortality in acute aluminium phosphide poisoning, Mansoura J. Forensic Med. Clin. Toxicol. 26 (2018) 101–111, https://doi.org/10.21608/mjfmct.2018.47196.
[63] O. Mehrpour, M. Abdollahi, M.Davood Sharifi, Oxidative stress and hyperglycemia in aluminum phosphide poisoning, J. Res. Med. Sci. 19 (2014) 196.
[64] S.N. Chugh, S. Ram, A. Sharma, B.B. Arora, A.S. Saini, K.C. Malhotr, Adernocortical involvement in aluminium phosphide poisoning, Indian J. Med. Res. Sect. B Biomed. Res. Other Infect. Dis. 90 (1989) 289–294.

[65] M.Ö. Çolak, A. Erdoğan, M.A. Baydin, C. Afacan, L. Kati, Duran, Epidemiology of organophosphate intoxication and predictors of intermediate syndrome, Turk. J. Med. Sci. 44 (2014) 279–282, https://doi.org/10.3906/sag-1211-31.
[66] H. Sanaei-Zadeh, S.K. Esfeh, N. Zamani, F. Jamshidi, S. Shadnia, Hyperglycemia Is a Strong prognostic factor of lethality in methanol poisoning, J. Med. Toxicol. 7 (2011) 189–194, https://doi.org/10.1007/s13181-011-0142-x.
[67] M. Levine, E.W. Boyer, C.N. Pozner, A.J. Geib, T. Thomsen, N. Mick, S.H. Thomas, Assessment of hyperglycemia after calcium channel blocker overdoses involving diltiazem or verapamil, Crit. Care Med. 35 (2007) 2071–2075, https://doi.org/10.1097/01.CCM.0000278916.04569.23.
[68] O. Mehrpour, A. Aghabiklooei, M. Abdollahi, S. Singh, Severe hypoglycemia following acute aluminum phosphide (rice tablet) poisoning; a case report and review of the literature, Acta Med. Iran. 50 (2012) 568–571.
[69] N. Jamshed, M. Ekka, P. Aggarwal, S. Narayan, Severe hypoglycemia as a presenting feature of aluminum phophide poisoning, Ann. Saudi Med. 34 (2014) 189–190, https://doi.org/10.5144/0256-4947.2014.189.
[70] D.K. Kochar, N. Shubhakaran, B.V. Jain, C.B. Sharma, Meena, Successful management of hypokalaemia related conduction disturbances in acute aluminium phosphide poisoning, J. Indian Med. Assoc. 98 (2000) 461–462.
[71] K. Kjeldsen, Hypokalemia and sudden cardiac death, Exp. Clin. Cardiol. 15 (2010) e96. ⟨https://pmc.ncbi.nlm.nih.gov/articles/PMC3016067/⟩ (accessed November 26, 2024).
[72] Z.Q. Liu, H.S. Wang, Y. Gu, Hypokalemia is a biochemical signal of poor prognosis for acute paraquat poisoning within 4 h, Intern. Emerg. Med. 12 (2017) 837–843, https://doi.org/10.1007/s11739-016-1491-x.
[73] M.W. Chang, S.S. Chang, L.E.E. Chien-Chang, B.F. Sheu, Y.R. Young, Hypokalemia and hypothermia are associated with 30-day mortality in patients with acute paraquat poisoning, Am. J. Med. Sci. 335 (2008) 451–456, https://doi.org/10.1097/MAJ.0b013e318157cb6d.
[74] D. Basnet, Y.L. Shakya, S. Gupta, N.A. Shah, B. Gupta, M. Yadav, Serum potassium level in acute organophosphorus poisoning and its correlation with severity in patients presenting to emergency room in a tertiary hospital, J. Karnali Acad. Heal. Sci. 7 (2024), https://doi.org/10.61814/jkahs.v7i2.943.
[75] F.F. Alamri, D.A. Almarghalani, E.A. Alraddadi, A. Alharbi, H.S. Algarni, O. M. Mulla, A.M. Alhazmi, T.A. Alotaibi, D.H. Beheiry, A.S. Alsubaie, A. Alkhiri, Y. Alatawi, M.S. Alzahrani, A.Y. Hakami, A. Alamri, K. Al Sulaiman, The utility of serum glucose potassium ratio as a predictive factor for haemorrhagic transformation, stroke recurrence, and mortality among ischemic stroke patients, Saudi Pharm. J. 32 (2024) 102082, https://doi.org/10.1016/j.jsps.2024.102082.
