

IDENTIFYING PREDICTORS OF INTENSIVE CARE UNIT ADMISSION AND MORTALITY AMONG ACUTE ORGANOPHOSPHORUS POISONED PATIENTS

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ABSTRACT

Background: Acute organophosphorus (OP) poisoning is a critical toxicological problem with substantial morbidity and mortality. **Aim of the study:** Identifying applicable predictors of intensive care unit (ICU) admission and mortality in acute OP poisoning. **Patients and Methods:** This study was conducted on patients with acute OP poisoning admitted to Tanta University Poison Control Center from January 2023 to December 2023. Personal and toxicological history, findings of clinical examination, and laboratory investigations were collected from the patients' files. Poisoning Severity Score (PSS) and Rapid Emergency Medicine Score (REMS) were documented. Patients were sub-grouped considering ICU admission and mortality. **Results:** Out of 66 patients, 15 (22.7%) required ICU admission and 6 (9.1%) died. The age of patients ranged from 18 to 66 years, 59.1% were males, and 80.3% were from rural areas. Suicidal poisoning and oral route were found in 57.6% and 72.2%, respectively. Pulse and respiratory rates, creatinine, urea, serum glutamic pyruvic transaminase (SGPT), and serum glutamic-oxaloacetic transaminase (SGOT) were significantly higher while Glasgow Coma Scale, partial arterial oxygen pressure (PaO₂), pH, and serum bicarbonate (HCO₃) were significantly lower in adverse outcome groups. Both PSS and REMS predicted ICU admission with excellent performance (AUC = 0.971 and 0.955, respectively) and mortality with good performance (AUC = 0.890 0.879, respectively). **Conclusion:** PaO₂, pH, serum HCO₃, creatinine, urea, SGPT, SGOT, PSS, and REMS could predict adverse outcomes of acute OP poisoning. **Recommendations:** REMS could be recommended as a simple and applicable tool to predict ICU admission and mortality in acute OP poisoning.

Keywords: Organophosphorus pesticides, poisoning, intensive care unit, mortality, prediction, outcomes

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INTRODUCTION

Organophosphorus (OP) pesticides are organic chemicals obtained from phosphoric acid and from its derivatives with at least only one carbon-phosphorus bond (Mukherjee and Gupta, 2020). They are used widely for industrial purposes and to control insects in both agricultural and domestic sets in a lot of countries including Egypt (El-Ebiary et al., 2016; Ganie et al., 2022). Unfortunately, accidental and suicidal poisoning is responsible for substantial rates of morbidity and mortality (Maksimović et al., 2021).

Organophosphorus pesticides phosphorylate then inactivate acetylcholinesterase (AChE); an enzyme which breaks down acetylcholine neurotransmitter. Consequently, the accumulated acetylcholine causes hyper

stimulation of the cholinergic system (Chen et al., 2021).

The classic toxidrome of acute OP poisoning, includes muscarinic manifestations in the form of diarrhea, urination, miosis, bronchorrhea, bronchospasm, bradycardia, vomiting, salivation, lacrimation and hypotension. Furthermore, stimulation of nicotinic receptors at sympathetic ganglia and neuromuscular junction results in pupil dilatation, tachycardia, weakness, hypertension, fasciculations, shallow breathing, and sweating (Pannu et al., 2021). Central nervous system involvement is manifested by seizures, coma, and respiratory failure (Zobeiri, 2021).

Treatment of acute OP poisoning depends mainly on cardio-respiratory stabilization and decontamination according to the route of exposure. Antidote therapy includes atropine and pralidoxime chloride. Atropine acts as a

peripheral and central anti-muscarinic agent while, pralidoxime chloride works to reactivate the inhibited AChE enzyme (*Abdel Baseer et al., 2021*).

Despite widely available treatment strategies, acute OP poisoning might be associated with significant morbidities. Furthermore, mortality rate may reach up to 10-20% (*Thakur et al., 2022*). In clinical situations, it is necessary to predict the outcome of acute poisoned patients to offer appropriate and in-time treatment.

Literature investigated different scoring systems and biochemical parameters to evaluate patients; however, controversy is still present about predictors of adverse outcomes (*Acikalin et al., 2017*).

THE AIM OF THE WORK

The current study aimed to identify simple and applicable predictors of adverse outcomes; intensive care unit (ICU) admission and mortality in cases of acute OP poisoning.

PATIENTS AND METHODS

Study design, setting, and ethical consideration

This retrospective cohort study was conducted on patients with acute OP intoxication who were admitted to Tanta University Poison Control Centre (TUPCC), Egypt from 1st January 2023 till 31th December 2023. The study was approved by the Research Ethical Committee - Faculty of Medicine - Tanta University (Approval code: 36264PR478/12/23) and went with the principles of the Declaration of Helsinki. Coding and anonymous analysis of data were considered to maintain patients' confidentiality. Informed consents from patients were waived because of the retrospective nature of the study.

Inclusion criteria

All acute OP poisoned patients aged ≥ 18 years were included in this study. Diagnosis was based on history of exposure, recognition of the poison bottle or label, as well as, characteristic odor of gastric lavage contents or vomitus and clinical manifestations of cholinergic and nicotinic toxidromes following exposure to OP preparations. These manifestations include salivation, vomiting,

diarrhea, sweating, fasciculations, and confusion (*Kamath and Gautam, 2021*).

Exclusion criteria

Patients younger than 18 years, both lactating and pregnant females, and unknown or mixed intoxicated patients were excluded. Furthermore, patients with medical illness like cardiovascular, renal or hepatic impairments, and diabetes were excluded. Other patients with pre-hospital intervention were also excluded.

Methods

The following data were collected cautiously from patients file

I- History: Personal data in the context of age, sex, and residence, as well as, toxicological history including mode, and route of poisoning and pre-hospitalization period (delay time).

II- Clinical Examination:

Clinical data were retrieved from the patients' files including systemic examination, vital signs (pulse rate, mean arterial blood pressure [MAP], respiratory rate [RR], and temperature), and Glasgow Coma Scale (GCS).

III- Laboratory investigations:

Results of laboratory investigations were also collected. These laboratory findings include arterial blood gases (partial arterial oxygen pressure [PaO₂], partial arterial carbon dioxide pressure [PaCO₂], pH, and serum bicarbonate [HCO₃]), serum sodium (Na) and potassium (K) levels, liver enzymes (serum glutamic pyruvic transaminase [SGPT] and serum glutamic-oxaloacetic transaminase [SGOT]), blood urea and serum creatinine.

IV- Scoring system:

1- Poisoning Severity Score (PSS) followed the International Programme on Chemical Safety (IPCS) (*Persson et al., 1998*). According to the patient's most severe symptoms or signs, poisoning severity was classified into five grades as follows:

- Grade 0: indicates no symptoms
- Grade 1: symptoms are mild, transient, and spontaneously resolving.
- Grade 2: prolonged or pronounced symptoms
- Grade 3: symptoms are severe or life-threatening
- Grade 4: death

2- Rapid Emergency Medicine Score (REMS) was calculated using 6 variables; age, pulse rate, MAP, RR, GCS, and peripheral oxygen saturation. The values of these variables range from 0 to 4 except the value of the age ranges from 0 to 6. The total REMS ranged from 0 to 26. The higher value indicates a bad prognosis (*Olsson et al. 2004*).

Outcome measures

Patients were divided according to ICU admission into ICU admitted and ICU-not admitted patients. Based on mortality they were divided into expired (not survived) and survived patients. According to *Smith and Nielsen (1999)*, indications of calling ICU medical staff to adult patients include threatened airway, respiratory arrest, respiratory rate more than 40 or less than 8 breaths/min, oxygen saturation less than 90% on more than 50%, cardiac arrests, pulse rate less than 40 or more than 140 beats/minute, systolic blood pressure less than 90 mmHg, sudden drop in consciousness level (GCS falls by more than 2 points), repeated or continued seizures, and increasing arterial carbon dioxide tension with respiratory acidosis.

