PROBABLE ROLE OF SERUM PROCALCITONIN LEVEL IN PREDICTING NEED FOR MECHANICAL VENTILATION IN ACUTE ORGANOPHOSPHORUS POISONING

***Samah M. Elbastawesy*[1], *Tarek Abdel-Hay Mostafa*[2], *Asmaa A. Ghalwash*[3], *Sohier F. Hasan*[4], *Rasha M. A. Nada*[1]**

[1]Forensic Medicine and Clinical Toxicology Department, Faculty of Medicine, Tanta University, Egypt,
[2]Anesthesiology and Intensive Care Department, Faculty of Medicine, Tanta University, Egypt,
[3]Medical Biochemistry Department, Faculty of Medicine, Tanta University, Egypt,
[4]Emergency Medicine and Traumatology Department, Faculty of Medicine, Tanta University, Egypt.

ABSTRACT

Background: Organophosphorus poisoning (OPs) is a global health issue leading to thousands of mortalities and morbidities annually. Respiratory failure remains the main mechanism of death. With the widespread use of ventilators, respiratory failure has been effectively treated. **Aim of the work:** The present study aimed to measure serum procalcitonin level in acute OPs poisoning and to assess its potential role as an early predictor for mechanical ventilation need in these patients. **Patients and Methods:** The study was conducted on 42 acutely OPs intoxicated patients. Adding to the routine management of acute OPs patients, serum procalcitonin level was measured in all patients within 2-4 hours of exposure. Outcomes were recorded in all patients. Patients were categorized into 2 groups according to need for mechanical ventilation (MV), group 1 (needed MV) and group 2 did not require MV during their management. **Results:** Nearly, 29% of all acutely intoxicated OPs patients required MV. Individuals presenting with lower GCS scores, some muscarinic and nicotinic manifestations were more likely to require intensive care and mechanical ventilation. Median procalcitonin level showed a statistically significant difference between groups that needed MV and those who did not. Admission procalcitonin level was able to predict the need for MV in acute OPs poisoned patients with a cut-off value >0.4ng/ ml, sensitivity = 100.0%, specificity = 83.3%, and overall accuracy = 88.1%. **Conclusions and Recommendations:** Procalcitonin level rather than sepsis marking may additionally have other predictive values. It may be used either even or with other scoring systems and biomarkers in early prediction of the need for MV in acute OPs poisoned patients.

Keywords: *Cholinesterase inhibitors, Pesticides, Respiratory failure, Biomarker*.

Corresponding author: *Dr. Samah M. Elbastawesy*
Email: doctor_samahmaher@yahoo.com,
Samah.elbastawesy@med.tanta.edu.eg
ORCID: 0000-0001-5626-3203

INTRODUCTION

Globally, about three million people are intoxicated by organophosphorus (Ops) insecticide annually, resulting in nearly 300,000 deaths ***(Robb et al., 2023)***. Organophosphate poisoning is prevalent in developing nations, Egypt among them ***(Abdel Baseer et al., 2021)***. Poisoning by OPs occurs through different routes of exposure either inhalation, ingestion, or via skin ***(Ganie et al., 2022)***.

Organophosphorus poisoning causes accumulation of acetylcholine (Ach) at cholinergic synapses through inhibition of esterase enzymes, specifically acetylcholinesterase. Subsequently, excessive Ach induces overstimulation followed by inhibition of cholinergic receptors located at the central nervous system (CNS), autonomic nervous system and neuromuscular junctions. This cholinergic load in addition to oxidative stress induced by OPs, results in different muscarinic, nicotinic and CNS symptoms and signs ***(El-Ebiary et al., 2016; Pang et al., 2019; Reddy et al., 2020)***.

The primary clinical manifestations of organophosphate toxicity involve excessive lacrimation, drooling, increased bronchial secretions, diarrhea, nausea, abdominal discomfort, muscle weakness, twitching, and ultimately, respiratory collapse ***(Vanova et al., 2018, Reddy et al., 2020)***.

Respiratory failure represents the prevailing complication resulting from organophosphate exposure and the most common cause of death in such patients. Respiratory failure in OPs toxicity occurs through various mechanisms. The respiratory center, found in the medulla, houses glutaminergic and muscarinic fibers. Elevated levels of acetylcholine in these regions can potentially inhibit respiratory function ***(Gaspari and Paydarfar, 2011)***. Some studies supported the fact that respiratory failure occurs as a result of organophosphates' direct impact on skeletal muscles, resulting in weakness and necrosis of skeletal muscle fibers. This is further compounded by the excessive stimulation of neuromuscular junctions due to an abundance of acetylcholine ***(John et al., 2003)***.

At all the mechanisms, if the respiratory failure is early diagnosed and adequately managed it improves the mortality rate in OPs intoxicated patients ***(Hodeib and Khalifa, 2020)***. Management of respiratory failure usually requires ventilatory support and mechanical ventilation ***(Giyanwani et al., 2017)***.

Ventilators are lifesaving in OPs poisoned patients however, in many developing countries there is a shortage of medical resources and inadequate numbers of ventilators ***(Krishnamoorthy et al., 2014)***. Hence, it is crucial to discover novel prognostic indicators to predict the necessity for mechanical ventilation in the treatment of patients poisoned by organophosphates.

Procalcitonin (PCT) is a widely recognized biomarker used for the identification of sepsis or infection occurrence ***(Tschiedel et al., 2018)***.

Sepsis stands out as the primary reason for an elevation in PCT levels. Nevertheless, other factors have been noted to increase PCT levels, encompassing severe cardiogenic shock, trauma, severe pancreatitis, certain autoimmune disorders, metastasis, and significant renal and hepatic impairment ***(Sugihara et al., 2017)***.

In addition, elevation of PCT level was reported in some poisoning like acetaminophen intoxication without evidence of any existing infection ***(Nuzzo et al., 2021)***.

THE AIM OF THE WORK

The objective of the current study was to measure serum procalcitonin level in acute OPs poisoning and to assess its potential role as an early predictor for mechanical ventilation need in these patients.

