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Prognostic value of PGI score compared to poison severity score (PSS) and simplified acute physiology score (SAPS) II as predictors of mortality and other adverse outcomes in acute poisoning with aluminum phosphide

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ABSTRACT

Aluminum phosphide (AlP) poisoning is a life-threatening emergency prevalent in the Middle East region including Egypt. Early prediction of prognosis is critical for initiating the utmost intensive interventions. Though many scoring systems were studied for predicting the prognosis of AlP poisoning, these scores received wide criticism. Complexity and reliability were the main concerns. Therefore, this retrospective cross-sectional study aimed to evaluate the performance of the recently introduced PGI score as a predictor of case fatality, the need for mechanical ventilation and vasopressor therapy in acute AlP poisoning. Moreover, it compares the performance of PGI with the known poison severity score (PSS), and the simplified acute physiology score (SAPS) II. Among 144 exposed patients, we reported a mortality rate of 61.1%. Non-survivors exhibited significantly higher PGI, PSS, and SAPS II than survivors. Though the PGI, PSS, and SAPS II proved their significance as predictors of mortality and, the need for MV and vasopressors, the PGI score showed a significantly higher area under the curve (AUC) as a predictor of MV (AUC = 0.848) compared to PSS (AUC = 0.731) and SAPS II (AUC = 0.749). Additively, PGI of 2 or more was a significant predictor of mortality (AUC = 0.831, sensitivity = 65.9%, and specificity = 89.3 %) and MV ($p < 0.001$), while PGI of 1 or more was another predictor of vasopressor need (AUC = 0.881, sensitivity = 89.0% and specificity = 79.4%). Given the PGI score's high AUCs across all outcomes, coupled with its balanced sensitivity and specificity, the PGI score could be a simple, and robust tool replacing the PSS and SAPS II for predicting mortality, clinical decision-making including the need for MV and vasopressor therapy in acute AlP exposure. Adopting the PGI score seems substantially useful in managing acute AlP poisoning, notably in resource-restricted countries.

1. Introduction

Acute pesticide poisoning is considered a long-lasting serious public health problem, accounting for more than 11,000 annual deaths worldwide. This number represents only the deaths due to unintentional exposure [1]. For decades, aluminum phosphide (AlP) has been widely used as a grain rodenticide especially in developing countries [2,3]. Acute AlP poisoning constitutes frequent emergency admissions in developing countries [4,5]. The seriousness of AlP poisoning symptoms, and the lack of antidote therapy result in a high mortality ranging from 30% to 80%. Nonetheless, the early supportive management is the ideal lifesaving solution [5–7].

AlP is available as rounded discs, rice tablets" or "wheat bills" used to fumigate the grains, mainly the wheat grains, where ingestion constitutes the most common route to exposure [8]. Exposure of AlP to moisture in the stomach yields the phosphine gas, which is promptly absorbed into the circulation. Phosphine gas targets the mitochondrial respiratory enzymes at the cellular level and inhibits the cytochrome C oxidase, leading to ADP depletion and alteration in the mitochondrial membrane potential, as well as electron leakage [9]. The electron leakage promotes the formation of free radicals and depletes the antioxidant cellular stores. Subsequently, lipid peroxidation, DNA damage, and even apoptotic cell death can supervene [5].

Regarding the clinical presentation of AlP poisoning, nausea and

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vomiting are the early pronounced symptoms classically described in almost all exposed patients [10]. Moderate and severe toxicity presented with resistant cardiovascular collapse, cardiogenic shock, respiratory failure and metabolic acidosis [8,11]. Early deaths in acute AlP poisoning within the first 24 h of exposure are typically due to cardiovascular failure. Nonetheless, delayed deaths are attributed to respiratory failure, and other target organ damage. Therefore, early discrimination of patients at risk primarily depends on the rapid identification of cardiac affection [5,12].

The prognosis of AlP toxicity depends on many factors, including circumstances of exposure, clinical and laboratory features on admission, and management-related factors. The exposure related factors may not be accurately reported making these factors unreliable in stratifying the patients or predicting outcomes [13]. Early prediction of the severity of AlP toxicity is the most critical factor guiding the initiation of appropriate interventions and allowing better allocation of the limited therapeutic resources [14,15].

Previous studies investigated several scoring systems including the poison severity score (PSS) [16], acute physiology and chronic health evaluation II and sequential organ failure assessment scores and reported their significance as predictors of adverse outcomes in acute AlP poisoning [4,17]. Nonetheless, these factors were criticized for being time-consuming and of a complex nature. They require several clinical and laboratory data for calculation, which is inapplicable in an emergency clinical context [18]. So, adopting a reliable score of simple nature and good predictive power reflecting the severity of poisoning following exposure to AlP is an urgent need rather than a luxury.

Pannu et al. investigated multiple toxidrome-specific parameters, and recognized that blood pH, Glasgow coma scale (GCS), and systolic blood pressure (SBP) were the most reliable predictors of fatality in acute AlP poisoning. They nominated this score as the PGI score and recommended assessing the discrimination of PGI using a large cohort and comparing this score with other prognostic scores [19,20]

To our knowledge, very few studies investigated the efficiency of using PGI score as a predictor in poisoning with AlP [10,21]. These studies focused on mortality and did not evaluate the efficacy of using the PGI as an outcome predictor compared to PSS, which is the well-known score adopted in acute toxic exposure. Therefore, and given the resource-restricted nature of developing countries, including Egypt, the current study aimed to investigate if using the PGI score might fulfil the urgent need for triaging and medical decision-making through evaluating the ability of PGI score to predict the mortality, need for mechanical ventilation (MV) and vasopressor therapy in acute AlP poisoning. Furthermore, to compare the performance of the PGI score with other the two commonly used scores in acute poisoning, the PSS and the simplified acute physiology score (SAPS) II.

2. Patients and methods

2.1. Study setting and design

The current study was a retrospective cross-sectional study conducted using medical records of patients admitted to Tanta University Poison Control Center (TUPCC) from January to December 2022.

2.2. Sampling and sample size

Convivence sampling allowed us to enrol all patients who met the inclusion criteria. The sample size was calculated using the OpenEpi software version 3.01 [22]. Hypothesizing 89.04 % frequency of mortality due to exposure to AlP in Egyptian population [10], and adopting a margin of approximately 0.051 with a 95% confidence interval (CI), the minimum required sample size was estimated to be no less than 144 patients.

2.3. Inclusion and exclusion criteria

The inclusion criteria included confirmation of oral poisoning with AlP in adults aged 18 years or above in a period of no more than 24 hr. Diagnosis of AlP poisoning was made through the history taken from the patients themselves and/or the relatives, who seldomly brought the container. All exposed adult patients were considered eligible regardless of the manner of exposure (intentional to attempt suicide, unintentional, or undetermined). Confirmation of diagnosis was achieved through the characteristic clinical signs and symptoms, which were not explained by pathological conditions or other types of toxic exposure.

Exclusion criteria included patients under 18 or over 60 years, those with query diagnoses, patients suffering from co-morbid conditions, and patients with incomplete medical records. Excluding elderly patients with co-morbid conditions reduced the bias in calculating the scores, where many parameters might be influenced by the aging process and co-morbid conditions [23]. Besides, the reported significant age-related alteration in pharmacokinetics and pharmacodynamics of the ingested substances and the pronounced hepatic and renal affection affect drug clearance [24]. Co-ingestion and drug-drug interactions, which are classically more described in elderly adults, were other reasons behind excluding this category of exposed patients [25]. Furthermore, we excluded all patients who arrived later than 24 h after exposure, those who received treatment in other hospitals, patients with co-ingestions, and patients who died on arrival. Fig. (1) explains the process of patient recruitment.

2.4. Ethical considerations

This study was performed in agreement with applicable laws and regulations, good clinical practices, and ethical principles. Before conducting the study, the study proposal was approved by the Institutional Review Board (IRB) at Faculty of Medicine, Tanta University (approval number 36264PR535/2/24). The IRB exempted obtaining informed consent due to the study's retrospective nature. However, the confidentiality of the studied patients was preserved through anonymizing the data.