Treatment

Standard treatment was given to all admitted patients as guided by the attending physician according to TUPCC Protocol. This standard treatment involved the following: patient resuscitation (when indicated), dermal and/or gastric decontamination with administration of 1gm/kg activated charcoal as a single dose. Beside, antidotal therapy (atropine and oxime) was also considered according to the patients' assessment. Atropine (1 to 3 mg IV) was given as a bolus dose and repeated every 10 minutes to 15 minutes. The end point of atropine therapy was when the heart rate exceeded 80 beats/minute, the systolic blood pressure elevated to more than 80 mmHg, and when the chest was clear. Then, atropine was maintained by continuous intravenous infusion of 10–20% of the loading dose every hour (*Eddleston et al., 2008*).

Toxogonin® (1 ampoule contains 250 mg of obidoxime chloride in 1 ml, produced by Merck, Darmstadt, Germany) was given for nicotinic manifestations. It was given as a loading dose of 250 mg bolus IV, followed by 750 mg every 24 hours until at least 12 hours

after stopping of atropine (*Roberts and Aaron, 2007*).

STATISTICAL ANALYSIS

Statistical Package for Social Sciences (SPSS) version 27 (IBM Corp., Armonk, N.Y., USA) was used for analyzing the data. The distribution of continuous numerical data was tested using the Shapiro-Wilk test for normality. Numerical data following the normal distribution were presented as mean \pm standard deviation and the comparisons between the two groups were done using the independent samples T-test. Data not following the normal distribution were presented using the median and interquartile range (25th–75th percentiles), and comparisons employed the Mann-Whitney test. Pearson's Chi-square test for independence or Fisher's exact test were used to assess the association between two categorical variables as appropriate. Receiver operating characteristics (ROC) curve was analyzed to assess the performance of the studied scores in discriminating patient's outcomes. A *p* value <0.05 was selected to interpret the significance of the results.

RESULTS

The study herein enrolled sixty six patients presented with acute OP poisoning who have fulfilled the eligibility criteria during the research duration. Fifteen patients (22.7%) required ICU admission and the expired were six patients (9.1%). As **table (1)** illustrated, no significant difference was detected between the studied groups regarding socio-demographic distribution (age, sex, and residence). Thirty eight patients (57.6%) alleged suicidal poisoning. Oral ingestion of OP was reported in 72.7% of patients. Delay time ranged from 1.5 to 5.5 hours. Significant difference was observed between ICU admitted and ICU-not admitted patients regarding both mode and route of poisoning (*p*=0.010 and 0.013, respectively). The mean values of pulse and respiratory rates showed significant increase in ICU admitted and expired patients. However, both MAP and temperature did not exhibit significant difference between the groups. For GCS, it was significantly lower in ICU admitted patients rather than ICU-not admitted patients

($p < 0.001$) and in non-survivors compared to the survivors ($p = 0.004$).

Figure (1) demonstrates that the most frequently reported manifestations were vomiting and abdominal colic while the least reported manifestations were disturbed consciousness and sweating.

Statistical analysis of the results of laboratory investigations is demonstrated in **table (2)**. Regarding PaO₂ and serum HCO₃, they were significantly lower in ICU admitted patients compared to ICU-not admitted patients ($p < 0.001$ in both). Dead patients had significantly lower PaO₂, pH, and serum HCO₃ rather than survivors ($p < 0.031$, 0.024, and 0.002, respectively).

Regarding serum Na and K levels, there was no statistical significant difference between the studied groups.

The median value of serum creatinine was significantly higher in ICU admitted patients and in non-survivors versus the corresponding groups ($p = 0.002$ and 0.013, respectively), however the mean value of urea showed a significant higher level in ICU admitted patients rather than ICU-not admitted patients ($p < 0.001$) and no significant difference between survived and not survived patients. For liver enzymes, SGPT was significantly higher in ICU admitted patients compared to ICU-not admitted patients ($p = 0.002$). Regarding SGOT, it was significantly higher in ICU admitted patients and non survivors rather than their comparable groups ($p < 0.001$ and 0.025, respectively).

Table (1): Socio-demographic characteristic, initial vital signs, and Glasgow coma scale of acute organophosphorus poisoning patients (n = 66 patients).

Variables		ICU admitted n = 15 (22.7%)	ICU-not admitted (n=51)	p value	Expired n = 6 (9.1%)	Survived (n = 60)	p value	Total
Age (years)	Mean ± SD (Min - Max)	34.5 ± 16.9 (18.0 - 66.0)	29.1 ± 11.2 (18.0 - 52.0)	0.256 t	35.8 ± 17.2 (18.00 - 66.00)	29.8 ± 12.3 (18.00 - 62.00)	0.271 t	30.3 ± 12.8 (18.0 - 66.0)
Sex	Male	7 (46.7%)	32 (62.7%)	0.266	5 (83.3%)	34 (56.7%)	0.388	39 (59.1%)
	Female	8 (53.3%)	19 (37.3%)	X ²	1 (16.7%)	26 (43.3%)	X ²	27 (40.9%)
Residence	Rural	12 (80.0%)	41 (80.4%)	1.000	4 (66.7%)	49 (81.7%)	0.377	53 (80.3%)
	Urban	3 (20.0%)	10 (19.6%)	X ²	2 (33.3%)	11 (18.3%)	X ²	13 (19.7%)
Mode	Suicidal	13 (86.7%)	25 (49.0%)	0.010*	5 (83.3%)	33 (55.0%)	0.230	38 (57.6%)
	Accidental	2 (13.3%)	26 (51.0%)	X ²	1 (16.7%)	27 (45.0%)	X ²	28 (42.4%)
Route	Oral	14 (93.3%)	34 (66.7%)	0.013*	6 (100.0%)	42 (70.0%)	0.441	48 (72.7%)
	Inhalation	0 (0.0%)	16 (31.4%)	X ²	0 (0.0%)	16 (26.7%)	X ²	16 (24.3%)
	Cutaneous	1 (6.7%)	1 (2.0%)		0 (0.0%)	2 (3.3%)		2 (3.0%)
Delay (hours)	Median [IQR] (Min - Max)	5.0 [2.5 - 6.5] (0.5 - 12.0)	3.0 [1.5 - 5.0] (1.0 - 12.0)	0.131 Z	3.0 [2.5 - 5.0] (1.5 - 12.0)	3.0 [1.5 - 5.8] (0.5 - 12.0)	0.752 Z	3.0 [1.5 - 5.5] (0.5 - 12.0)
Pulse rate (beats/minute)	Mean ± SD (Min - Max)	110.0 ± 25.9 (56.0 - 153.0)	77.0 ± 19.5 (55.0 - 128.0)	<0.001* t	114.7 ± 30.5 (64.0 - 153.0)	81.5 ± 22.7 (55.0 - 130.0)	0.002* t	84.5 ± 25.1 (55.0 - 153.0)
Respiratory rate (breaths/minute)	Mean ± SD (Min - Max)	26.6 ± 8.8 (15.0 - 40.0)	18.8 ± 3.4 (14.0 - 30.0)	0.004* t	31.7 ± 9.0 (15.0 - 40.0)	19.5 ± 4.4 (14.0 - 35.0)	0.020* t	20.6 ± 6.0 (14.0 - 40.0)
Mean arterial pressure (mmHg)	Median [IQR] (Min - Max)	90.0 [83.3 - 116.7] (63.3 - 126.7)	86.7 [70.0 - 96.7] (53.3 - 116.7)	0.069 Z	90.0 [86.7 - 116.7] (83.3 - 116.7)	86.7 [70.0 - 96.7] (53.3 - 126.7)	0.231 Z	86.7 [73.3 - 96.7] (53.3 - 126.7)
Temperature (°C)	Mean ± SD (Min - Max)	36.9 ± 0.3 (36.5 - 37.4)	36.8 ± 0.4 (36.0 - 37.8)	0.398 t	36.9 ± 0.2 (36.5 - 37.2)	36.8 ± 0.4 (36.0 - 37.8)	0.578 t	36.8 ± 0.4 (36.0 - 37.8)
GCS	Median [IQR] (Min - Max)	5.0 [3.0 - 10.0] (3.0 - 15.0)	15.0 [15.0 - 15.0] (13.0 - 15.0)	<0.001* Z	6.0 [3.0 - 13.0] (3.0 - 15.0)	15.0 [15.0 - 15.0] (3.0 - 15.0)	0.004* Z	15.0 [15.0 - 15.0] (3.0 - 15.0)