PATIENTS AND METHODS

The research was carried out following approval from the ethics committee of the Faculty of Medicine at Tanta University. Ethical approval number (35551/6/22). The study included 42 patients who experienced acute organophosphate poisoning and were admitted to the Tanta University Poison Control Center from July 2022 to December 2022. Informed written consent was obtained from each patient or their guardians if the patient was unable to participate in the consent process. Patient data were kept confidential by coding each patient.

Inclusion criteria:

The study included acute OPs poisoned patients aged above 18 years. The diagnosis of organophosphate poisoning was confirmed by assessing the patient's medical history, the manifestation of clinical symptoms suggestive of cholinergic toxicity, observed improvement in signs and symptoms following treatment with atropine and oximes, a reduction in serum cholinesterase activity ***(Aygun, 2004)***, and identifiable containers brought by patient attendants.

Exclusion criteria:

The study excluded individuals under 18 years old, asymptomatic patients with a history of recent organophosphate toxicity, pregnant and breastfeeding women, and patients who ingested substances other than organophosphorus compounds. Additionally, individuals with significant medical conditions such as cardiovascular disease, diabetes mellitus, renal or hepatic failure, and thyroid disorders were also excluded from the study. Patients with a history of infection or high-grade fever within the last 2 weeks were also excluded. Intoxicated patients presented more than 4 hours of consuming OPs compound and those who were previously treated for acute organophosphorus poisoning in any medical center before admission were additionally excluded from the study.

Methods

- Participants underwent collection of sociodemographic data, including age, gender, educational background, occupation, and place of residence. The type of ingested OPs, mode, route of exposure and time passed between exposure and hospital admission were also reported. All patients were clinically examined. Vital signs (pulse, blood pressure, respiratory rate and temperature) and Glasgow coma scale (GCs) for consciousness level assessment were additionally recorded. Respiratory, cardiovascular and abdominal examinations were done for all patients.
- All routine laboratory investigations were done for the patients at admission including the analysis of arterial blood gases, liver enzyme levels, kidney function tests, complete blood count (CBC), as well as serum sodium and potassium levels, along with random blood glucose measurements and serum cholinesterase level. Electrocardiogram (ECG) was recorded for all patients. Poison severity was assessed using the poison severity score.
- For assessment of serum procalcitonin (PCT) levels, Blood samples were collected upon admission, within 2-4 hours following organophosphate intoxication. The samples were left to clot at room temperature and then centrifuged at 3000 rpm for 10 minutes. Subsequently, the supernatant was isolated and stored at -80°C until further analysis. The assay was carried out using a QuicKey human PCT enzyme-linked immunosorbent assay (ELISA) kit applying a sandwich approach (Cat. No. E-TSEL-H0002).

  All assays were performed at 450 nm using a plate reader (Multiskan Spectrum, Thermo Labsystems) and were expressed in nanograms (ng) per ml.
- Outcomes of OPs poisoning were measured and reported as either primary (mortality and improvement) or secondary outcomes parameters.

  These parameters included the requirement for intubation and mechanical ventilation, duration of mechanical ventilation, duration of hospitalization, and the quantity of antidotes administered.

STATISTICAL ANALYSIS:

Statistical analysis was conducted using MedCalc Statistical Software version 15.8 (MedCalc Software bvba, Ostend, Belgium; 2015). The Shapiro-Wilk test was employed to assess the distribution of numerical variables for normality. Normally distributed variables were presented as mean ± standard deviation (SD) and compared using independent samples T-test. Abnormally distributed numerical variables were summarized as median and interquartile range (IQR, 25th – 75th percentiles) and compared using the Mann-Whitney test. Spearman's rank-order correlation was utilized to examine the relationship between the initial procalcitonin level upon admission and other numerical variables. Categorical variables were presented as counts and percentages, and the association between two categorical variables was assessed using Pearson's Chi-square test, Fisher's exact test, Fisher-Freeman-Halton exact test, or Cochran Armitage test for trend. Receiver operating characteristics (ROC) curve analysis was performed to evaluate the predictive performance of admission procalcitonin level and determine the appropriate cut-off value, sensitivity, specificity, and overall accuracy. The area under the curve (AUC) was categorized as excellent (0.90-1), good (0.80-0.90), fair (0.70-0.80), or poor (0.60-0.70). Statistical significance was set at $p < 0.05$.

RESULTS

Table (1) presents sociodemographic features and toxicological history among the studied groups. The analysis of the data revealed that 12 patients only needed mechanical ventilation (MV) representing about 28.5% of all acutely intoxicated OPs patients. The higher incidence of acute OPs poisoning was among young males, and farmers in rural areas. No significant disparities were found among the analyzed groups concerning age, gender, residence, and other sociodemographic characteristics. Suicidal ingestion of OPs rodenticide represented the most common mode and method of exposure to OPs but did not show statistically significant associations with the need for MV. However, it is noteworthy that a notable correlation was observed between the specific type of organophosphate utilized and the requirement for mechanical ventilation (p=0.003).

Specifically, the administration of Malathion was linked to an increased likelihood of needing mechanical ventilation when compared to the other types investigated (Chlorpyriphos and Rodenticide).

Table (2) illustrates the clinical characteristics of the studied patients. The Glasgow Coma Scale (GCS) exhibited a noteworthy difference between the two groups. (p < 0.001).

Group 1 (needed MV) had lower median GCs compared to group 2 (didn't need MV) 10 and 15 respectively.

Regarding muscarinic manifestations, the presence of crepitation/wheezy chest (p=0.040), diarrhea (p=0.001), colic (p=0.041), and sweating (p=0.002) showed significant associations with the need for MV. These symptoms were more prevalent in group 1. For nicotinic manifestations, the presence of fasciculation (p=0.040) and muscle weakness (p=0.003) were more common in group 1 and showed significant associations with the need for MV. The severity of poisoning assessed using the Poisoning Severity Score (PSS), demonstrated a notable correlation with the necessity for mechanical ventilation (p<0.001). Group 1 had a higher proportion of individuals with severe poisoning (PSS 2 and 3) compared to Group 2.