[76] G. Dorooshi, S. Samsamshariat, F. Gheshlaghi, S. Zoofaghari, A. Hasanzadeh, S. Abbasi, N. Eizadi-Mood, Comparing sequential organ failure assessment score, acute physiology and chronic health evaluation ii, modified acute physiology and chronic health evaluation ii, simplified acute physiology score ii and poisoning severity score for outcome prediction of, J. Res. Pharm. Pract. 12 (2023) 49–57, https://doi.org/10.4103/jrpp.jrpp_43_23.
[77] A.K. Pannu, L. Jhuria, A. Bhalla, N. Sharma, PGI score: Prospective validation and correlation with SOFA, SAPS-II, and APACHE-II scores for predicting outcomes in acute aluminum phosphide poisoning, Toxicol. Res. 11 (2022) 361–366, https://doi.org/10.1093/toxres/tfac015.
[78] S. Sakr, M. Atef, N. Shalaby, PGI score as a predictor of cardiotoxicity and mortality in patients with acute aluminum phosphide poisoning, Zagazig J. Forensic Med. 0 (0) (2022) 21, https://doi.org/10.21608/zjfm.2022.170264.1130.
[79] G. Yalçın, H. Tunca, B. Sayınbatur, M. Anıl, Predictive Value of complete blood count, venous blood gas measurements, and glucose/potassium ratio for delayed neuropsychiatric syndrome in children with acute carbon monoxide poisoning due to coal-burning stove, Turk. Arch. Pediatr. 58 (2023) 328–335, https://doi.org/10.5152/TurkArchPediatr.2023.22282.
[80] Y.H. Kim, J.H. Yeo, M.J. Kang, J.H. Lee, K.W. Cho, S.Y. Hwang, C.K. Hong, Y. H. Lee, Y.W. Kim, Performance assessment of the SOFA, APACHE II scoring system, and SAPS II in intensive care unit organophosphate poisoned patients, J. Korean Med. Sci. 28 (2013) 1822–1826, https://doi.org/10.3346/jkms.2013.28.12.1822.
[81] E.S. Schwarz, K.T. Kopec, T.J. Wiegand, P.M. Wax, J. Brent, Should we be using the poisoning severity score? J. Med. Toxicol. 13 (2017) 135–145, https://doi.org/10.1007/s13181-017-0609-5.
[82] A.K. Pannu, A. Bhalla, J. Gantala, N. Sharma, S. Kumar, D.P. Dhibar, Glucose-insulin-potassium infusion for the treatment of acute aluminum phosphide poisoning: an open-label pilot study, Clin. Toxicol. 58 (2020) 1004–1009, https://doi.org/10.1080/15563650.2020.1719131.
[83] N. Kalay, I. Ozdogru, A. Gul, Y. Yucel, Y. Cetinkaya, M.T. Inanc, A. Dogan, M. G. Kaya, N.K. Eryol, Effects of intermittent and long-term Glucose-insulin-potassium infusion in patients with systolic heart failure, Exp. Clin. Cardiol. 13 (2008) 85–88. ⟨https://pubmed.ncbi.nlm.nih.gov/19343122/⟩ (accessed January 21, 2025).
[84] A. Ullah, S. Jan, H. Shahzad, M.R. Dar, S. Khan, K. Ahmad, A. Abdullah, M.R. Khan, M. Obaid, M. Khalid, A. Ullah, S. Jan, H. Shahzad, M. Dar, S. Khan, K. Ahmad, A. Abdullah, M.R. Khan, M. Obaid, M. Khalid, Therapeutic effect of glucose-insulin-potassium (GIK) infusion therapy in the treatment of acute aluminum phosphide poisoning: an institutional study, Cureus 16 (2024), https://doi.org/10.7759/CUREUS.76182.
[85] B. Adel, N.M. Elgharbawy, M.M. Shahin, A.A. Abo-Elfadl, K.M. Saad, Insulin-euglycemia therapy in acute aluminum phosphide poisoning: a randomized clinical trial, Clin. Toxicol. 61 (2023) 1032–1039, https://doi.org/10.1080/15563650.2023.2279495.
[86] H. Hassanian-Moghaddam, N. Zamani, Therapeutic role of hyperinsulinemia/euglycemia in aluminum phosphide poisoning, Medicine 95 (2016), https://doi.org/10.1097/MD.0000000000004349.
[87] A. Mohammadinejad, K.S. Rizi, R.K. Oskuee, E. Aryan, Z. Meshkat, A. Ulianas, M. Rezayi, Development of detection methods for the diagnosis and analysis of highly toxic metal phosphides: a comprehensive and critical review, Biotechnol. Appl. Biochem. 69 (2022) 1121–1147, https://doi.org/10.1002/bab.2190.
[88] A. Banagozar Mohammadi, M.Zaare Nahandi, H. Solhi, Utility of silver nitrate test for post-mortem diagnosis of aluminum phosphide poisoning, J. Iran. J. Toxicol. 7 (2013) 804–807.