2.5. Data collection

2.5.1. Demographics and individual parameters

Data were collected in a predesigned case report form, including the demographics (age and sex) and the delay time from exposure until receiving the emergency treatment. Besides, we have reported the vital signs including the heart rate, and systolic and diastolic blood pressure (DBP). Single parameters constituting the PGI and SAPS II were reported, including the GCS, in addition to the patient's need for MV and vasopressor therapy.

2.5.2. Scoring calculated on admission

Three scoring were calculated as follows:

i. PGI score: The PGI score consists of 3 variables (pH $<$ 7.25, GCS $<$ 13, and impaired SBP $<$90 mmHg), where every parameter is scored with a single point. Scores ranged between 0 and 3, where high scores demonstrating severe toxidrome [19,20].
ii. PSS: The original PSS assesses 12 areas, including the gastrointestinal tract, respiratory system, nervous system, cardiovascular system, metabolic balance, liver, kidney, blood, and muscular system, Local effects on the skin, Local effects on the eye, and local effects from bites and stings. Each area is given a score (0−4), where 0 refers to no signs or symptoms, score 1 stands for mild, transient, and spontaneously resolving symptoms or signs, score 2 means Pronounced or prolonged symptoms or signs, score 3 describes severe or life-threatening symptoms or signs, and 4 describes the death. The patient is scored according to the most

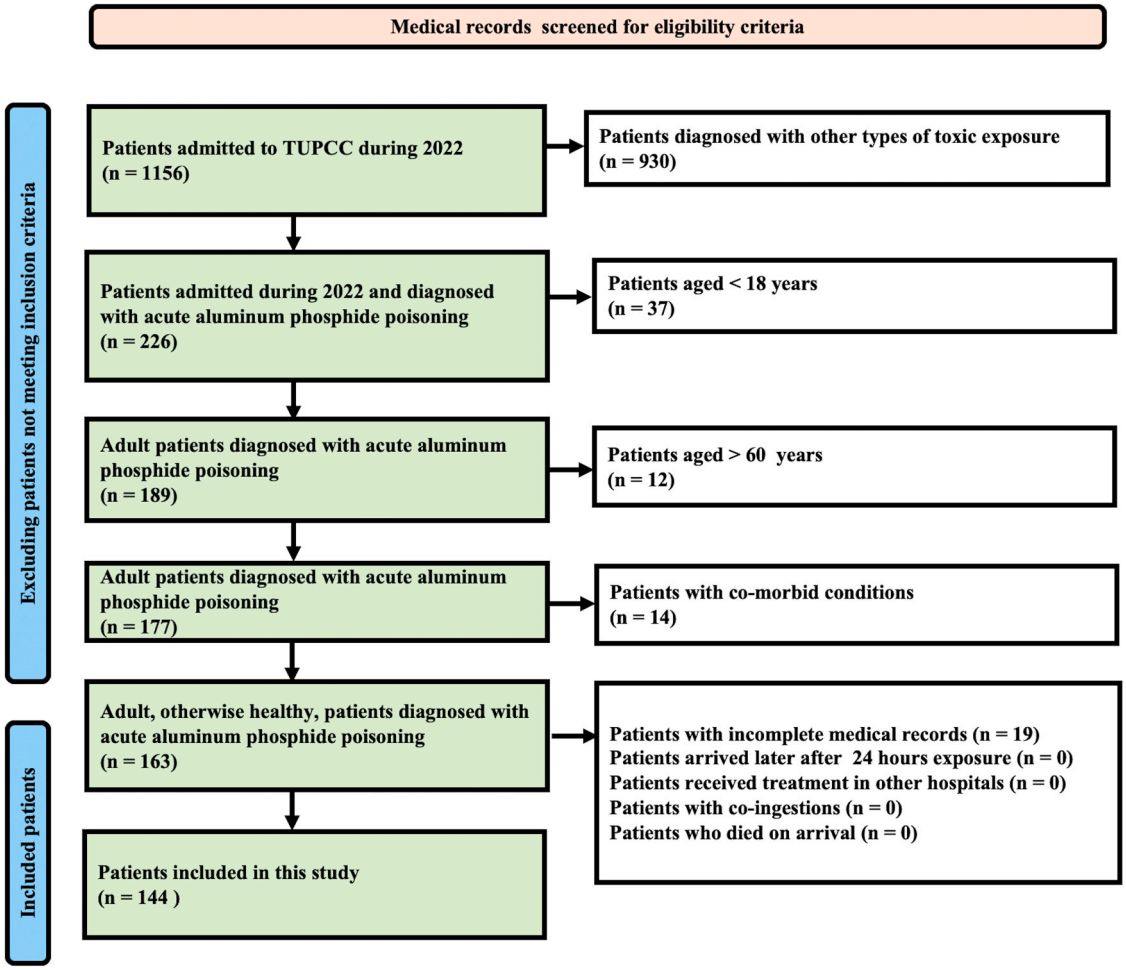

Fig. (1). Flow chart illustration of the process of recruiting the patients included in the current study.

severe clinical sign or symptom in any area. PSS doesn't consider only the signs and symptoms but also other observed parameters, including the amount of drug ingested, serum concentrations of a drug, radiological findings, ECG findings, arterial blood gas analysis, liver and renal function tests, and other laboratory investigations [26]. Every patient was given a grading score describing the severity of the poisoning, where patients with asymptomatic presentation in all areas were given a score of 0, those showing mild severity were given a score of 1, a score of 2 described patients of moderate severity, and a score of 3 described patients with severe poisoning. Patients of PSS grade 4, described as those presented with fatal poisoning were excluded [27].

iii. SAPS II: SAPS II was also calculated based on 17 variables including physiology variables (heart rate, SBP, PaO_2 (arterial oxygen tension)/FiO_2 (fraction of inspired oxygen) ratio, body temperature in Celsius, serum sodium and potassium, serum bicarbonate (HCO_3), Blood urea nitrogen, serum bilirubin, white blood cells (WBCs) count, urinary output, and GCS), age, type of admission and three underlying disease variables. Scores range from 0 to 163, with higher scores indicating more severe disease [23]. However, according to the inclusion criteria, all patients scored zero regarding the type of admission and the three underlying disease conditions.

2.5.3. Therapeutic regimens

Upon admission, all patients received supportive medical care preserving the airway, breathing, and circulation. The management of AlP poisoning was tailored according to TUPCC protocols adopted from the international guidelines, where the supportive treatment was the main therapy [28]. Vasopressors and judicious use of intravenous fluids were given whenever needed. Patients with respiratory failure or a significant need for high-volume oxygen were admitted to the intensive care unit and mechanically ventilated. All patients were followed up throughout their hospital stay, where supportive treatment was delivered.

2.6. Grouping and outcomes

As the primary outcome was the prediction of mortality, the studied patients were categorized into two groups according to their survival including survivors and non-survivors. Other two secondary outcomes were investigated including the need for MV and vasopressor therapy.

2.7. Data analysis

Data was analyzed using the Statistical Package for Social Sciences (IBM SPSS Statistics) version 26 (IBM Corp., Armonk, NY, USA). Number and proportions were used to express categorical variables. Pearson's chi-square test was used to assess the association between the investigated outcomes and categorical variables. Regarding the quantitative variables, mean ± standard deviation (SD) and independent samples t-test were used to describe these variables and compare them among different outcomes. Abnormally distributed data were presented as the median and interquartile range (IQR) (expressed as the 25th–75th percentiles) where the Mann–Whitney U test was used to analyse these variables.