Table (1): Sociodemographic features and toxicological history among studied patients.

		Need for MV			**Test of significance**	
		Total (n = 42)	Group 1 Yes (n = 12)	Group 2 No (n = 30)	Test statistic	p-value
Age	Median [IQR]	22.5 [18.0 - 45.0]	24.0 [18.5 - 42.5]	21.5 [18.0 - 45.0]	Z = 0.294	0.773
	Min - Max	18.0 - 75.0	18.0 - 63.0	18.0 - 75.0		
Gender	Male	24 (57.1%)	7 (58.3%)	17 (56.7%)	X^2_{ChS} = 0.010	0.921
	Female	18 (42.9%)	5 (41.7%)	13 (43.3%)		
Occupation	Farmer	21 (50.0%)	5 (41.7%)	16 (53.3%)	X^2_{FFH} = 2.908	0.204
	Student	13 (31.0%)	6 (50.0%)	7 (23.3%)		
	Housewife	8 (19.0%)	1 (8.3%)	7 (23.3%)		
Level of education	Illiterate	16 (38.1%)	4 (33.3%)	12 (40.0%)	X^2_{FFH} = 1.754	0.652
	Read & write	14 (33.3%)	3 (25.0%)	11 (36.7%)		
	Secondary school	7 (16.7%)	3 (25.0%)	4 (13.3%)		
	University	5 (11.9%)	2 (16.7%)	3 (10.0%)		
Residence	rural	27 (64.3%)	7 (58.3%)	20 (66.7%)	FE	0.726
	urban	15 (35.7%)	5 (41.7%)	10 (33.3%)		
mode of poisoning	Accidental	17 (40.5%)	5 (41.7%)	12 (40.0%)	FE	1.000
	Suicidal	25 (59.5%)	7 (58.3%)	18 (60.0%)		
Route of exposure	Inhalation	19 (45.2%)	5 (41.7%)	14 (46.7%)	X^2_{ChS} = 0.086	0.769
	Ingestion	23 (54.8%)	7 (58.3%)	16 (53.3%)		
Type of organophosphate	Chlorpyriphos	9 (21.4%)	0 (0.0%)	9 (30.0%)	X^2_{FFH} = 11.252	0.003*
	Malathion	15 (35.7%)	9 (75.0%) $+	6 (20.0%) $-		
	Rodenticide	18 (42.9%)	3 (25.0%)	15 (50.0%)		
Delay time in hours	Median [IQR]	3.0 [2.0 - 4.0]	3.0 [2.0 – 4.0]	3.0 [2.0 - 4.0]	Z = 0.722	0.483

*MV: mechanical Ventilation; FE: Fisher's exact test; IQR: interquartile range (25^{th} – 75^{th} percentiles); Max: maximum; Min: minimum; n: number; SD: standard deviation; t: independent samples T-test; X^2_{ChS}: Pearson's Chi-square test for independence of observations; X^2_{FFH}: Fisher-Freeman-Halton exact test; Z: Mann-Whitney test; * significant at p-value <0.05; $+ significantly higher frequency than expected by chance based on adjusted residuals; $- significantly lower frequency than expected by chance based on adjusted residuals.*

Table (2): Comparison between studied groups as regard their clinical characteristics.

		Need for MV			Test of significance	
		Total (n = 42)	Group 1 Yes (n = 12)	Group 2 No (n = 30)	Test statistic	p-value
GCS	Median [IQR]	15.0 [12.0 - 15.0]	10.0 [9.0 - 12.0]	15.0 [15.0 - 15.0]	Z = 6.105	<0.001*
	Min - Max	7.0 - 15.0	7.0 - 13.0	13.0 - 15.0		
pulse (beat per minutes)	Mean ± SD	85.5 ± 23.8	84.3 ± 30.3	85.9 ± 21.4	T = 0.167	0.869
	Min - Max	45.0 - 130.0	55.0 - 130.0	45.0 - 120.0		
systolic blood pressure (mmhg)	Mean ± SD	118.6 ± 23.8	119.2 ± 28.1	118.4 ± 22.4	T = 0.097	0.923
	Min - Max	80.0 - 160.0	80.0 - 160.0	80.0 - 160.0		
diastolic blood pressure (mmhg)	Mean ± SD	74.8 ± 16.7	74.2 ± 17.3	75.0 ± 16.8	T 0.144	0.886
	Min - Max	40.0 - 100.0	50.0 - 100.0	40.0 - 100.0		
respiratory rate (cycle/min)	Mean ± SD	21.6 ± 5.2	23.3 ± 5.4	20.9 ± 5.1	T = 1.4	0.169
	Min - Max	11.0 - 34.0	15.0 - 32.0	11.0 - 34.0		
Temperature	Mean ± SD	37.2 ± 0.4	37.4 ± 0.4	37.2 ± 0.3	T = 1.898	0.065
	Min - Max	36.5 - 38.2	37.0 - 38.2	36.5 - 38.1		
Muscarinic manifestations	miosis	34 (81.0%)	12 (100.0%)	22 (73.3%)	FE	0.080
	Crepitations/wheeze	32 (76.2%)	12 (100.0%)	20 (66.7%)	FE	0.040*
	bradycardia	12 (28.6%)	6 (50.0%)	6 (20.0%)	FE	0.069
	hypotension	10 (23.8%)	3 (25.0%)	7 (23.3%)	FE	1.000
	vomiting	36 (85.7%)	12 (100.0%)	24 (80.0%)	FE	0.159
	diarrhea	15 (35.7%)	9 (75.0%)	6 (20.0%)	FE	0.001*
	colic	33 (78.6%)	12 (100.0%)	21 (70.0%)	FE	0.041*
	sweating	19 (45.2%)	10 (83.3%)	9 (30.0%)	X^2_{ChS} = 9.842	0.002*
Nicotinic manifestations	fasciculation	21 (50.0%)	9 (75.0%)	12 (40.0%)	X^2_{ChS} = 4.200	0.040*
	muscle weakness	27 (64.3%)	12 (100.0%)	15 (50.0%)	FE	0.003*
	hypertension	13 (31.0%)	4 (33.3%)	9 (30.0%)	FE	1.000
	tachycardia	17 (40.5%)	4 (33.3%)	13 (43.3%)	FE	0.731
Severity (PSS)	1	12 (28.6%)	0 (0.0%)	12 (40.0%)	X^2_L = 26.281	<0.001*
	2	17 (40.5%)	0 (0.0%)	17 (56.7%)		
	3	13 (31.0%)	12 (100.0%)	1 (3.3%)		