n: number; ICU: intensive care unit, mmHg: millilitre mercury, GCS: Glasgow Coma Scale, IQR: interquartile range (25th – 75th percentiles); Max: maximum; Min: minimum; SD: standard deviation; t: Independent samples T-test; Z: Mann-Whitney test; X²: Pearson's Chi-square test/ Fisher's exact test; * significant at $p < 0.05$

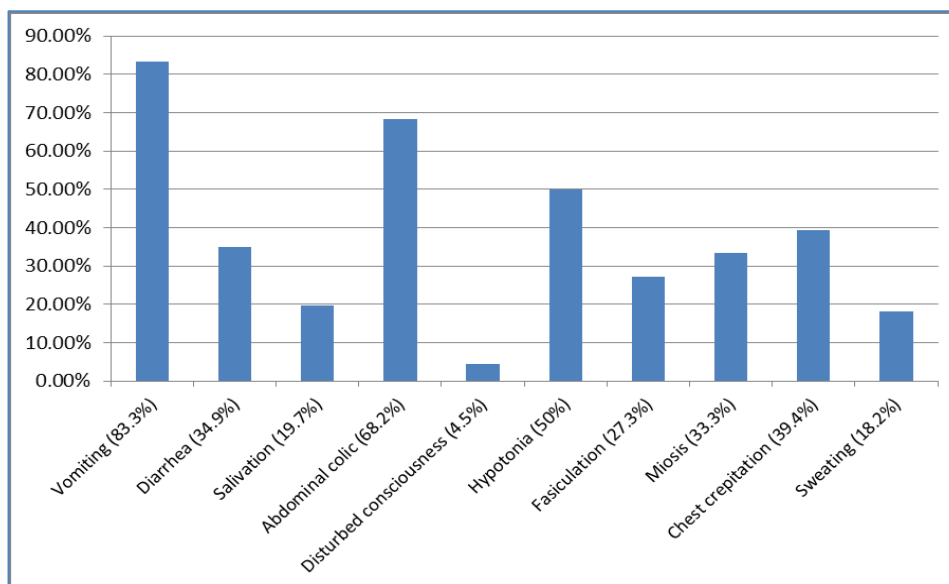


Figure (1): Clinical manifestations among acute organophosphorus poisoned patients.

Table (2): Initial laboratory investigations of acute organophosphorus poisoning patients (n = 66 patients).

Variables		ICU admitted n = 15 (22.7%)	ICU-not admitted n = 51	p value	Expired n = 6 (9.1%)	Survived n=60	p value	Total
PaO ₂ (mmHg)	Mean ± SD (Min - Max)	80.9 ± 13.2 (60.0 - 100.0)	95.7 ± 3.3 (88.0 - 100.0)	<0.001* t	76.2 ± 14.8 (60.0 - 99.0)	94.0 ± 6.8 (62.0 - 100.0)	0.031* t	92.4 ± 9.3 (60.0 - 100.0)
pH	Median [IQR] (Min - Max)	7.37 [7.28 - 7.48] (6.90 - 7.55)	7.40 [7.38 - 7.45] (7.34 - 7.51)	0.170 Z	7.27 [7.22 - 7.41] (6.90 - 7.47)	7.40 [7.37 - 7.45] (7.30 - 7.55)	0.024* Z	7.40 [7.37 - 7.45] (6.90 - 7.55)
Serum HCO ₃ (mmol/L)	Median [IQR] (Min - Max)	21.7 [18.2 - 23.8] (12.2 - 26.0)	26.4 [22.3 - 29.0] (18.0 - 34.2)	<0.001* Z	18.8 [18.2 - 22.0] (12.2 - 23.8)	25.9 [21.7 - 28.2] (16.8 - 34.2)	0.002* Z	24.5 [20.9 - 27.9] (12.2 - 34.2)
PaCO ₂ (mmHg)	Median [IQR] (Min - Max)	32.2 [28.7 - 44.2] (20.4 - 81.7)	37.0 [30.1 - 45.2] (22.5 - 52.1)	0.515 Z	36.1 [28.7 - 48.4] (24.2 - 81.7)	36.4 [30.0 - 45.0] (20.4 - 52.1)	0.905 Z	36.4 [29.9 - 45.1] (20.4 - 81.7)
Potassium level (mmol/L)	Mean ± SD (Min - Max)	3.94 ± 0.66 (3.00 - 5.40)	3.78 ± 0.54 (2.80 - 5.30)	0.364 t	4.26 ± .95 (3.00 - 5.40)	3.77 ± 0.50 (2.80 - 5.30)	0.269 t	3.82 ± 0.57 (2.80 - 5.40)
Sodium level (mmol/L)	Mean ± SD (Min - Max)	142.7 ± 4.0 (133.0 - 150.0)	140.4 ± 5.0 (130.6 - 153.0)	0.108 t	142.3 ± 5.0 (133.0 - 147.0)	140.8 ± 4.9 (130.6 - 153.0)	0.451 t	140.9 ± 4.9 (130.6 - 153.0)
Creatinine (mg/dL)	Median [IQR] (Min - Max)	1.10 [1.00 - 1.20] (0.80 - 2.80)	0.90 [0.80 - 1.00] (0.50 - 1.40)	0.002* Z	1.16 [1.10 - 1.20] (0.80 - 2.80)	0.90 [0.80 - 1.02] (0.50 - 1.40)	0.013* Z	0.90 [0.80 - 1.10] (0.50 - 2.80)
Urea (mg/dL)	Mean ± SD (Min - Max)	44.8 ± 12.5 (25.0 - 78.0)	28.9 ± 7.5 (15.0 - 50.0)	<0.001* t	45.8 ± 20.0 (25.0 - 78.0)	31.2 ± 9.0 (15.0 - 53.0)	0.134 t	32.5 ± 11.1 (15.0 - 78.0)
SGPT (U/L)	Median [IQR] (Min - Max)	25.0 [21.0 - 31.0] (17.0 - 88.0)	20.0 [17.0 - 23.0] (11.0 - 42.0)	0.002* Z	26.0 [21.0 - 32.0] (17.0 - 88.0)	20.0 [17.0 - 24.5] (11.0 - 42.0)	0.076 Z	20.0 [17.0 - 25.0] (11.0 - 88.0)
SGOT (U/L)	Median [IQR] (Min - Max)	39.0 [30.0 - 46.0] (23.0 - 62.0)	25.0 [20.0 - 33.0] (14.0 - 47.0)	<0.001* Z	39.5 [33.0 - 54.0] (23.0 - 62.0)	26.0 [21.0 - 36.5] (14.0 - 52.0)	0.025* Z	26.5 [22.0 - 39.0] (14.0 - 62.0)

n: number, ICU: intensive care unit, PaO₂: partial arterial oxygen pressure, mmHg: millilitre mercury, HCO₃: bicarbonate, mmol/L: millimol per litre, PaCO₂: partial arterial carbon dioxide pressure, mg/dL: milligram per decilitre, SGPT: serum glutamic pyruvic transaminase, SGOT: serum glutamic-oxaloacetic transaminase, U/L: unit per litre, SD: standard deviation, IQR: interquartile range (25th – 75th percentiles); Max: maximum; Min: minimum; t: Independent samples T-test; Z: Mann-Whitney test, * significant at p < 0.05.