Additionally, receiver operating characteristics (ROC) curves were created for the parameters that proved their high significance in the baseline analysis ($p < 0.001$) and for the studied scores regarding the three studied outcomes. The area under the curve (AUC) was interpreted as follows: 0.90 – 1 = excellent; 0.80–< 0.90 = good; 0.70 – < 0.80 = fair; and 0.60 – < 0.70 = poor. Pair-wise comparisons were made between the AUCs of the studied scores according to the method described by DeLong et al. [29]. ROC curve analysis reported the sensitivity, specificity, positive and negative predictive values (PPV, NPV), and positive and negative likelihood ratios. Binary logistic regression was conducted to assess the discriminatory power of the studied scores as mortality predictors. Hosmer and Lemeshow test was run to assess the goodness-of-fit of a logistic regression model.

To assess the reliability of the obtained findings regarding changes in model assumptions. Sensitivity analysis was conducted by applying an alternative statistical model in the form of univariate regression to the dataset to assess the significance of the three studied scores previously identified by the original mode as mortality predictors.

3. Results

The present study commenced by screening 1156 medical records of patients admitted to TUPCC during the study period. We found 226 patients, representing the total number of admitted patients to TUPCC during 2022, who were diagnosed with acute AlP poisoning. These records were reviewed precisely to assess their eligibility. After excluding those who did not fulfil the inclusion criteria, we ended up with 144 patients (Fig. 1).

The studied patients showed a mean age of 28 years and a mortality rate of 61.1%. Females constituted 61.1 % of the studied patients, and there were no significant sex variations between the studied patients. Survivors showed relatively higher ages (mean = 29.36 years) than survivors (mean = 25.86 years). However, the observed age variations did not reach statistical significance ($p = 0.052$). Nevertheless, all studied patients were intentionally exposed in trials to attempt suicide. We did not encounter patients exposed unintentionally. Toxidrome features upon admission included metabolic acidosis (n=122, 84.7%), hypoxemia [PaO_2/FiO_2 ratio < 400] (n=106, 73.6%), hypotension [SBP < 90] (n= 100, 69.4%) and severe altered mental status [GCS ≤ 8] (n= 114, 79.2 %). Non-survivors showed significantly more frequent metabolic acidosis (95.4% versus 67.8%), hypoxemia (83% versus 17%), hypotension (78% versus 22%), and severe alteration in consciousness level (88.6% versus 64.2%) compared to survivors ($p < 0.001$).

As Table (1) shows, compared to survivors, the non-survivors had significantly lower median of SBP (60 versus 90 mmHg), DBP (30 versus 60 mmHg), PaO_2/FiO_2 ratio (140.0 versus 450.3), urine output (1332 versus 1980 mL/first 24 hr.) and GCS (12 versus 14) and ($p < 0.001$). The body temperature was significantly lower among non-survivors who showed a mean temperature of 36.79°Celsius compared to survivors (mean = 36.94°Celsius) ($p < 0.05$). Analysing laboratory investigations showed that non-survivors had a significantly lower serum K level, pH, and HCO_3 than survivors ($p < 0.001$). However, there were no significant differences between survivors and non-survivors regarding heart rate, blood urea nitrogen, serum Na level, bilirubin, and leukocytic count. Non-survivors were in significant need of vasopressors and MV than survivors ($p < 0.001$). Nonetheless, vasopressor support was given in (n= 110, 76.4%), and invasive MV was initiated in (n= 79, 54.9%) within the first 24 hr. of admission.

The means and medians PSS, PGI, and SAPS II were significantly higher in non-survivors than in survivors ($p < 0.001$). About 91.7% of patients with a PGI score of 3 died (n=24), compared with 90% (n =36/40) of those with a score of 2, 53.7% (n=22/41) with a score of 1, and 20.5% (n= 8/39) of patients with score 0 as shown at Table (2).

The ROC curve analyses of the potential individual predictors of mortality, MV, and vasopressor therapy showed that the SBP, PaO_2/FiO_2 ratio, potassium level, pH, bicarbonate level, urinary output, and GCS

Table. 1
Baseline characteristics, demographic, predicting parameters and some therapeutic agents of the studied patients at time of admission according to the survival.

Variables	Total (No = 144)	Survivors (No = 56)	Non-survivors (No = 88)	P value
Sex	**No (%)**	**No (%)**	**No (%)**	0.533[b]
Male	56 (38.9)	20 (35.7)	36 (40.9)	
Female	88 (61.1)	36 (64.3)	52 (59.1)	
	Median (IQR) Mean ± SD	**Median (IQR) Mean ± SD**	**Median (IQR) Mean ± SD**	
Age (years)	23 (18 – 31.8) 28.00 ± 12.87	20 (18 – 30) 25.86 ± 11.09	25 (20 – 36.5) 29.36 ± 13.77	0.052[a]
Delay time (hr.)	2 (1 – 4) 2.68 ± 1.93	2.25 (1.0 – 4.0) 2.69 ± 2.18	2.00 (1.0 – 4.0) 2.68 ± 1.76	0.739[a]
Heart rate (bpm)	89.5 (70 – 105) 88.60 ± 22.73	99 (86 – 112) 97.36 ± 15.66	80 (60–102) 83.02 ± 24.76	0.331[a]
Systolic blood pressure (mmHg)	70 (40 – 90) 72.29 ± 33.50	90 (80 – 120) 93.57 ± 31.24	60 (30 – 79) 58.75 ± 27.39	< 0.001 *[a]
Diastolic blood pressure (mmHg)	40 (20 – 60) 44.31 ± 22.80	60 (43 – 78) 58.21 ± 20.37	30 (20 – 50) 35.45 ± 19.71	< 0.001 *[a]
PaO_2/FiO_2	214 (114.4 – 417.2) 274.13 ± 185.25	450.3 (392.2 – 515.6) 472.69 ± 125.19	140.00 (88.8 – 185.3) 147.78 ± 70.53	< 0.001*[a]
Temperature (°C)	37 (36 – 37) 36.85 ± 0.36	37 (36.6–37.0) 36.94 ± 0.39	37 (36.6 – 37.0) 36.79 ± 0.33	0.007*[a]
Serum Na (mEq/L)	142 (138.0 – 144.0) 141.63 ± 6.05	142.0 (138.1 – 144.0) 142.43 ± 5.69	142.0 (138.0 – 144.3) 141.12 ± 6.24	0.856[a]
Serum K (mEq/L)	3.61 (3.18 – 4.02) 3.60 ± 0.54	3.8 (3.47 – 4.28) 3.86 ± 0.49	3.4 (2.9 – 3.8) 3.43 ± 0.51	< 0.001*[a]
Blood pH	7.35 (7.28 – 7.44) 7.34 ± 0.12	7.40 (7.32 – 7.45) 7.39 ± 0.08	7.30 (7.23 – 7.41) 7.31 ± 0.12	< 0.001*[a]
HCO_3 level (mEq/L)	14.0 (10.38–17.55) 14.0 ± 5.60	17.80 (13.10–21.21) 17.5 ± 5.90	12.30 (9.63–14.28) 11.7 ± 4.00	< 0.001*[c]
Blood urea nitrogen (mg/dL)	16.1 (13.2 – 20.6) 18.45 ± 9.17	15.9 (13.5 – 19.1) 19.09 ± 12.02	16.8 (12.1 – 20.5) 18.04 ± 6.83	0.681[a]
Serum bilirubin (mg/dL)	0.3 (0.2 – 0.4) 0.32 ± 0.09	0.31 (0.22 – 0.40) 0.29 ± 0.09	0.30 (0.20 – 0.41) 0.34 ± 0.10	0.0857[a]
WBCs /mm^3	8050 (5225 – 11275) 8523.47 ± 3665.71	9450 (5800 – 11350) 8974.64 ± 3201.75	6800 (4950 – 11100) 8236.36 ± 3923.23	0.0863[a]
Urine output (mL/first 24 hr.)	1584 (636 – 2022) 1470.33 ± 673.86	1980 (1608–2280) 1861.71 ± 587.25	1332 (564–1716) 1221.27 ± 605.81	< 0.001 *[a]
GCS	13 (11–14) 12.61 ± 1.83	14 (13 – 15) 14.07 ± 0.85	12 (10 – 13) 11.75 ± 1.74	< 0.001 *[a]
	No (%)	**No (%)**	**No (%)**	
Vasopressor therapy	110 (100 %)	22 (20 %)	88 (80 %)	< 0.001*[b]
Mechanical ventilation	79 (100 %)	6 (7.6 %)	73 (92.4 %)	< 0.001*[b]

IQR: interquartile range, No: number, SD: standard deviation,
* significance at $p < 0.05$.
[a] Mann-Whitney U test,

[b] Pearson's Chi-square test for independence of observations,
[c] independent samples t-test.