*MV: mechanical Ventilation; FE: Fisher's exact test; IQR: interquartile range (25th – 75th percentiles); Max: maximum; Min: minimum; n: number; SD: standard deviation; t: independent samples T-test; X^2_{ChS}: Pearson's Chi-square test for independence of observations; X^2_L: Chi-square for linear-by-linear association (Cochran Armitage test for trend); Z: Mann-Whitney test; * significant at p-value <0.05; PSS: poison severity score.*

Table (3) compares the laboratory investigations between the studied groups. Among the parameters analyzed, several significant findings were observed. First, the oxygen saturation levels (% O2) revealed a substantial disparity among the groups under examination (p<0.001). Group 1 had lower mean oxygen saturation compared to Group 2. Regarding arterial blood gas parameters, the admission pH (p=0.001) and PCO2 (p<0.001) demonstrated significant differences between the groups. Group 1 had lower pH and higher PCO2 values compared to Group 2. In addition, liver enzyme levels were notably elevated in Group 1 compared to Group 2.

Other laboratory parameters such as random blood sugar, creatinine, electrolytes, CBC parameters and serum cholinesterase level did not illustrate notable discrepancies between both groups. Median procalcitonin levels were 4.4 and 0.2 in group 1 and group 2 respectively, showing a higher statistically significant difference between the group that needed MV than those who did not (p<0.001).

Figure (1) The Receiver Operating Characteristics (ROC) curve analysis was conducted to assess the predictive capability of procalcitonin levels regarding the requirement for mechanical ventilation. The procalcitonin level could potentially serve as an indicator for predicting the requirement of mechanical ventilation in patients with acute organophosphate poisoning with a cut-off value > 0.4ng/ ml, sensitivity = 100.0%, specificity = 83.3%, and overall accuracy = 88.1%.

Table (3): Comparison between studied groups as regard results of laboratory investigations.

		Need for MV			Test of significance	
		Total (n = 42)	Group 1 Yes (n = 12)	Group 2 No (n = 30)	Test statistic	p-value
o2 saturation %	Mean ± SD	90.9 ± 6.4	82.1 ± 3.5	94.4 ± 2.8	T = 11.949	<0.001*
	Min - Max	75.0 - 99.0	75.0 - 88.0	89.0 - 99.0		
PH	Mean ± SD	7.36 ± 0.09	7.28 ± 0.09	7.39 ± 0.08	T = 3.784	0.001*
	Min - Max	7.10 - 7.54	7.10 - 7.39	7.11 - 7.54		
PCO2	Mean ± SD	41.9 ± 9.9	53.3 ± 7.1	37.3 ± 6.7	T = 6.836	<0.001*
	Min - Max	30.0 - 62.2	35.7 - 62.2	30.0 - 62.0		
PO2	Mean ± SD	93.9 ± 14.0	98.3 ± 16.8	92.1 ± 12.6	T = 1.294	0.203
	Min - Max	53.5 - 137.8	68.3 - 137.8	53.5 - 117.0		
HCO3 meq/L	Mean ± SD	23.7 ± 2.4	24.3 ± 1.6	23.4 ± 2.6	T = 1.050	0.300
	Min - Max	19.9 - 31.3	22.0 - 28.0	19.9 - 31.3		
Random blood glucose mg/dl	Mean ± SD	119.1 ± 25.6	110.4 ± 30.1	122.5 ± 23.2	T = 1.405	0.168
	Min - Max	65.0 - 230.0	65.0 - 170.0	90.0 - 230.0		
Sodium level (Na) (mmol/L)	Mean ± SD	139.4 ± 4.0	139.3 ± 5.2	139.4 ± 3.6	T = 0.091	0.929
	Min - Max	132.0 - 145.0	132.0 - 145.0	132.0 - 145.0		
Potassium (K)(mmol/L)	Mean ± SD	3.99 ± 0.60	4.07 ± 0.67	3.96 ± 0.57	T = 0.511	0.612
	Min - Max	2.50 - 5.40	2.50 - 5.40	2.80 - 5.30		
Urea (mmol/L)	Mean ± SD	21.6 ± 6.5	19.8 ± 6.4	22.3 ± 6.5	T = 1.110	0.273
	Min - Max	13.0 - 40.0	13.0 - 31.0	15.0 - 40.0		
Creatinine (mg/dl)	Median [IQR]	1.00 [0.80 - 1.20]	1.10 [0.85 - 1.20]	0.90 [0.80 - 1.20]	Z = 1.297	0.208
	Min - Max	0.50 - 2.10	0.80 - 1.40	0.50 - 2.10		
Alanine transaminase (IU/L)	Median [IQR]	29.0 [21.0 - 58.0]	67.0 [58.5 - 83.5]	25.5 [19.0 - 31.0]	Z = 4.737	<0.001*
	Min - Max	10.0 - 249.0	48.0 - 249.0	10.0 - 63.0		
Aspartate transaminase (IU/L)	Median [IQR]	32.0 [16.0 - 54.0]	63.5 [54.0 - 83.5]	21.0 [16.0 - 33.0]	Z = 4.590	<0.001*
	Min - Max	12.0 - 223.0	38.0 - 223.0	12.0 - 90.0		
Admission haemoglobin %	Median [IQR]	13.0 [12.4 - 13.8]	13.6 [12.6 - 14.5]	13.0 [12.3 - 13.7]	Z = 1.674	0.098
	Min - Max	10.8 - 15.9	11.2 - 15.9	10.8 - 14.3		
Platelets count (*1000)	Mean ± SD	203.7 ± 35.9	201.1 ± 30.3	204.8 ± 38.3	T = 0.301	0.765
	Min - Max	112.0 - 327.0	146.0 - 274.0	112.0 - 327.0		
Leucocytic count (*1000)	Mean ± SD	8.7 ± 3.7	10.5 ± 4.7	8.0 ± 2.9	T = 1.715	0.108
	Min - Max	4.0 - 16.3	4.3 - 16.3	4.0 - 14.2		
Cholinesterase level (IU/L	Mean ± SD	3177.9 ± 903.7	3020.3 ± 973.8	3241.0 ± 883.4	T = 0.711	0.481
	Min - Max	1117.0 - 4898.0	1180.0 - 4111.0	1117.0 - 4898.0		
Procalcitonin level (ng/ml)	Median [IQR]	0.20 [0.10 – 2.20]	4.40 [2.20 – 9.35]	0.20 [0.10 – 0.30]	Z = 4.673	<0.001*
	Min - Max	0.10 - 11.60	0.50 - 11.60	0.10 - 6.20		