Regarding PSS, the median value in ICU admitted patients (3) was significantly higher than ICU-not admitted patients (1) ($p < 0.001$) and it was significantly higher in non-survivors compared to the survivors (3 versus 2, respectively; $p < 0.001$). Likewise, the median value of REMS was significantly higher in ICU admitted patients rather than ICU-not admitted patients (9 versus 2, respectively; $p < 0.001$) and in non survivors (11) compared to the survivors (2) ($p < 0.001$) as shown in **table (3)**.

Table (4) and **figures (2, 3)** depicts the result of ROC curve analysis to predict ICU admission and mortality among acute OP poisoned patients. The highest AUC values to predict ICU admission (0.971 and 0.955) were observed for PSS and REMS at cut-off > 2 and > 4 , respectively. Likewise, PSS and REMS had the highest AUC values (0.890 and 0.879) to predict mortality at cut-off > 2 and > 5 , respectively.

Table (3): Comparison of poisoning severity score and rapid emergency medicine score between acute organophosphorus poisoning groups (n = 66 patients).

Variables		ICU admitted n = 15 (22.7%)	ICU-not admitted n = 51	p value	Expired n = 6 (9.1%)	Survive d n = 60	p value	Total
PSS	Median [IQR] (Min - Max)	3 [3 - 3] (2 - 3)	1 [1 - 2] (1 - 2)	<0.001* Z	3 [3 - 3] (2 - 3)	2 [1 - 2] (1 - 3)	<0.001* Z	2 [1 - 2] (1 - 3)
REMS	Median [IQR] (Min - Max)	9 [5 - 12] (2 - 15)	2 [0 - 3] (0 - 6)	<0.001* Z	11 [6 - 13] (2 - 15)	2 [0 - 4] (0 - 12)	0.001* Z	2 [0 - 4] (0 - 15)

n: number; *ICU*: intensive care unit, *PSS*: poisoning severity score, *REMS*: rapid emergency medicine score, *IQR*: interquartile range (25th – 75th percentiles); *Min*: minimum; *Max*: maximum; *Z*: Mann-Whitney test, * significant at $p < 0.05$.

Table (4): The receiver operating characteristic (ROC) curve analysis of poisoning severity score, rapid emergency medicine score, and some laboratory parameters in predicting intensive care unit admission and mortality in acute organophosphorus poisoned patients (n = 66).

Variable	AUC	95% CI	p	Cut-off	Sensitivity %	Specificity %
ICU admission						
HCO ₃	0.816	0.709 to 0.924	<0.001*	≤24.1	93.33	66.67
SGPT	0.767	0.635 to 0.900	<0.001*	>22	73.33	72.55
SGOT	0.801	0.684 to 0.917	<0.001*	>29	80.00	70.59
Urea	0.882	0.771 to 0.992	<0.001*	>35	86.67	88.24
Creatinine	0.761	0.627 to 0.896	<0.001*	>0.9	86.67	62.75
PSS	0.971	0.932 to 1.000	<0.001*	>2	86.67	100.00
REMS	0.955	0.893 to 1.000	<0.001*	>4	80.00	98.04
Mortality						
pH	0.776	0.490 to 1.000	0.059	≤7.28	66.67	100.00
HCO ₃	0.863	0.724 to 1.000	<0.001*	≤18.9	66.67	95.00
SGOT	0.774	0.570 to 0.978	0.009*	>32	83.33	68.33
Creatinine	0.801	0.569 to 1.000	0.011*	>1.06	83.33	80.00
PSS	0.890	0.793 to 0.988	<0.001*	>2	83.33	86.67
REMS	0.879	0.710 to 1.000	<0.001*	>5	83.33	88.33

ICU: intensive care unit; *SGPT*: serum glutamic pyruvic transaminase, *SGOT*: serum glutamic-oxaloacetic transaminase *PSS*: poisoning severity score; *REMS*: rapid emergency medicine score; *AUC*: area under the curve; *CI*: confidence interval; *p*: p-value from a Z-test comparing observed AUC to the null-hypothesis AUC of 0.5; * significant at $p < 0.05$

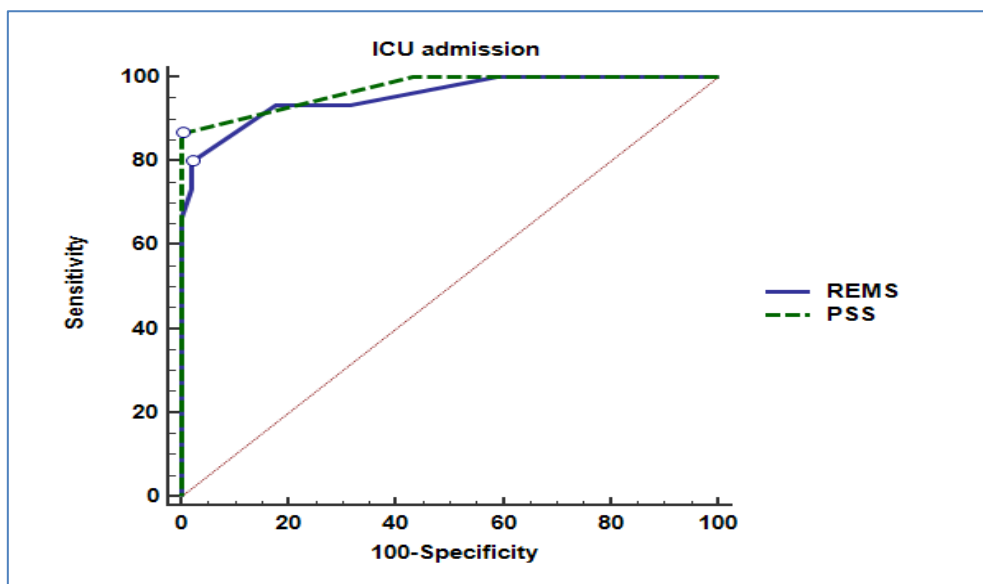


Figure (2): The receiving operating characteristic (ROC) analysis of poisoning severity score (PSS) and rapid emergency medicine score (REMS) in predicting the need of intensive care unit (ICU) admission among organophosphorus poisoned patients.