Table. 2
Comparison between survivor and non-survivor regarding PGI, SAPS II, and PSS scores at admission.

Scoring System	Total (No = 144)	Survivors (No = 56)	Non-survivors (No = 88)	P value
	Median (IQR) Mean ± SD	Median (IQR) Mean ± SD	Median (IQR) Mean ± SD	
PGI score	1 (0–2) 1.34 ± 1.05	0 (0–1) 0.43 ± 0.50	2 (1 – 2.75) 1.82 ± 0.92	< 0.001[*a]
PSS	3 (2 – 3) 2.47 ± 0.69	2 (1.25 – 2) 1.96 ± 0.68	3 (3 – 3) 2.80 ± 0.46	< 0.001[*a]
SAPS II	27.5 (20.0 – 41.0) 30.69 ± 14.09	21.50 (13.3 – 26.0) 22.15 ± 11.47	36.5 (27.0 – 44.5) 36.12 ± 12.91	< 0.001[*a]
	No (%)	No (%)	No (%)	
PGI	39 (100 %) 41 (100 %)	31 (79.5 %) 19 (46.3 %)	8 (20.5 %) 22 (53.7 %)	< 0.001[*b]
• PGI score = 0	40 (100 %) 24 (100 %)	4 (10 %) 2 (8.3 %)	36 (90 %) 22 (91.7 %)	
• PGI score = 1				
• PGI score = 2				
• PGI score = 3				
PSS	16 (100 %) 44 (100 %)	14 (87.5 %) 30 (68.2 %)	2 (12.5 %) 14 (31.8 %)	< 0.001[*b]
• Mild	84 (100 %)	12 (14.3 %)	72 (85.7 %)	
• Moderate				
• Severe				

IQR: interquartile range, No: number,
[*] significance at $p < 0.05$.
[a] Mann-Whitney U test,
[b] Pearson's Chi-square test for independence of observations

were significant predictors of mortality, need for MV and need for vasopressor therapy ($p < 0.001$). The level of hypoxia indicated by the PaO_2/FiO_2 ratio showed the highest AUC (0.994 and 0.943) among predictors of mortality and vasopressor therapy, respectively. SBP < 80 was a significant predictor of vasopressor therapy (AUC = 0.914). Other cutoffs and their sensitivities and specificities are shown in Table (3).

As Table (4) shows, the ROC curve analyses of the studied scores as predictors of mortality, need for MV, and vasopressor therapy showed that the PGI score demonstrated high accuracy across all outcomes with particularly strong specificity for predicting all outcomes (89.3%, 87.7%, and 79.4%, for mortality, need for MV, and vasopressor therapy; respectively). The PGI score of more than one significantly predicted the mortality and the need for MV, showing the highest AUC among the other studied scores (0.831 and 0.848, respectively).

Fig. (2A and 2B) shows the ROC curves of potential individual predictors of mortality with their AUC, including SBP (0.813), PaO_2/FiO_2 ratio (0.994), potassium level (0.728), pH (0.701), bicarbonate level (0.787), urinary output (0.774), and GCS (0.852). Fig. (2C) also demonstrates the ROC curve analyses of the three assessed scores, showing comparable performance as mortality predictors. As Table (4) illustrates, the PPV of PGI as a mortality predictor was 90.6%, and the NPV was 62.5%, suggesting that a high PGI score is a strong indicator of mortality, although a low score does not entirely rule out the risk. In comparison, the PSS score had an AUC of 0.816 (95% CI: 0.74–0.88) with a cutoff of > 2, demonstrating good accuracy. The sensitivity was higher at 81.3%, indicating better identification of patients who died. However, the specificity was slightly lower at 78.6%, suggesting a higher rate of false positives than the PGI score. The SAPS II score, with an AUC of 0.79 (95 % CI: 0.72–0.86) and a cutoff of > 26, had a sensitivity of 77.3% and a specificity of 78.6 %, indicating a balanced but slightly less accurate performance compared to PGI and PSS.

For predicting the need for MV, Fig. (3A and 3B) shows that a SBP (< 70 mmHg), PaO_2/FiO_2 ratio (< 292), potassium level (< 4 mEq/L), pH (< 7.31), bicarbonate level (< 15.5 mEq/L), urinary output (< 1344 mL/first 24 hr.), and GCS (< 12) were significant predictors of MV showing AUCs above 0.7. Moreover, as also shown in Fig. (3), the PGI score showed a significantly higher AUC (0.848) as a predictor of MV

Table. 3
Receiver operating characteristics (ROC) curve analyses comparing the accuracy of individual parameters in predicting mortality, the need for mechanical ventilation and vasopressor therapy among the studied patients.

	AUC	95 %CI of AUC	Cut-off	Sensitivity %	Specificity %	PPV %	NPV %	+ LR	- LR	p-value
	Predictors of mortality									
SBP (mmHg)	0.813	0.742–0.869	<75	75.0	82.1	86.8	67.6	4.20	0.30	< 0. 001*
PaO_2/FiO_2 ratio	0.994	0.961–1.002	<292	97.7	96.4	97.7	96.4	27.36	0.024	< 0. 001*
K level (mEq/L)	0.728	0.641–0.792	<3.3	45.5	89.3	87.0	51.0	4.24	0.61	< 0. 001*
pH	0.701	0.691–0.768	<7.31	54.5	78.57	80.0	52.4	2.55	0.58	< 0. 001*
HCO_3 level (mEq/L)	0.787	0.712–0.852	<15.5	88.6	67.9	81.2	79.2	2.76	0.17	< 0. 001*
UOP (mL/first 24 hr.)	0.774	0.691–0.843	<1776	81.82	67.86	80.0	70.4	2.55	0.27	< 0. 001*
GCS	0.852	0.778–0.901	<12	65.9	96.4	96.7	64.3	18.45	0.35	< 0. 001*
	Predictors of the need for mechanical ventilation									
SBP (mmHg)	0.785	0.709–0.849	< 70	74.6	76.9	79.7	71.4	3.24	0.33	< 0. 001*
PaO_2/FiO_2 ratio	0.862	0.795–0.914	< 292	91.1	75.3	81.8	87.5	3.70	0.12	< 0. 001*
K level (mEq/L)	0.699	0.617–0.772	< 4	87.3	43.0	65.1	73.7	1.53	0.29	< 0. 001*
pH	0.737	0.657–0.807	< 7.31	62.0	83.0	81.7	64.3	3.67	0.46	< 0. 001*
HCO_3 level (mEq/L)	0.785	0.709–0.849	< 15.5	92.4	64.6	76.0	87.5	2.61	0.12	< 0. 001*
UOP (mL/first 24 hr.)	0.751	0.672–0.819	< 1344	55.7	87.7	84.6	62.0	4.53	0.51	< 0. 001*
GCS	0.843	0.773–0.898	<12	69.6	92.3	91.7	71.4	9.05	0.33	< 0. 001*
	Predictors of the need for vasopressor therapy									
SBP (mmHg)	0.914	0.851–0.948	<80	85.4	82.3	94.0	63.6	4.84	0.18	< 0. 001*
PaO_2/FiO_2 ratio	0.943	0.890–0.971	<405	92.6	82.3	94.4	77.3	5.25	0.08	< 0. 001*
K level (mEq/L)	0.772	0.692–0.833	<3.7	70.64	76.47	90.6	44.8	3.00	0.38	< 0. 001*
pH	0.725	0.639–0.791	<7.3	47.71	94.12	96.3	36.0	8.11	0.56	< 0. 001*
HCO_3 level (mEq/L)	0.841	0.771–0.892	<17.9	92.7	70.6	91.1	75.0	3.15	0.10	< 0. 001*
UOP (mL/first 24 hr.)	0.878	0.813–0.918	<2112	92.66	64.71	89.4	73.3	2.63	0.11	< 0. 001*
GCS	0.886	0.821–0.932	<13	83.49	88.24	95.8	62.5	7.10	0.19	< 0. 001*

AUC: area under curve, CI: confidence interval, NPV: negative predictive value, PPV: positive predictive value, + LR: positive likelihood ratio, -LR: negative likelihood ratio,
[*] significance at $p < 0.05$, UOP: urine output.