*MV: mechanical Ventilation; IQR: interquartile range (25^{th} – 75^{th} percentiles); Max: maximum; Min: minimum; n: number; SD: standard deviation; t: independent samples T-test; Z: Mann-Whitney test; * significant at p-value <0.05; %: percentage; meq/L: milliequivalents per liter ; mg/dl: Milligrams per deciliter; mmol/L: Millimoles per liter; IU/L :International units per liter; ng/ml: nanogram per milliliter.*

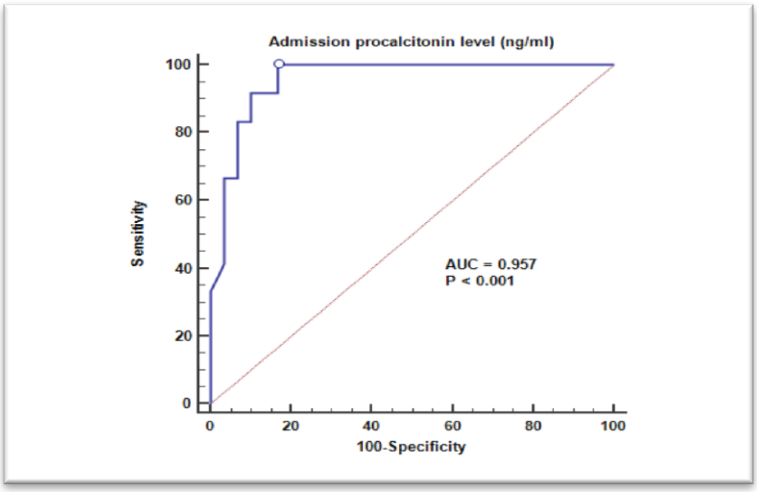

Figure (1): The Receiver Operating Characteristics (ROC) curve analysis to assess the predictive capability of procalcitonin levels regarding the requirement for mechanical ventilation. Area under the curve (AUC) = 0.957 (95% CI: 0.902 to 1.000), the optimal cut-off value >0.4, sensitivity = 100.0%, specificity = 83.3%, and overall accuracy = 88.1%.

Table (4) reveals a comparison between the studied groups in terms of poisoning outcomes. A notable distinction was observed in the quantity of administered atropine and oxime ampoules (p=0.001 and p<0.001). Group 1 received a higher mean number of atropine and oxime ampoules compared to Group 2. Group 1 had a significantly longer median hospital stay period (7.3 days) compared to group 2 (2.3 days) (p<0.001). Group 1 had a significantly higher mortality rate, with the death of 41.7% of patients in that group representing 16.7% of all acute OPs intoxicated patients. All patients in group 2 survived.

Table (5) illustrates Spearman's rank-order correlation among procalcitonin level and other parameters. Delay time, Hospital stay duration, and oximes ampoules number demonstrated a moderate positive correlation with procalcitonin levels (rs=0.343, p=0.026*, rs=0.436, p=0.004*, rs=0.467, p=0.002* respectively). While poison severity (PSS), mechanical ventilation duration, and number of atropine ampoules exhibited a strong positive correlation with procalcitonin levels (rs=0.650, 0.704, 0.520 respectively and p<0.001 in all).

Table (4): Comparison between studied groups as regard poisoning outcomes

		Need MV			Test of significance	
		Total (n = 42)	Group 1 Yes (n = 12)	Group 2 No (n = 30)	Test statistic	p-value
Atropine ampoules number	Mean ± SD	19.2 ± 13.5	34.2 ± 16.0	13.2 ± 5.5	T = 4.431	0.001*
	Min - Max	3.0 - 72.0	8.0 - 72.0	3.0 - 28.0		
oximes ampoules number	Median [IQR]	8.0 [4.0 - 12.0]	16.0 [13.0 - 18.0]	6.00 [0.0 - 8.0]	Z = 4.337	<0.001*
	Min - Max	0.0 - 24.0	4.0 - 24.0	0.0 - 12.0		
hospital stay period by days	Median [IQR]	2.5 [2.0 - 4.8]	7.3 [4.8 - 9.9]	2.3 [2.0 - 2.7]	Z = 3.667	<0.001*
	Min - Max	1.0 - 20.0	1.8 - 20.0	1.0 - 6.5		
Mortality	Survived	35 (83.3%)	5 (41.7%)	30 (100.0%)	FE	<0.001*
	Died	7 (16.7%)	7 (58.3%)	0 (0.0%)		

*MV: mechanical Ventilation; FE: Fisher's exact test; IQR: interquartile range (25^{th} – 75^{th} percentiles); Max: maximum; Min: minimum; n: number; SD: standard deviation; t: independent samples T-test; Z: Mann-Whitney test; * significant at p-value <0.05.*

Table (5): Spearman's rank-order correlation between procalcitonin level and other parameters.