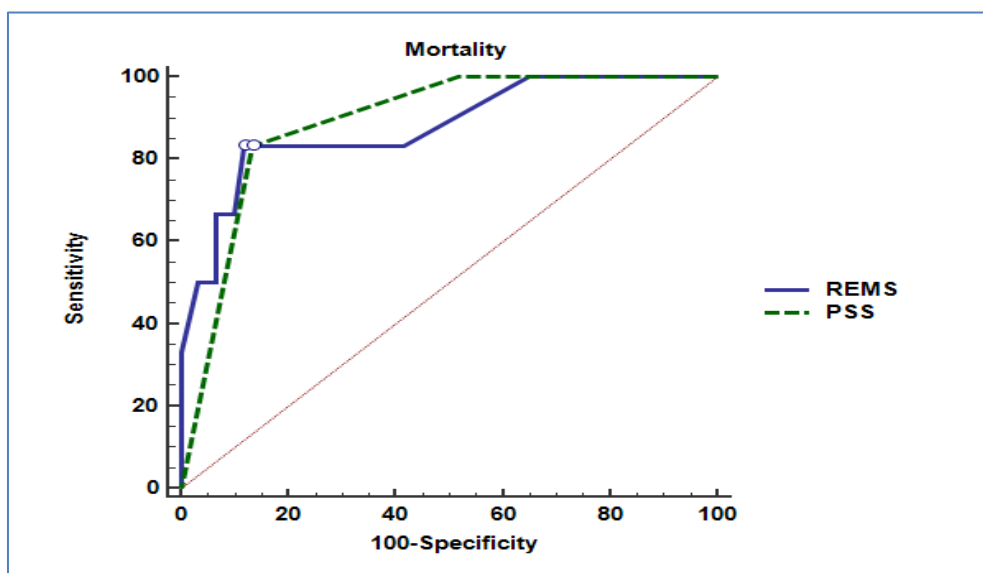


Figure (3): The receiving operating characteristic (ROC) analysis of poisoning severity score (PSS) and rapid emergency medicine score (REMS) in mortality among organophosphorus poisoned patients.

DISCUSSION

Acute OP poisoning has continued to be a significant health problem in developing countries including Egypt due to accidental poisoning, as well as, the potential use of OP pesticides as a tool for self-poisoning. This is because of their low cost and easy availability (Abdel Baseer *et al.*, 2021). World Health Organization classified organophosphorus compounds as highly toxic pesticides (Tadesse *et al.*, 2023). Accordingly, acute OP poisoned patients are at a high risk of potential adverse outcomes due to unbalanced health status. For this reason, early detection

of such patients is important to start a rapid and an appropriate treatment including intensive care measures especially when resources are deficient (Dong *et al.* 2021). Hence, the aim of this work is to identify simple and applicable predictors of ICU admission and mortality in acute OP poisoned patients.

In the present study, ICU admission and mortality were reported in 22.7% and 9.1% of the enrolled patients, respectively. Consistent with these results, Elagamy and Gabr (2019) and El-Gharbawy and Wahdan (2022)

documented ICU admission among 20.54% and 25.3% of acute OP poisoned patients.

While mortality was reported in 11.8%, 13.9%, and 8.6% according to *Amin et al. (2018)*; *Eisa et al. (2021)* and *Sontakke and Kalantri (2023)*, respectively.

However, variations may be attributed to the differences in the severity of the included cases.

Data analysis of the present study showed that the mean age value of the included patients was 30.3 years. In the same context, *Hodeib and Khalifa (2020)* reported a median age value of 35 years. *Krishna Moorthy et al. (2023)* reported a mean age value of 32.8 years among patients with acute OP poisoning. Furthermore, males accounted for more than half of the participants (59.1%). Likewise, *Reddy et al. (2020)* and *Raveendra and Mohan (2021)* reported male predominance. They explained this finding as males are the main working power on the farms predisposing them to accidental poisoning and easier access to OP pesticides in case of intentional poisoning. Conversely, other studies reported a higher incidence of acute OP poisoning among females (*Masoud et al., 2022*). Females are more credited to stress compared to males. Furthermore, OP preparations are available as household products (*Mohamed et al., 2019*).

The most studied patients were from rural areas. This is in match with the observations of *El-Ebiary et al. (2016)*; *Elagamy and Gabr (2019)*, and *Shama et al. (2021)*. It could be explained by the widespread use of OP pesticides in farming, especially in agriculture rural areas of the Delta region and their easy accessibility in rural homes, as well as, lack of protective equipment.

Most of our patients were poisoned with suicidal intention (57.6%) and ingestion was the main route of exposure in this work. Similar findings were extensively detected in other studies (*Beltagy et al., 2018*; *Reddy et al., 2020*; *El-Gharbawy and Wahdan, 2022*; *Pradhan et al., 2022*). The median value of delay time between OP exposure and hospital admission in this research was 3 hours. A similar result was observed by *Masoud et al. (2022)*. However, *Anjana and Neeta (2019)* reported that majority of their patients

presented after 3 hours of exposure. Our results could be attributed to the geographical position of TUPCC in the Delta region with easy accessibility and availability of transportation.

In the current study, the clinical findings at time of admission were variable. The main manifestations were vomiting, abdominal pain, and hypotonia. Sweating and disturbed consciousness were the least reported manifestations. Reference-wise, *Elagamy and Gabr (2019)* documented vomiting and miosis in 88.61% and 85.89%, respectively while convulsion was reported only in 3.22%. *Kamath and Gautam (2021)* reported vomiting (94%) and excessive secretions (84%) as the commonest presentations while impaired level of consciousness (22%) and seizures (12%) were relatively uncommon among their studied patients.

Vital signs are postulated to offer important physiologic cues for both the severity of poisoning and the outcome as they can be altered by the poisonous agents through the sympathetic and/or parasympathetic pathways (*Abd Elfatah et al., 2022*).

In the current study, pulse rate was significantly higher in ICU admitted patients and non-survivors when compared to ICU not-admitted patients and survivors while there was no significant difference between the studied groups regarding MAP. Nicotinic signs are expected to occur early in cases with severe OP poisoning, the incidence of tachycardia increases with toxidrome severity. In harmony with our finding, *Pannu et al., (2021)* reported tachycardia in acute OP poisoned patients due to direct sympathetic stimulation as accumulated acetylcholine causes stimulation of nicotinic receptors, hypovolemia resulting from sialorrhea, diaphoresis, diarrhea, and urination, or anti-muscarinic medications.

However, this finding comes in different with *Balmuchu et al. (2023)* who found no significant difference between survivors and dead patients regarding heart rate. Furthermore, *Kim et al. (2013)* detected a significant lower MAP mean value in non-survivors versus survivors in acute OP poisoning.

However, *Shahin and Hafez (2020)* reported normal blood pressure in 81.8% of acute OP poisoned patients while hypertension and hypotension were equally distributed.

In the present study, respiratory rate was significantly higher in patients with adverse outcomes. This could be explained by respiratory distress initiated by excessive secretion, bronchospasm, associated aspiration pneumonia, or septicemia (*Abdel Baseer et al., 2021*).

Furthermore, the main fatal complication following acute OP poisoning is respiratory failure (*Sert et al., 2019*). This could also explain a significantly lower mean value of PaO₂ in ICU admitted patients and non-survivors. This finding is equivalent to the results of *Abdel Baseer et al., (2021)*. Abundant chest secretions prevent oxygen from accessing the alveolar epithelium (*Eddleston and Chowdhury, 2016*). Subsequently, patients with lower PaO₂ are at a high risk of ICU admission and mortality.

Furthermore, there was no significant difference between the studied groups regarding temperature. In contrast, *Moussa et al. (2018)*, in their study on patients with acute OP poisoning, fever was documented in 20% of expired patients and 6% of survivors which was significantly different.

However, when they used linear regression analysis, temperature was not a significant predictor for mortality. On the other hand, in a pediatric study done by *Açıkgoz (2021)*, it was noticed that there was no significant difference in temperature among acute OP poisoned patients who were admitted to ICU and those who were not admitted. Additionally, *Moffatt et al. (2010)* concluded that OP poisoning causes an initial hypothermia followed by a period of normal to high body temperature. Many factors could affect body temperature in acute OP poisoning including lower respiratory tract infection, drug administration as atropine, and the occurrence of convulsion (*Moussa et al., 2018*).