Table. 4
Receiver operating characteristics (ROC) curve analyses comparing the accuracy of PGI, PSS and SAPS II in predicting mortality, the need for mechanical ventilation and vasopressor therapy among the studied patients.

Score	AUC	95 %CI	Cut-off	Sensitivity %	Specificity %	PPV %	NPV %	+ LR	- LR	p-value
	Prediction of mortality									
PGI	0.831	0.760–0.889	> 1	65.9	89.3	90.6	62.5	6.15	0.38	< 0. 001*
PSS	0.816	0.743–0.875	> 2	81.3	78.6	85.7	73.3	3.82	0.23	< 0. 001*
SAPS II	0.794	0.723–0.860	> 26	77.3	78.6	85.0	68.7	3.61	0.29	< 0. 001*
Pairwise comparison of AUC	PSS ~ PGI (p = 0.698) PGI ~ SAPS II (p = 0.267) PSS ~ SAPS II (p = 0.548)									
	Prediction of the need for mechanical ventilation									
PGI	0.848	0.771–0.902	> 1	70.9	87.7	87.5	71.2	5.7	0.33	< 0. 001*
PSS	0.731	0.652–0.801	> 2	77.2	64.6	72.6	70.0	2.1	0.35	< 0. 001*
SAPS II	0.749	0.689–0.821	> 26	75.9	69.2	75.0	70.3	2.5	0.35	< 0. 001*
Pairwise comparison of AUC	PGI~PSS (p = 0.004*) PGI~SAPA II (p = 0.001*) PSS~SAPS II (p = 0.615)									
	Predictors of the need for vasopressor therapy									
PGI	0.881	0.812–0.923	> 0	89.0	79.4	93.3	69.2	4.32	0.14	< 0. 001*
PSS	0.855	0.778–0.901	> 2	73.4	88.2	95.2	50.8	6.24	0.30	< 0. 001*
SAPS II	0.896	0.831–0.939	> 26	70.64	94.12	97.5	50.0	12.01	0.31	< 0. 001*
Pairwise comparison of AUC	PGI~PSS (p = 0.59). PGI~SAPA II (p = 0.659) PSS~SAPS II (p = 0.2006)									

AUC: area under curve, CI: confidence interval, PPV: positive predictive value, NPV: negative predictive value, + LR: positive likelihood ratio, -LR: negative likelihood ratio,
* significance at $p < 0.05$

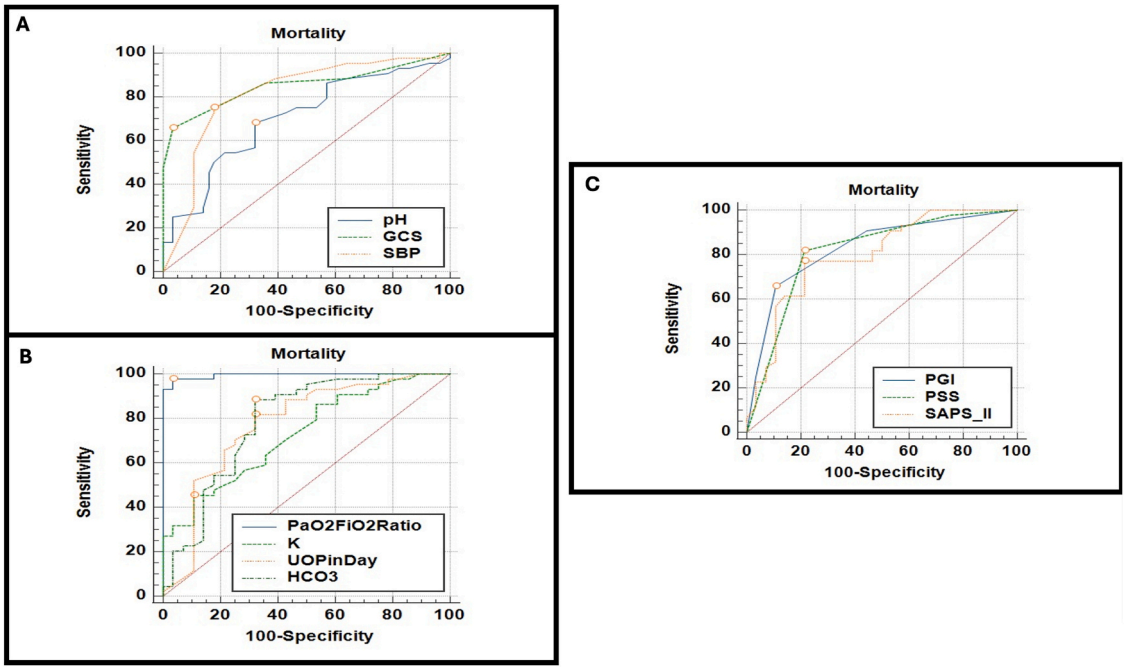

Fig. (2). Receiver operating characteristics (ROC) curve analyses of individual parameters, PGI, PSS and SAPS II as mortality predictors.

compared to the PSS (AUC = 0.731) and SAPS II (AUC = 0.749). Table (4) depicts that a PGI score above one could predict the need for MV with a sensitivity was 70.9 % indicating a moderate ability to correctly identify patients requiring ventilation. However, the specificity was higher at 87.7%, which indicates that the PGI score effectively identifies patients who do not need MV, thus reducing false positives. In contrast, the PSS showed significantly lower accuracy than PGI with an AUC of 0.731 (p= 0.004) and lower specificity (64.6%), which suggests less reliability for this outcome. Furthermore, the SAPS II showed significantly lower performance than PGI (AUC 0.749, p= 0.001).

For predicting the need for vasopressor therapy, Fig. (4A and 4B) shows other independent predictors of vasopressor therapy including SBP (< 80 mmHg), PaO_2/FiO_2 ratio (< 405), potassium level (< 3.7 mEq/L), pH (< 7.30), bicarbonate level (< 17.9 mEq/L), urinary output (< 2112 mL/first 24 hr.), and GCS (< 13). All these predictors showed very good AUCs ranging between 0.841 and 0.943. Additionally, Fig. (4C) highlights the comparable performance of PGI score, PSS and SAPS II as discriminators for the need for vasopressors. The PGI score demonstrated outstanding accuracy with an AUC of 0.881 (95% CI: 0.81–0.92). The PSS and SAPS II scores also performed well, with AUCs of 0.855 and 0.896, respectively. As Table (4) shows, at cut-off of > 0, a sensitivity of 89.0%, and a specificity of 79.4%, the PGI score exhibited a high ability to correctly identify both patients needing vasopressors and those who do not. This balance ensures that the PGI score is both a