	Admission procalcitonin level (ng/ml)	
	rs	p-value
Age	0.200	0.204
Delay time in hours	0.343	0.026*
Severity (PSS)	0.650	<0.001*
Mechanical ventilation period/d	0.704	<0.001*
Hospital stay period by days	0.436	0.004*
Atropine ampoules number	0.520	<0.001*
Oximes ampoules number	0.467	0.002*

*r_s: Coefficient for Spearman's rank-order correlation (r_s <0.3 weak, r_s =0.3 – 0.7 moderate, r_s >0.7 strong correlation); * significant at p-value <0.05; PSS: poison severity score.*

DISCUSSION

Organophosphate poisoning ranks as the predominant pesticide poisoning encountered in the emergency department, with mortality rates ranging widely from 3% to 40% ***(Xu et al., 2023)***. Respiratory failure, profound coma, and shock are the primary contributors to fatalities resulting from organophosphate poisoning. The widespread utilization of ventilators has proven effective in managing respiratory failure ***(Hulse et al., 2014)***. However, there is shortage of medical resources including ventilators in developing countries ***(Krishnamoorthy et al., 2014)***.

This may delay patients from obtaining their proper chance of survival. Consequently, researchers try to study different risk factors, scores or even biomarkers that can predict patients' need for mechanical ventilators later on. The primary objective of the present study was to measure serum procalcitonin level in acute OPs poisoning and to assess its potential role as an early predictor for mechanical ventilation need in these patients.

In this study, a total of 12 patients needed MV (representing about 28.5% of acute OPs intoxicated patients included in the study).

Giyanwani et al. (2017) reported that 24.6% of acute OPs patients required mechanical ventilation. While 20% and only 14% of these patients needed MV according to ***Patil et al. (2016)*** and ***Soni et al. (2016)*** respectively.

Respiratory failure in OPs poisoning has been explained through different mechanisms. An abundance of acetylcholine may inhibit respiratory function within the brainstem ***(Gaspari and Paydarfar, 2011)***.

Fung and St John (1998) in an animal study revealed that following the administration of acetylcholine into the exposed brainstem, respiratory inhibition and decreased output of the phrenic nerve were observed. The pulmonary edema observed in organophosphate poisoning is attributed to an excess of acetylcholine at muscarinic receptors and pulmonary irritation. Additionally, organophosphate agents elevate the effort required for breathing by inducing airway obstruction ***(Fryer et al., 2004)***. Another mechanism contributing to the deterioration of respiratory function due to organophosphate poisoning involves the stimulation of excitatory activity, which worsens cerebral hypoxia and undermines respiratory function ***(Dickson et al., 2003; Giyanwani et al., 2017)***. Added to these, excessive neuromuscular junction stimulation and direct toxicity of OPs on skeletal muscles, cause later weakness and necrosis of the muscles ***(John et al., 2003, Giyanwani et al., 2017)***.

Acute respiratory failure represents the most communal indication for either intubation and/or mechanical ventilation. Impaired consciousness with the inability of airway protection and respiratory distress may be other added indications for MV in acute ops-poisoned patients ***(Mora Carpio and Mora, 2023)***.

In our study, Malathion intoxication was linked to a greater requirement for mechanical ventilation compared to other types of organophosphates (Chlorpyriphos and Rodenticide).

Malathion is a systemic toxin that is efficiently absorbed through all exposure routes. The predominant cause of death in malathion poisoning cases is respiratory failure. This failure may arise from respiratory depression, along with paralysis of the respiratory muscles and the gradual obstruction of airways caused by increased bronchial secretions. Moreover, commercial malathion formulations frequently include hydrocarbon solvents such as xylene or toluene. Pulmonary aspiration of these hydrocarbon solvents has been reported, causing chemical pneumonitis ***(ATSDR, 2021)***.

Our results revealed that individuals presenting with lower GCS scores, some muscarinic and nicotinic manifestations were more likely to require intensive care and mechanical ventilation. The presence of sweating, diarrhea, colic, wheezy chest, crepitations, muscle weakness, fasciculation and higher poisoning severity were associated with increased need for mechanical ventilation. Peradeniya Organophosphorus poisoning (POP) scale applied a scoring system to determine the early need for ventilation. This scale utilized six clinical parameters, miosis, fasciculations, respiration, bradycardia, and level of consciousness, to evaluate the severity of poisoning ***(Vernekar and Shivaraj, 2017; Chaudhary and Kalmegh, 2018)***.

Chest crepitation and wheeze caused by bronchoconstriction would lead to impaired oxygen transfer and gas exchange in the lung causing hypoxia and hypercapnia and subsequently metabolic acidosis ***(Bhutta et al., 2022)***. These explain significant differences in on-admission oxygen saturation levels, pH, and PCO2 between group 1 and group 2 in this study. All these in addition to muscle weakness may lead to respiratory insufficiency together with unconsciousness may indicate MV in these patients.

The current study revealed a significant elevation of liver enzymes in group 1 than in group 2. Organophosphate insecticide usually causes ultrastructural, biochemical, and mitochondrial damage to hepatocytes, inducing liver injury evidenced by elevation of serum aminotransferases and bilirubin ***(Al-Attar, 2010)***.

The study results showed higher antidotal medication requirements, longer hospital stay duration and a higher risk of mortality in group 1 than group 2. Group 1 had significantly more manifestation of ops toxicity as mentioned before, worse lab investigation results and higher poison severity score. This may indicate more severity of poisoning in this group, more cholinesterase enzyme inhibition with accumulation of excessive Ach. As a result, more doses of antidotes and longer hospital stays were indicated to receive the regimen of Ops poisoning. The severity of poisoning in group 1 would explain the higher risk of mortality. Moreover, patients in the ICU who are MV might have a higher risk of mortality for either risk of sepsis or even complications in artificial ventilation ***(Ramirez-Estrada et al., 2022)***.

In their study, ***Muley et al. (2014)*** declared that acetylcholinesterase levels below 1000, ingestion-to-presentation time of at least 2 hours, and initial oxygen saturation (SpO2) below 85% were positively correlated with the need for ventilation. Additionally, Glasgow Coma Scale (GCS) scores equal to or below 12 were significantly associated with both the necessity for ventilation and hospital stays exceeding 7 days.