Regarding GCS, it showed a significantly lower median values in both ICU admitted patients (5) and not survived groups (6) rather than ICU-not admitted and survived patients (15 for each). These findings are in line with

El-Gharbawy and Wahdan (2022) who documented a significant lower median GCS value in ICU admitted (6) and expired patients (6) in comparison with ICU-not admitted patients and survivors (15 for each). Furthermore, *Shahin and Hafez (2020)* found that the median value of GCS was significantly lower in non-survivors (7) rather than the survivors (15). They also concluded significant negative correlation between GCS and duration of hospital stay. In the same context, *Oreby and El-Madah (2017)* identified a cut-off 9 for GCS to predict mortality in acute OP poisoning. Low GCS is frequently seen in severe OP poisoned patients. Direct cerebral toxic effects of the agents, as well as, hypoperfusion and hypoxemia caused by respiratory failure usually contribute to low GCS scores (*Acikalin et al., 2017*).

In the current study, pH was significantly lower in expired patients and serum HCO₃ was significantly lower in patients who had adverse outcomes. However, PaCO₂ did not show significant difference between the studied groups. This finding comes in line with the results of *Lee et al. (2019)* and *El-Gharbawy and Wahdan (2022)*.

Furthermore, *Subikshavarthni and Selvan (2019)* found acidosis as a risk factor for prolonged ICU admission and mortality. Hypotension accompanied with hypoperfusion and electrolytes disturbance may contribute to acidosis in acute OP poisoning.

Serum creatinine level was statistically significant higher in ICU admitted patients and in non-survivors. Additionally, urea showed a significantly higher median value in ICU admitted patients versus ICU-not admitted patients. *Zafar et al., (2017)* published a case of acute OP poisoning complicated with acute renal failure. He stated that OP could affect the kidney through oxidative stress, direct injury to renal tubules and parenchyma, dehydration, and hypovolemia. Myoglobinuria and renal failure may occur due to rhabdomyolysis caused by muscle fasciculation.

Furthermore, *Kaya et al. (2018)* observed renal degenerative changes in kidney of rats exposed to fenthion.

Regarding liver enzymes, SGOT was significantly higher in ICU admitted and expired patients while SGPT was significantly higher in ICU admitted patients. Organophosphorus compounds are metabolized in the liver through oxidation and conjugation with sulphate or glucuronate exposing hepatic cells to oxidative damage. Hence, SGOT and SGPT are expected to be raised in severe OP poisoning cases (*Shivcharan et al., 2023*).

However, data recruited from literature are controversy. *Kollur and Mulimani (2018)* and *Pradhan et al. (2023)* detected significant association between liver enzymes and the severity of acute OP poisoning with marked elevation of liver enzymes in acute OP poisoned patients. On the other hand, *Kang (2009)* did not find significant difference between survivors and non survivors regarding SGOT and SGPT.

Additionally, *Banday et al. (2015)* did not document a strict relationship between liver enzymes and the severity of OP poisoning or clinical outcomes ($p>0.05$).

Statistical analysis of the present study demonstrated that PSS and REMS had statistically significant difference between the studied groups. The median PSS was 3 in ICU admitted patients, 1 in ICU-not admitted patients, 3 in non-survivors, and 2 in survivors.

Furthermore, at cut off > 2 , PSS could predict ICU admission with excellent performance and mortality with good performance. For REMS, the median values were 9, 2, 11, and 2 in ICU admitted, ICU-not admitted, expired, and survived patients, respectively. REMS could predict ICU admission with excellent performance (AUC=0.955) and mortality with good performance (AUC=0.879).

However, REMS is outperforming PSS as it is more applicable and simple and depends on vital signs, GCS, and O₂ saturation.

These findings are in harmony with previous studies. According to *Shahin and Hafez (2020)*, REMS predicted mortality at cut-off > 4 with excellent performance (AUC=0.920). *Shama et al. (2021)* found that the best cut-off value of PSS to predict ICU admission was > 2 with fair performance (AUC=0.711). They also evaluated REMS and found that at

cut-off > 6 , REMS could predict ICU admission with good performance (AUC=0.882) and at cut-off > 9 , it could predict mortality with good performance also (AUC=0.868). Furthermore, *El Sarnagawy et al. (2022)* found PSS median values were 3 and 1 in ICU admitted and ICU-not admitted OP poisoned patients.

Moreover, they identified a value > 2 as the best cut-off to predict ICU admission (with excellent performance; AUC=0.912) and mortality (with good performance; AUC=0.878).

The current study has some limitations. First: the study was retrospective so we did not include some patients because of missed data. Second: the study was conducted in only one poison control center. Third: the relatively small number of the included patients.

CONCLUSION

Based on previous findings, there are many predictors which could help to identify acute OP poisoned patients who are at a significant risk for adverse outcomes. These predictors include PaO₂, pH, HCO₃, blood urea, serum creatinine, SGOT, and SGPT. Furthermore, scoring systems like PSS and REMS could predict ICU admission with excellent performance and mortality with good performance. However, REMS is considered to be superior to PSS as it is easier and more applicable.

RECOMMENDATIONS

Authors recommend considering initial PaO₂, pH, HCO₃, blood urea, serum creatinine, SGOT, and SGPT as simple predictors for adverse outcomes in acute OP poisoning. In addition, REMS is recommended to be applied in the prediction of the outcome of acute OP poisoned patients as it showed excellent and good performance in predicting the need of ICU admission and mortality, respectively, beside it is simple, applicable, and easily calculated. Furthermore, future studies including larger samples are recommended to be conducted in different poison control centers.