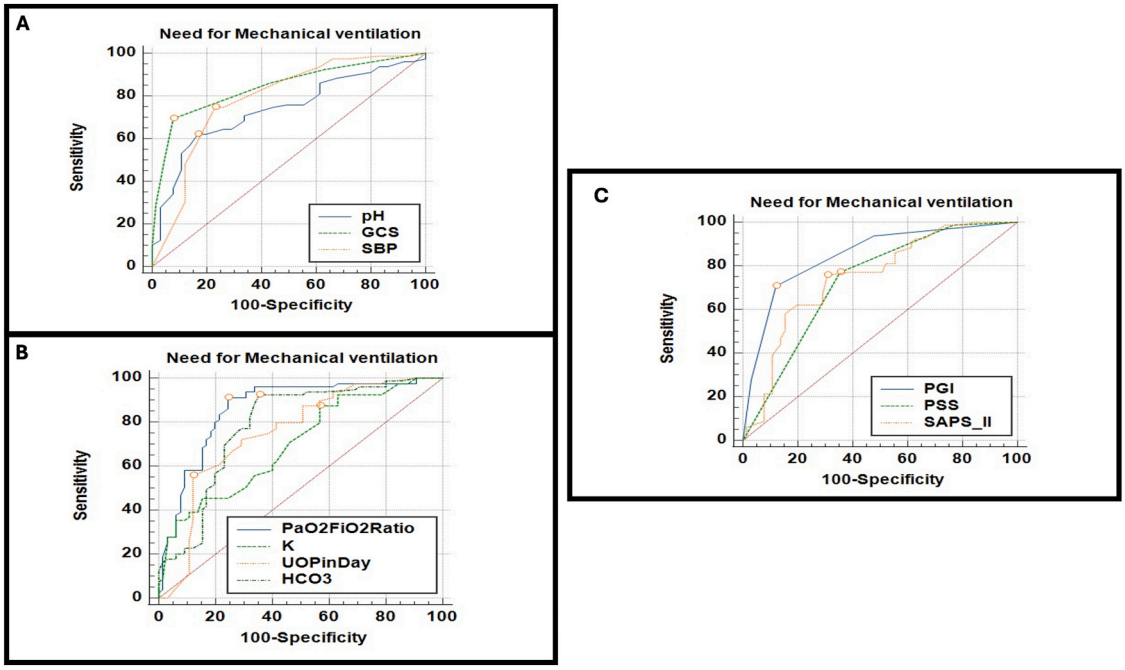

Fig. (3). Receiver operating characteristics (ROC) curve analyses of individual parameters, PGI, PSS and SAPS II as mechanical ventilation predictors.

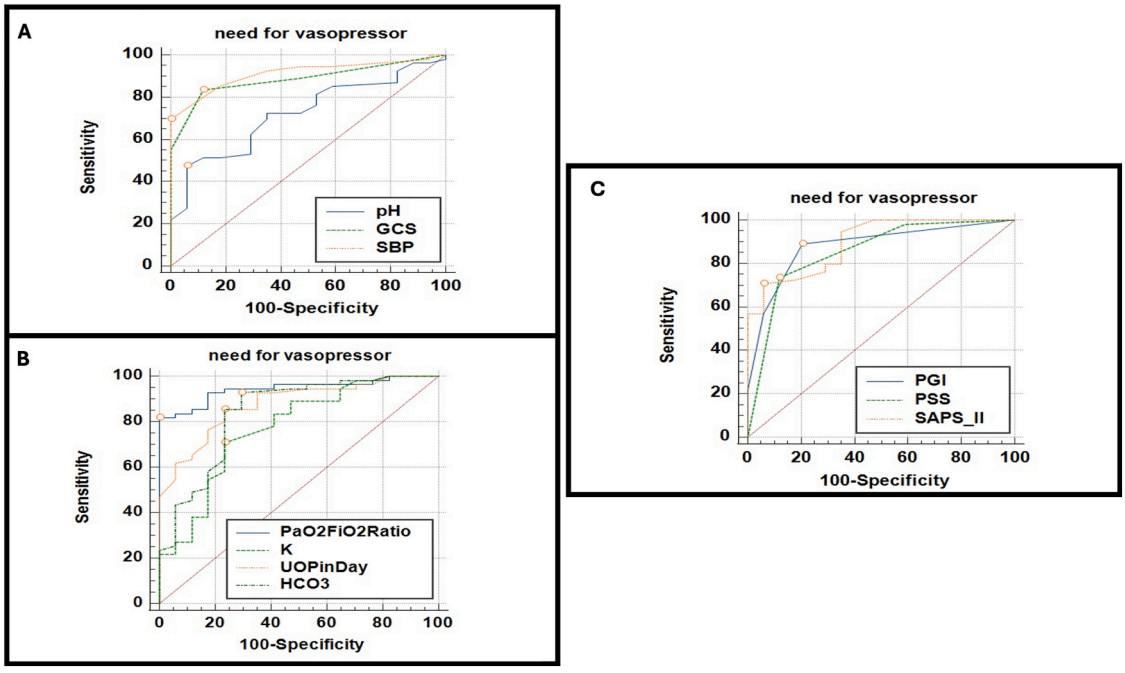

Fig. (4). Receiver operating characteristics (ROC) curve analyses of individual parameters, PGI, PSS and SAPS II as vasopressor therapy predictors.

reliable predictor and an effective tool for clinical decision-making. However, the SAPS II had the highest specificity (94.1%), suggesting it is particularly effective in ruling out patients who do not require vasopressor therapy.

Table (5) demonstrates the Hosmer-Lemeshow test and the sensitivity analyses using univariate analysis for PGI, PSS, and SAPS II. The R^2 was 44% for the PGI, compared to 43% and 32.2% for PSS and SAPS II, respectively. Moreover, regarding the PGI score categories, the odds were 4.487, 34.875, and 42.625 for PGI scores 1, 2, and 3 by taking PGI score 0 as a reference. For the PSS categories, taking the mild score as the reference, the model showed an odd of 3.267 for a moderate score, while the odds for the severe score were significantly higher (42), p-value $<$ 0.001. The odds (95% CI) for the SAPS II were 1.099 (1.060 – 1.138), and the p-value $<$ 0.001.

4. Discussion

Acute AlP poisoning is a major emergency in Egypt and some other developing countries. Due to the increasing prevalence and associated mortality in developing countries, clinicians continue to pursue a tool of risk stratification for these patients to allow better allocation of limited resources such as MV and other interventions [14,15]. To date, there is

Table. 5
Omnibus test, Hosmer and Lemeshow test and sensitivity analysis using univariate regression using the PGI, PSS and SAPS II as predictors of mortality among the studied patients.

	PGI score			PSS		SAPS II
Omnibus test X^2 (p value)	54.482 (< 0.001*)			56.456 (< 0.001*)		39.010 (< 0.001*)
Nagelkerk R^2	44 %			43 %		32.2 %
Chi-square (Hosmer and Lemeshow) (p value)	2.288 (0.319)			1.853 (0.173)		17.039 (0.030*)
Accuracy (%)	77.1			80.6		70.8
Sensitivity (%)	90.9			81.8		81.8
Specificity (%)	55.4			78.6		53.6
PPV (%)	76.2			85.7		73.5
NPV (%)	79.5			73.3		69.6
Univariate regression (with mortality)	**PGI score = 1**	**PGI score = 2**	**PGI score = 3**	**Moderate**	**Severe**	**SAPS-II**
Odds	4.487	34.875	42.625	3.267	42.0	1.099
95 % CI	1.667 – 12.080	9.574 – 127.041	8.244 – 220.399	0.652 – 16.370	8.457 – 208.586	1.060 – 1.138
Significance	0.003*	< 0. 001*	< 0. 001*	< 0. 001*	< 0. 001*	< 0. 001*

CI: confidence interval, PPV: positive predictive value, NPV: negative predictive value,
* significance at $p < 0.05$

no simple, reliable, and outcome predictive tool for acute AlP poisoning. Our study aimed to evaluate the performance of the recently introduced PGI score in predicting mortality and the need for MV and vasopressor therapy in acute AlP poisoning compared to PSS and SAPS II. The PGI showed comparable and sometimes better performances as a predictor of mortality and other adverse outcomes than PSS and SAPS II, suggesting that while all scores are helpful, the PGI score offers a slight edge in specificity and overall accuracy for these critical outcomes.