Median procalcitonin levels was 4.4 and 0.2 in group 1 and group 2 respectively, showing a higher statistically significant difference between groups that needed MV than those who did not ($p < 0.001$). The procalcitonin level served as an indicator for predicting the necessity of mechanical ventilation in patients with acute organophosphate poisoning with a cut-off value >0.4ng/ ml, sensitivity = 100.0%, specificity = 83.3%, and overall accuracy = 88.1%.

In healthy individuals, serum procalcitonin concentration is usually below 0.1 µg/L ***(Foushee et al., 2012)***. Procalcitonin is a member of the calcitonin superfamily. The rapid increase in procalcitonin levels during infection, along with its association with the severity of the illness, renders it a suitable biomarker for bacterial infection ***(Vijayan et al., 2017)***.

Procalcitonin likewise was higher in patients with acute kidney injury ***(Chun et al., 2019)***. Elevated procalcitonin levels are commonly observed in patients following recent major surgery, severe trauma, severe burns, and prolonged cardiogenic shock. Additionally, patients receiving medications that stimulate cytokine release, such as anti-lymphocyte globulins, alemtuzumab, and granulocyte transfusions, may also exhibit elevated PCT levels ***(Samsudin and Vasikaran, 2017)***. Elevation of PCT level was reported in some poisoning like acetaminophen intoxication without evidence of any existing infection ***(Nuzzo et al., 2021)***. Acetaminophen would produce liver cell injury through inflammatory pathophysiology ***(Lawson et al., 2000)***.

The elevated procalcitonin levels in these patients are attributed to an increase in pro-inflammatory mediators that activate the immune system, resulting in inflammation and subsequent release of procalcitonin into the bloodstream ***(Dahaba et al., 2003)***. There is compelling experimental evidence indicating that acute organophosphate (OPs) intoxication triggers a pronounced inflammatory response. Furthermore, OPs have been found to induce seizures, which correlate with increased levels of cytokines and chemokines ***(Banks and Lein 2012; Camacho-Pérez et al., 2022)***.

Another explanation of PCT elevation is that OPs patients are liable to aspiration pneumonitis and pneumonia. In an autopsy case series involving 85 patients with organophosphate (OPs) insecticide poisoning, more than two-thirds of the 49 patients who died after 24 hours exhibited segmental or lobar consolidation, which was likely attributed to aspiration ***(Kamat et al., 1989)***. Aspiration pneumonia was observed in 82% (27 out of 33) of patients with organophosphate insecticide poisoning admitted to an intensive care unit (ICU) in Germany ***(Hrabetz et al., 2013)***. This would relate the elevation of procalcitonin in acute OPs patients to early subtle infection.

Higher procalcitonin levels in patients who required MV may correlate procalcitonin level itself with poisoning severity. This was proved by Spearman's rank-order correlations. Procalcitonin level was found to be positively correlated with delay time before hospitalization, PSS, and of course the number of atropine and oxime ampoules administered. In addition, higher procalcitonin levels are associated with longer mechanical ventilation and hospital stay durations. Hospital stay would be prolonged due to either the severity of the poisoning which required more antidotal therapy or MV.

CONCLUSION

Procalcitonin level was higher in acute organophosphorus-intoxicated patients who needed MV than those who did not. Moreover, higher procalcitonin levels in the studied patients were associated with longer mechanical ventilation and hospital stay durations. Procalcitonin level, in addition, was found to be positively correlated with longer delay time before hospitalization, PSS and total administered antidotal dose.

RECOMMENDATIONS

Procalcitonin level with other parameters and scoring systems would be helpful in the early prediction of acute OPs patients' need for MV, expressly in areas with limited medical resources.

A multicenter study on a large sample size of acute OPs intoxicated patients would help ascertain the possible role of PCT.

Serial and repeated measurements of PCT would give a clear vision of its role in predicting different outcomes in OPs patients.

Limitations of the study:

- This is a pilot study to correlate procalcitonin levels with mechanical ventilation need in acute OPs poisoned patients. The level was measured once upon hospital admission and hasn't been repeated.
- The study had a small sample size, comprising only a limited number of patients. Many patients were excluded from the study due to many unfitting criteria.

REFRENCES

1. **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.*
2. **Agency for Toxic Substances and Disease Registry (ATSDR) (2021):** Malathion. *Available from: https://wwwn.cdc.gov/*