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REFERENCES

1. **Abd Elfatah, A. G. S. A.; Ghonem, M. M.; Wahdan, A. A. et al. (2022):** A Proposal for a Novel Scoring System for Triage of Acutely Poisoned Patients at Tanta University Poisoning Treating Center, Egypt. *J. Adv. Med. Res.* 34(20):31–45. DOI: 10.9734/jammr/2022/v34i2031466
2. **Abdel Baseer, K. A.; Gad, E. F. and Abdel Raheem, Y. F. (2021):** Clinical profile and outcome of acute organophosphate poisoning in children of Upper Egypt: A cross-sectional study. *BMC, Pediatr.* 21(1):98. DOI:10.1186/s12887-021-02563.
3. **Acikalin, A.; Dişel, N. R.; Matyar, S. et al. (2017):** Prognostic Factors Determining Morbidity and Mortality in Organophosphate Poisoning. *Pak. J. Med. Sci.*, 33(3):534-539. DOI:10.12669/pjms.333.12395.
4. **Açıkgöz, M. (2021):** Evaluation of Clinical and Laboratory Prognostic Risk Factors in Organophosphate or Carbamate-Poisoned Pediatric Patients. *Eurasian. J. Emerg. Med.*, 20(2):71-8. DOI:10.4274/eajem.galenos.2020.97720.
5. **Amin, D.; Abaza, M.; Azawy, D. et al. (2018):** Morbidity and Mortality Indicators in Acute Organophosphate Poisoning in Zagazig University Hospital, Egypt: Retrospective Study. *Occup. Environ. Med.*, 6:130-140. DOI:10.4236/odem.2018.64011.
6. **Anjana, D. and Neeta, D. (2019):** Predictors of respiratory failure in acute organophosphorus compound poisoning. *Int. J. Health Res. Medico. Leg. Prac.*, 5(1):15-18.
7. **Balmuchu, G.; Mohanty, M. K.; Sahu, M. R. et al. (2023):** Outcome prediction using sequential organ failure assessment (SOFA) score and serum lactate levels in organophosphate poisoning. *J. Fam. Med. Prim. Care*, 12(4):777-782. DOI: 10.4103/jfmpc.jfmpc_1713_22.
8. **Banday, T. H.; Tathineni, B.; Desai, M. S. et al. (2015):** Predictors of Morbidity and Mortality in Organophosphorus Poisoning: A Case Study in Rural Hospital in Karnataka, India. *N. Am. J. Med. Sci.*, 7(6):259-65. DOI:10.4103/1947-2714.159331.
9. **Beltagy, D. M.; Sadek, K. M. and Hafez, A. S. (2018):** Serum β -glucuronidase activity as a biomarker for acute cholinesterase inhibitor pesticide poisoning. *Toxicol. Ind. Health.* 34(12):891–897.
10. **Chaney, A. L. and Marbach, E. P. (1962):** Urea determination by phenol hypochlorite method using Berthelot reaction. *Clin. Chem.* 8:130132.
11. **Chen, J.; Zhang, Y.; Chai, Y. et al. (2021):** Synergistic enhancement of the emergency treatment effect of organophosphate poisoning by a supramolecular strategy. *Chem. Sci.* 12(14):5202-5208.
12. **Dong, N.; Wang, S.; Li, X. (2021):** Prognostic nomogram for the severity of acute organophosphate insecticide self-poisoning: a retrospective observational cohort study. *BMJ. Open.* 11(5):e042765. DOI: 10.1136/bmjopen-2020-042765.
13. **Eddleston, M.; Buckley, N. A.; Eyer, P. et al. (2008):** Management of acute organophosphorus pesticide poisoning. *The Lancet*, 371(9612), 597–607.
14. **Eddleston, M. and Chowdhury, F. R. (2016):** Pharmacological treatment of organophosphorus insecticide poisoning: the old and the (possible) new. *Br. J. Clin. Pharmacol.* 81(3):462–470.
15. **Eisa, H. S.; Nomier, M. A.; Arafa, M. H. et al. (2021):** Predictors of severity and survival in acute cases of organophosphorous poisoning at Zagazig university hospitals: Prospective cohort study. *Toxicol. Int.*, 28:185-201.
16. **Elagamy, S. E. and Gabr, H. M. (2019):** Predictors of the need for Intensive Care Unit admission in acute organophosphorus poisoning: One year prospective study. *EJFSAT.*, 19(4):1–9.
17. **El-Ebiary, A. A.; Gad, S. A.; Wahdan, A. A. et al. (2016):** Clonidine as an adjuvant in the management of acute poisoning by anticholinesterase pesticides. *Hum. Exp. Toxicol.*, 35(4):371-376.
18. **El-Gharbawy, D. and Wahdan, A. (2022):** Predictors of major outcome in acute organophosphorus poisoned cases. *EJFSAT.* 22(4), 99–114.
19. **El-Sarnagawy, G. N.; Abdelnoor, A. A.; Abuelfadl, A. A. et al. (2022):** Comparison between various scoring systems in predicting the need for intensive care unit admission of acute pesticidepoisoned patients. *Environ. Sci. Pollut. Res.*, 29(23):33999–34009.

20. **Fawcett, I. K. and Scott, J. E. (1960):** Phenol hypochlorite method for urea determination. *J. Clin. Pathol.* 13:156-159.
21. **Ganie, S. Y.; Javaid, D.; Hajam, Y. A. et al. (2022):** Mechanisms and treatment strategies of organophosphate pesticide induced neurotoxicity in humans: A critical appraisal. *Toxicolo.*, 472:153181. DOI:10.1016/j.tox.2022.153181.
22. **Hodeib, A. and Khalifa, H. (2020):** Corrected QT interval as a predictor of outcomes in acute organophosphate poisoning cases. *Ain Shams J. Forensic Med. Clin. Toxicol.*, 34(1):34-40.
23. **Houot, O. (1985):** Kinetic determination of creatinine. In: Interpretation of Clinical Laboratory Tests. Siest, G. Henny, J. and Schiele, F. (Eds), 8th Edition. *Biochem. Pub.*, pp. 220-234.
24. **Kamath, S. D. and Gautam, V. K. (2021):** Study of organophosphorus compound poisoning in a tertiary care hospital and the role of Peradeniya Organophosphorus Poisoning scale as a prognostic marker of the outcome. *J. Fam. Med. Prim. Care.*, 10(11):4160-4167. DOI:10.4103/jfmpc.jfmpc_518_21.
25. **Kang, E. J.; Seok, S. J.; Lee, K. H. et al. (2009):** Factors for determining survival in acute organophosphate poisoning. *Korea. J. Intern. Med.* 24(4):362-367. DOI: 10.3904/kjim.2009.24.4.362.
26. **Kaya, Y.; Bas, O.; Hanci, H. et al. (2018):** Acute renal involvement in organophosphate poisoning: histological and immunochemical investigations. *Ren. Fail.*, 40(1): 410-415.
27. **Kim, Y. H.; Yeo, J. H.; Kang, M. J. et al. (2013):** Performance Assessment of the SOFA, APACHE II Scoring System, and SAPS II in Intensive Care Unit Organophosphate Poisoned Patients. *J. Korea. Med. Sci.*, 28(12):1822-1826. DOI: 10.3346/jkms.2013.28.12.1822
28. **Kokholm, G. (1990):** Simultaneous measurements of blood pH, p CO₂, p O₂ and concentrations of hemoglobin and its derivatives-A multicenter study. *Scand. J. Clin. Lab. Investig.*, 50(203): 75-86.
29. **Kollur, B. S. and Mulimani, M. S. (2018):** Study of levels of serum creatine phosphokinase and liver enzymes as a prognostic indicators in acute organophosphorus poisoning. *Int. J. Clin. Biomed. Res.* 4(1):57-61.
30. **Krishna Moorthy, D. G. S. R.; Priya, S. M.; Rajesh, K. et al. (2023):** Performance of new Poisoning Mortality Score in comparison with SOFA and APACHE II scores in acute organophosphate poisoning. *JEMTAC.*, 2023(1):9. DOI:10.5339/jemtac.2023.
31. **Lee, S.; Kim, D.; Kim, T. et al. (2019):** Anion gap and base deficit are predictors of mortality in acute pesticide poisoning. *Hum. Exp. Toxicol.*, 38:185-192.
32. **Maksimović, Z. M.; Jović-Stošić, J.; Vučinić, S.ić-Vukčević, N. et al. (2021):** Acute organophosphate and carbamate pesticide poisonings – a five-year survey from the National Poison Control Center of Serbia. *Drug Chem. Toxicol.*, 46(1): 113-121
33. **Masoud, W.; Heshmat, M.; Soliman, N. et al. (2022):** The role of cortisol and thyroid stimulating hormone in prognosis of acute anticholinesterase pesticides poisoned patients admitted to Tanta Poison Control Center. *Ain Shams J. Forensic Med. Clin. Toxicol.*, 38(1):33-45.
34. **Moffatt, A.; Mohammed, F.; Eddleston, M. et al. (2010):** Hypothermia and fever after organophosphorus poisoning in humans- a prospective case series. *J. Med. Toxicol.*, 6:379-385.
35. **Mohamed, S.; Hasb Elnabi, M.; Moussa, M. et al. (2019):** The accuracy comparison of scoring Systems in the Outcome Prediction of acute organophosphate poisoning. *Ain Shams J. Forensic Med. Clin. Toxicol.*, 33(2):8-15.
36. **Moussa, M. E.; Mohamed, S. A.; Hilal, M. A. et al. (2018):** Predictive value of Triage Vital Signs and Conscious Level for Outcome Evaluation in Acutely Organophosphate Poisoned Patients. *Ain Shams J. Forensic Med. Clin. Toxicol.*, 31:33-40.
37. **Mukherjee, S. and Gupta, R. D. (2020):** Organophosphorus nerve agents: Types, toxicity, and treatments. *J. Toxicol.* 2020:3007984. DOI:10.1155/2020/3007984.
38. **Olsson, T.; Terent, A. and Lind, L. (2004):** Rapid Emergency Medicine score: a new prognostic tool for in-hospital mortality in nonsurgical emergency department patients. *J. Intern. Med.*, 255:579-587. DOI:10.1111/j.1365-2796.2004.01321.x
39. **Oreby, M. and El-Madah, E. (2017):** Prediction of acute organophosphate poisoning using Glasgow coma scale, serum cholinesterase and S100B. *Ain Shams J. Forensic Med. Clin. Toxicol.*, 28(1):100-107.