The current study was conducted enrolling 144 patients diagnosed with acute AlP poisoning, in which the mortality represented 61.1%, which is higher than the mortality rates reported by Sheta et al. (43 %) [30], Pannu et al. (51%) [20], and Singh at al. (58.9 %) [31], but lower than the mortality reported by Shadina et al. (66.7 %) [32]. Additionally, the current study revealed a mean age of 28 years among patients attempting suicide by AlP. Furthermore, we observed a relatively higher age, although not significant, among non-survivors (mean = 29.36 years) compared to survivors (mean = 25.86 years). Though the females represented 61.1% of the studied patients, we observed comparable sex distribution among the studied patients regarding mortality. The predominance of the females, along with the non-significant differences in age and sex, was reported earlier in another Egyptian study [30].

Consistent with the present study, Ghonem et al. reported significantly higher age among non-survivors exposed to AlP than survivors [33]. Contradicting the obtained findings, Abd Elghany et al. reported significantly higher deaths among patients aged 16–20 than older patients, where 82.1 % of non-survivors were between 16 and 20 [34]. El-Ebiary et al. also reported no age-significant variations regarding mortality and found that 67.50 % of exposed patients were less than 20 years old. They mentioned that the age group between 20 and 40 constituted the second most vulnerable category [35]. Nonetheless, the reported means and ranges of age in the present study agree with most of the published literature [30,33]. The significant involvement of young adults in AlP poisoning is attributed to the increasingly stressful conditions younger persons meet. They tend to be quickly excited and depressed. Besides, at this age, there are several family conflicts, teacher scolding, and education failure [36–38].

In agreement with obtained findings, Abdulghafar et al. [39] and Bogale et al. supported the preponderance of young females using AlP to commit suicide [40]. Abdel Wahab et al. reported that a similar proportion of studied patients exposed to AlP were females (63.3%) [41]. An earlier Egyptian study denoted that about 97.2% of the studied patients were admitted after suicidal exposure, and females constituted 91.8% of admitted patients [10]. However, the results regarding sex variation in exposure to AlP were conflicting and carried great discrepancy. Contradicting the current study, Kapoor et al. reported a predominance of males over females (2:1 ratio) [36]. Moreover, a systematic review and meta-analysis conducted in Iran found that suicidal ingestion of AlP was more prevalent among males [42]. The difference in the setting of the study is one factor that could justify some of the reported discrepancies. The availability of AlP in rural areas and the preference of either sex to work in farming are other contributing factors [41].

In Egypt, Kasemy et al. conducted their earlier study in two rural provinces and found that AlP was the leading cause of death due to self-poisoning. They revealed that generally, females and individuals aged less than 25 years old were significantly at higher risk of death following suicidal self-poisoning. They attributed the reported sex variations to the hormonal disturbances, family conflicts and psychological stress inflicted on the females [43]. The widespread availability and affordable price of AlP tablets in our region make it a common way to commit suicide. The lack of knowledge about the seriousness of AlP among females and the lack of an antidote to save exposed patients may be another cause [44].

In partial agreement with the current study, Sheta et al. reported significantly lower systolic and DBP among non-survivors. Moreover, they reported non-significant variations in the pulse and temperatures [30]. The current study depicted that SBPs < 75, < 70 and < 80 mmHg were significant predictors of mortality, need for MV, and vasopressor therapy, respectively. Higher cut-offs for mortality prediction were suggested by Sheta et al. (SBP < 80 mmHg) and Farzaneh et al. (SBP < 92.5 mmHg.) [4,30]. In further agreement with the current study, Pannu et al. stated that all exposed patients with hypotension were administered vasopressor agents [21].

The AlP-associated hypotension is multifactorial. Volume depletion is the leading cause of hypotension. Hypovolemia results from vascular wall insufficiency potentiated by the direct toxic effect of phosphine gas on the cardiac myocytes, resulting in tissue hypoperfusion. Tissue deprivation of oxygen shifts the body toward anaerobic respiration and intracellular acidosis, which further suppress cardiac functions and induce profound irreversible circulatory collapse [45,46]. Additively, hypovolemia is a common consequence of excessive vomiting, besides the cytotoxic effect of phosphine gas on the suprarenal glands, which leads to cortisol hyposecretion predisposing to hypotension [47].

The current study conveyed that the hypoxia, indicated by the low PaO_2/FiO_2 ratio, was a significant finding among non-survivors and a predictor of mortality and other adverse outcomes. Sheta et al. mentioned that survivors' oxygen saturation was significantly higher [30]. Although the mechanism of action of phosphine gas is not fully understood, Nakakita et al. suggested that, in animal studies, the phosphine gas inhibits ADP uncoupler and ion-stimulated respiration [48]. Likewise, it was hypothesized that the released phosphine gas impedes the cytochrome oxidase activity and suppresses mitochondrial oxidative respiration [49]. Additively, it was postulated that phosphine gas inhibits the acetylcholinesterase and results in alteration of acetylcholine signaling [50].

Because of the high oxygen requirement, the cardiac myocytes are

the most susceptible to the hypoxic damage induced by AlP. This damage can be early seen as an alteration in the cardiac electrical activity on electrocardiography [11]. Correspondingly, Sakr et al. reported higher troponin T levels among non-survivors exposed to AlP than survivors [10]. Another mechanism that explains cardiotoxicity is the oxidative stress injury due to the accumulation of reactive oxygen species and free radicles in the myocardial cell [5]. The successful therapeutic management using different antioxidants supported the role of oxidative stress in mediating phosphide-associated organ toxicity [51]. Antioxidants reduced the risk of mortality by three folds and the risk of MV by two folds [8].

Due to the mentioned cardiotoxic effects of AlP, cardiogenic shock should be aggressively managed with vasopressor therapy. A higher dosage of vasopressor therapy denotes significant cardiotoxicity [30]. As per TUPCC, which follows international guidelines, norepinephrine is the main used vasopressor. Norepinephrine improves tissue perfusion by increasing the stroke volume and cardiac output [52]. A systematic review conducted by Sobh et al. reported that amiodarone, digoxin, levosimendan, lidocaine, milrinone, trimetazidine, hydrocortisone, and vasopressin proved to counteract the phosphide-induced toxic cardiogenic shock and circulatory collapse [8].

The present work revealed that hypokalaemia was another significant predictor of adverse outcomes. Lower potassium levels were significant findings among AlP-associated fatalities in a previous study [21]. Hypokalaemia is a common finding after exposure to AlP and is usually secondary to vomiting. Catecholamine release and suprarenal damage with subsequent hypocortisolaemia are other proposed mechanisms [53]. The metabolic acidosis reported in this study was in concordance with several studies in which most admitted patients suffered from metabolic acidosis [54,55]. Previous studies reported that about 50% and 90.5% of exposed patients and 92.3% of non-survivors suffered from metabolic acidosis [19,30]. Sheta et al. admitted that metabolic acidosis, indicated by low pH and bicarbonate, was considered a significant predictor of mortality in AlP poisoning [30], Shadina et al., considered metabolic acidosis as a bad omen sign for other adverse outcomes [7]. Farzaneh et al. identified a bicarbonate level of 12.9 mEq/L, below which patients are likely to suffer from death [4]. Metabolic acidosis results from poison-associated hypoxia, in which the body switches to anaerobic conditions, inhibiting oxidative phosphorylation and accumulating lactic acid [56].

The current study highlighted the role of GCS in predicting adverse outcomes in AlP poisoning. GCS < 12 was a significant predictor of mortality and MV while GCS < 13 was a significant predictor of vasopressor therapy. These findings were parallel with Sheta et al., who conveyed that deaths in this type of poisoning were associated with low GCS < 12 [30], and with Pannu et al. who reported a lower median GCS of 11 as a benchmark for mortality [21]. On the other side, Fazaneh et al. reported a higher cutoff (GCS < 14.5) to predict mortality with good sensitivity and specificity [4]. Similar observations indicating a high prevalence of low GCS following this type of exposure were reported previously [32,57].

In acute AlP poisoning, admission to ICU and mortality are inseparable events. Sheta et al. mentioned that non-survivors showed higher mean values of ICU length of stay and higher percentages of ICU admission before death. Moreover, they noticed that the duration of MV was a significant predictor of death [30]. This study yielded that 54.9% of the studied patients needed MV, and of them, 92.4% did not survive. Louriz et al. and Pannu et al. conveyed that approximately 40% of the patients exposed to AlP suffered from respiratory failure, and MV was needed [21,32]. Shadina et al. reported that 100% of the studied patients needed MV [18]. Respiratory failure in AlP poisoning is attributed to myocardial toxicity and metabolic acidosis. Altered mental status may be another indication of MV, which manages the lack of airway reflexes and avoids aspiration and lung injury [32]. Mostafazadeh mentioned that although pulmonary edema was a common finding in AlP poisoning, it was unclear whether it was cardiogenic or non-cardiogenic in aetiology. They described the edema fluid as protein-rich and of hemorrhagic appearance [53]. The noticed linkage between respiratory failure and death following exposure to this poison could justify the similar cut-offs of scores predicting the MV and deaths obtained in the current study, given the known fact that MV is a primary cause of ICU admission.

As demonstrated in the current study, comparing the survivor and non-survivor showed significantly higher medians of PGI, SAPS II, and PSS scores at the time of admission. Several studies have been conducted to assess the predictive value of different scoring systems in acute AlP poisoning [17,30,32]. Pannu et al. recognized that pH < 7.25, GCS < 13, and SBP < 87 mmHg were the most robust predictors of mortality in AlP poisoning. All patients with a total PGI score of 2 and above died [20]. Later, they conveyed a positive correlation between the PGI and more complex scores, including the SAPS II scores [21]. Nonetheless, the medians of different scores among non-survivors reported by Pannu et al. were higher than those obtained in the current study (median PGI $= 3$ versus 2 and median SAPS II $= 44$ versus 36.5). One advantage of the PGI score is attributed to its ability to assess the most common clinical features associated with AlP poisoning (metabolic acidosis and cardiovascular collapse) [8].

Sakr et al. applied the PGI among 73 patients exposed to AlP, where 100% of patients who scored 3 died compared to only 25% of those who scored zero. Concurrent with the obtained findings, all patients with a PGI of 3 underwent MV and were administered vasopressors. They reported a positive correlation between the PGI score from one side and ECG changes, serum troponin T levels, MV, and vasopressors needed from the other side [10].

Considering the PGI score 0 as a reference, the current study reported that PGI scores 1, 2, and 3 significantly increased mortality odds by 4.487, 34.875, and 42.625, respectively. A PGI score of > 1 could be a strong indicator of both mortality and MV, with a specificity of 89.3% and 87.7% and PPV of 90.6% and 87.5%, respectively. Pannu et al. reported that a total PGI score of 3 had a 98.2% specificity and a positive predictive value of 96.4% [19]. Sakr et al. recognized a PGI score ≥ 1 as the best cut-off for cardiotoxicity prediction with 85.7% specificity, while a higher cut-off ≥ 2 was a significant mortality predictor with 87.5% specificity [10].

The present study revealed that SAPS II significantly predicted all adverse outcomes, notably in ruling out patients who do not require vasopressor therapy. Consistently, SAPS II was among the predictive scores used in the emergency room, which was found to predict multisystem organ failure in several poisoning cases, including AlP [21] organophosphate [58], paraquat [59] cardiotoxic drugs [60], and other non-specified pharmaceutical poisoning [59]. Hajouji et al. mentioned that SAPS II, shock, arrhythmia, use of vasopressors, and the need for MV were significantly correlated with the severity of AlP and adverse outcomes [61]. Similarly, Shadina et al. privileged the SAPS II for its accuracy in predicting mortality in this type of poisoning [18]. Dorooshi et al. identified significant higher SAPS II (> 30.02) among deaths due to pesticides including AlP and defined a cutoff of more than 16.5 as the most accurate outcome predictor [17]

Calculating urine output is one advantage of the SAPS II compared to the PGI score, given that urinary output was among the parameters that showed their significance as an adverse outcome predictor in baseline and ROC curve analyses. Consistent with the obtained findings in the present study, Pannu et al. reported lower urine output of 870.7 (mL/ in the first 24 hr.) in non-survivors compared to 1353.3 in survivors [21]. Singh et al. [31] and Farzaneh et al. also reported similar findings [4]. The significantly noticed oliguria reflects the shock state commonly seen in this type of poisoning [23].

It is noteworthy to underscore the obtained findings showing the significant improved performance of PGI score as a MV predictor compared to the PSS and SAPS II. Concurrent with the obtained findings, the PSS was considered a significant predictive score in AlP poisoning in previous studies [16,17]. Albeit the current study illustrated that the PSS

was a significant predictor of mortality and the need for MV and vasopressor therapy, we could not ignore the criticism the PSS received, which necessitates the need to develop simple, reliable other scores. The main limitations of SAPS II and PSS are the effort, and the time needed for calculations, which may be inapplicable in the context of acute toxic exposure [10]. The PSS is a historic score initially invented to evaluate the severity of poisoning in all types of acute toxic exposure. It encompasses several data points assessing the condition of 12 body systems. Aside from the fact that one score cannot assess all adverse outcomes in all types of exposure, the reliability of the PSS is questionable [62]. Ponnusankar et al. recommended adopting the PSS only as a basic model to establish more reliable simple models [63].

Furthermore, the validity of the PSS is doubtful. Most researchers deviated from the original score by misapplying or modifying the original PSS [64]. Moreover, the PSS's static nature limits its use in following the patient's condition. It adopts the worst values on admission, which is inconsistent with the principles of providing care and monitoring the poisoned patient [62]. Eventually, several studies proved comparable or better performances using other simple objective scoring systems compared to the PSS [65,66].

5. Conclusion

In acute AlP poisoning, non-survivors showed significantly lower blood pressures, PaO_2/FiO_2 ratios, temperatures, serum potassium levels, pH, bicarbonate levels, urine outputs, and GCS. Non-survivors exhibited significantly higher PGI, PSS and SAPS II than survivors. Though the PGI, PSS, and SAPS II proved their significance as predictors of mortality and, the need for MV and vasopressors, the PGI score showed a significantly higher AUC as a predictor of MV compared to PSS and SAPS II, suggesting that while all scores are helpful, the PGI score offers a slight edge in specificity, simplicity, and resource preserving nature for these critical outcomes. The PGI score could be a simple, and robust tool replacing the PSS and SAPS II for predicting mortality, clinical decision-making including the need for MV and vasopressor therapy in acute AlP exposure. Adopting the PGI score seems substantially useful in managing acute AlP poisoning, notably in resource-restricted countries.

Limitations

The current study adds to the knowledge gap by validating a simple, reliable score to predict mortality and other adverse outcomes in a serious type of poisoning; we admit some limitations. The main limitation is conducting this study in a single institute in one population. To generalize the obtained findings, we recommend future prospective studied on different population. Moreover, excluding patients over 60 and those suffering from co-morbid conditions might hamper comparisons in this category of patients and reduce the trustworthiness of generalizing the obtained findings.

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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. We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us. We understand that the Corresponding Author is the sole contact for the Editorial process. She is responsible for communicating with the other authors about progress, submissions of revisions and final approval of proofs.

CRediT authorship contribution statement

Aisha Emad ElMehy: Writing – original draft, Methodology, Formal analysis, Conceptualization. **Asmaa Fady Sharif:** Writing – review & editing, Visualization, Formal analysis. **Fatma Gaber Sobeeh:** Writing – original draft, Methodology, Formal analysis, 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

Data availability

Data will be made available on request.

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