40. **Pannu, A.; K. Bhalla, A.; Vishnu, R. I. et al. (2021):** Cardiac injury in organophosphate poisoning after acute ingestion. *Toxicol. Res.*, 10(3):446–445. DOI:10.1093/toxres/tfab036
41. **Persson, H. E.; Sjöberg, G. K.; Haines, J. A. et al. (1998):** Poisoning severity score. Grading of acute poisoning. *J. Toxicol. Clin. Toxicol.*, 36(3):205–213.
42. **Pradhan, B.; Pandey, S.; Niroula, A. et al. (2023):** Mean Cholinesterase Level among Organophosphorus Poisoning Patients Visiting the Emergency Department in a Tertiary Care Centre: A Descriptive Cross-sectional Study. *JNMA J. Nepal. Med. Assoc.*, 61(257):72–75.
43. **Pradhan, M.; Upadhyay, H. P.; Shrestha, A. et al. (2022):** Epidemiological Study of Organophosphorus Poisoning at College of Medical Sciences and Teaching Hospital, Bharatpur, Nepal. *JCMS, Nepal*, 18(3):304–310.
44. **Raveendra, K. R. and Mohan, C. N. (2021):** A study of serum amylase as a probable prognostic marker in acute organophosphorus poisoning. *APIK J. Intern. Med.*, 9(2):94–98.
45. **Reddy, B. S.; Skaria, T. G.; Polepalli, S. et al. (2020):** Factors associated with outcomes in organophosphate and carbamate poisoning: a retrospective study. *Toxicol. Res.* 36(3):257–266.
46. **Reitman, S. and Frankel, S. (1957):** A colorimetric method for determination of serum glutamic oxalacetic and purivic transaminase. *Am. J. Clin. Pathol.* 28(1):58-63.
47. **Roberts, D. M. and Aaron, C. K. (2007):** Management of acute organophosphorus pesticide poisoning. *BMJ.* 334(7594):629–634.
48. **Sert, A. İ.; Kılıç, E. T.; Akdemir, M. S. et al. (2019):** Retrospective Analysis of Organophosphate Poisonings in an Intensive Care Unit in Turkey: A Single-Center Study. *Dubai Med. J.*, 1(1-4):13–18. Doi: <https://doi.org/10.1159/000493768>
49. **Shahin, M and Hafez, A. (2020):** Comparison of different scoring systems in poisoning with cholinesterase inhibitors. *MJFMCT.* 28(1):25–42.
50. **Shama, W. S.; El-Gharbawy, D. M.; Wahdan, A. A. et al. (2021):** Assessment of the efficacy of four scoring systems in prediction of acute organophosphorous poisoning outcome. *Tanta Med. J.* 49(3):187–197.
51. **Shivcharan, J.; Banwari, L. and Divya, A. (2023):** Biochemical indicators and the Peradeniya Organophosphate Poisoning scale in prediction and prognosis of organophosphorus poisoning: An observational prospective study. *J. Acute Dis.*, 12(4):133-139. DOI: 10.4103/2221-6189.385679
52. **Sontakke, T. and Kalantri, S. (2023):** Predictors of Mortality in Hospitalized Patients With Pesticide Poisoning. *Cureus.* 15(7):e41284. DOI:10.7759/cureus.41284
53. **Subikshavarthni, S. and Selvan, R. (2019):** Arterial Blood Gas as a Prognostic Tool in Organophosphorus Poisoning Patients—A Prospective Observational Study. *Int. J. Sci. Study.* 7:82-87.
54. **Tadesse, B.; Kibret, H.; Heluf, H. et al. (2023):** Pattern and outcome of acute organophosphate poisoning at health facilities of Harari Region Eastern Ethiopia. *SAGE Open Med.*, 11. DOI:10.1177/ 20503121231216603.
55. **Thakur, D. K.; Mahaseth, R. and Jha, S. (2022):** Predictors of Morbidities in Organophosphate Poisoning. *IJISRT.* 7(3):878-894.
56. **Woo, O. F. (1999):** Organophosphates and carbamates. In: *Poisoning and Drug Overdose.* Anderson, I. B. and Clark, R.F. (Eds.), 3rd Edition. *Appleton & Lange, Stanford*, pp. 224-248.
57. **Zafar, R.; Munawar, K.; Nasrullah, A. et al. (2017):** Renal failure due to organophosphate poisoning: a case report. *Cureus.* 27:523.
58. **Zobeiri, M. (2021):** Serum amylase as a prognostic marker of organophosphate poisoning. *J. Inj Violence Res.*, 13(2):117-120. DOI: 10.5249/jivr.v13i2.1632.

تحديد متنبات النتائج السلبية بين مرضى التسمم الحاد بالفوسفات العضوية

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المقدمة: التسمم الحاد بالفوسفات العضوية هو مشكلة صحية شديدة الأهمية ذات معدلات اعتلال ووفيات عالية.

الهدف من الدراسة: تحديد عوامل يسهل استخدامها للتنبؤ بالنتائج السلبية (دخول وحدة العناية المركزة والوفاة) للتسمم الحاد بالفوسفات العضوية.

المرضى وطرق البحث: أجريت هذه الدراسة على مرضى التسمم الحاد بالفوسفات العضوية الذين تم إدخالهم إلى مركز مكافحة السموم بجامعة طنطا في الفترة من يناير ٢٠٢٣ إلى ديسمبر ٢٠٢٣ حيث تم أخذ التاريخ الشخصى والسمى وتسجيل نتائج الفحص السريري والتحاليل المخبرية وكذلك توثيق نظام قياس شدة التسمم (PSS) ونظام قياس ريمس (REMS) ، وقد تم تقسيم المرضى إلى مجموعات بناء على دخول وحدة العناية المركزة أو حدوث الوفاة.

النتائج: من بين ٦٦ مريضاً، احتاج ١٥ (22.7%) دخول وحدة العناية المركزة وتوفى ٦ (9.1%). وتراوحت أعمار المرضى من ١٨ إلى ٦٦ سنة، ٥٩.١% منهم ذكور، و٨٠.٣% من الريف. وتم تسجيل التسمم بالانتحار والتسمم عن طريق الفم فى 57.6% و 72.6% على التوالي. وكانت قيم معدل النبض والتنفس، والكرياتينين، واليوريا، وانزيمات الجلوتاميك البيروفيك ترانس أمينيز، والجلوتاميك أوكسالوسيتيك ترانس أمينيز أعلى بدلالة احصائية بينما كانت قيم مقياس الوعى جلاسجو، وضغط الأوكسجين الشرياني الجزئي، وحموضة الدم، والكربونات أقل بدلالة احصائية فى مجموعات النتائج السلبية. وتوقع كل من PSS و REMS دخول وحدة العناية المركزة بأداء ممتاز وحدث الوفاة بأداء جيد.

الخلاصة: يمكن لكل من معدل النبض والتنفس، مقياس الوعى جلاسجو، وضغط الأوكسجين الشرياني الجزئي، وحموضة الدم، والكربونات، والكرياتينين، واليوريا، وانزيمات الجلوتاميك البيروفيك ترانس أمينيز والجلوتاميك أوكسالوسيتيك ترانس أمينيز، PSS، REMS التنبؤ بدخول وحدة العناية المركزة أو حدوث الوفاة فى التسمم الحاد بالفوسفات العضوية.