TSP/substances/ToxSubstance.aspx?toxid=92

3. **Al-Attar, A. M. (2010):** Physiological and histopathological investigations on the effects of alpha-lipoic acid in rats exposed to malathion. *J. Biomed. Biotechnol.,203503.*
4. **Aygun, D. (2004):** Diagnosis in an acute organophosphate poisoning: report of three interesting cases and review of the literature. *Eur. J. Emerg. Med., 11(1):55-58.*
5. **Banks, C. N. and Lein, P. J. (2012):** A review of experimental evidence linking neurotoxic organophosphorus compounds and inflammation. *Neurotoxicol., 33(3):575-584.*
6. **Bhutta, B. S.; Alghoula, F. and Berim, I. (2022):** Hypoxia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK482316/
7. **Camacho-Pérez, M. R.; Covantes-Rosales, C. E.; Toledo-Ibarra, G. A. et al. (2022):** Organophosphorus Pesticides as Modulating Substances of Inflammation through the Cholinergic Pathway. *Int. J. Mol. Sci., 23(9).*
8. **Chaudhary, S. and Kalmegh, R. (2018):** Study of role of prognostic markers in the management of organophosphorus poisoning patients. *Int. J. Res. Med. Sci., 6(6):1996-1999.*
9. **Chun, K.; Chung, W.; Kim, A. J. et al. (2019):** Association between acute kidney injury and serum procalcitonin levels and their diagnostic usefulness in critically ill patients. *Sci. Rep., 9(1):4777.*
10. **Dahaba, A. A.; Rehak, P. H. and List, W. F. (2003):** Procalcitonin and C-reactive protein plasma concentrations in nonseptic uremic patients undergoing hemodialysis. *Intensive Care Med., 29(4):579-583.*
11. **Dickson, E. W.; Bird, S. B.; Gaspari, R. J. et al. (2003):** Diazepam inhibits organophosphate-induced central respiratory depression. *Acad. Emerg. Med., 10(12):1303-1306.*
12. **El-Ebiary, A. A.; Elsharkawy, R. E.; Soliman, N. A. et al. (2016):** N-acetylcysteine in Acute Organophosphorus Pesticide Poisoning: A Randomized, Clinical Trial. *Basic. Clin. Pharmacol. Toxicol., 119(2):222-227.*
13. **Foushee, J. A.; Hope, N. H. and Grace, E. E. (2012):** Applying biomarkers to clinical practice: a guide for utilizing procalcitonin assays. *J. Antimicrob. Chemother., 67(11):2560-2569.*
14. **Fryer, A. D.; Lein, P. J.; Howard, A. S. et al. (2004):** Mechanisms of organophosphate insecticide-induced airway hyperreactivity. *Am. J. Physiol. Lung Cell Mol. Physiol., 286(5):L963-L969.*
15. **Fung, M. L. and St John, W. M. (1998):** Pontine cholinergic respiratory depression in neonatal and young rats. *Life Sci., 62(24):2249-2256.*
16. **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. *Toxicol.,472:153181.*
17. **Gaspari, R. J. and Paydarfar, D.(2011):** Dichlorvos-induced central apnea: effects of selective brainstem exposure in the rat. *Neurotoxicol., 32(2):206-214.*
18. **Giyanwani, P.R.; Zubair, U.; Salam, O. et al. (2017):.** Respiratory Failure Following Organophosphate Poisoning: A Literature Review. *Cureus., 9(9):e1651.*
19. **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.*
20. **Hrabetz, H.; Thiermann, H.; Felgenhauer, N. et al. (2013):** Organophosphate poisoning in the developed world - a single centre experience from here to the millennium. *Chem. Biol. Interact., 206(3):561-568.*
21. **Hulse, E. J.; Davies, J. O.; Simpson, A. J. et al. (2014):** Respiratory complications of organophosphorus nerve agent and insecticide poisoning. Implications for respiratory and critical care. *Am. J. Respir. Crit. Care Med., 190(12):1342-1354.*
22. **John, M.; Oommen, A. and Zachariah, A. (2003):** Muscle injury in organophosphorous poisoning and its role in the development of intermediate syndrome. *Neurotoxicol., 24(1):43-53.*
23. **Kamat, S. R.; Heera, S.; Potdar, P.V. et al. (1989):** Bombay experience in intensive respiratory care over 6 years. *J. Postgrad. Med., 35(3):123-34.*
24. **Krishnamoorthy, V.; Vavilala, M. S. and Mock, C. N. (2014):** The need for ventilators in the developing world: An opportunity to improve care and save lives. *J. Glob Health. 2014;4(1):010303.*
25. **Lawson, J. A.; Farhood, A.; Hopper, R. D. et al. (2000):** The hepatic inflammatory

response after acetaminophen overdose: role of neutrophils. *Toxicol. Sci., 54(2):509-516.*

26.**Mora Carpio, A.L. and Mora, J.I. (2023):** Ventilator Management. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from:*https://www.ncbi.nlm. nih.gov/books/NBK448186/*

27.**Muley, A.; Shah, C.; Lakhani, J. et al. (2014):** To identify morbidity and mortality predictors in acute organophosphate poisoning. *Indian. J. Crit. Care Med., 18(5):297-300.*

28.**Nuzzo, A.; Salem, S.; Malissin, I.et al. (2021):** Plasma procalcitonin may be an early predictor of liver injury in acetaminophen poisoning: A prospective cohort study. *United European Gastroenterol. J., 9(5):571-580.*

29.**Pang, L.; Liu, J.; Li, W. et al. (2019):** Serum ubiquitin C-terminal hydrolase L1 predicts cognitive impairment in patients with acute organophosphorus pesticide poisoning. *J. Clin. Lab. Anal., 33(7):e22947.*

30.**Patil, G.; Murthy, N. and Nikhil, M. (2016):** Contributing Factors for Morbidity and Mortality in Patients with Organophosphate Poisoning on Mechanical Ventilation: A Retrospective Study in a Teaching Hospital. *J. Clin. Diagn. Res.,10(12):Uc18-uc20.*

31.**Ramirez-Estrada, S.; Peña-Lopez, Y.; Vieceli, T. and Rello, J. (2022):** Ventilator-associated events: From surveillance to optimizing management. *J. Intensive Med., 3(3):204-211.*

32.**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.*

33.**Robb, EL.; Regina, A.C. and Baker, M.B. (2023):** Organophosphate Toxicity. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan- *Available from:https://www.ncbi.nlm. nih.gov/ books/NBK470430/*

34.**Samsudin, I. and Vasikaran, S. D. (2017):** Clinical Utility and Measurement of Procalcitonin. *Clin. Biochem. Rev., 38(2):59-68.*

35.**Soni, P.; Solu, M.; Garg, V. et al. (2016):** Organophosphate poisoning predicting the need for mechanical ventilator support. *Int. J. Sci. Study, 4(6):168-172.*

36.**Sugihara, T.; Koda, M.; Okamoto, T. et al. (2017):** Serum Procalcitonin in Patients with Acute Liver Failure. *Yonago Acta Med., 60(1):40-46.*

37.**Tschiedel, E.; Assert, R.; Felderhoff-Müser, U. et al. (2018):** Undue Elevation of Procalcitonin in Pediatric Paracetamol Intoxication is Not Explained by Liver Cell Injury Alone. *Ann. Hepatol., 17(4):631-637.*

38.**Vanova, N.; Pejchal, J.; Herman, D. et al. (2018):** Oxidative stress in organophosphate poisoning: role of standard antidotal therapy. *J. Appl. Toxicol., 38(8):1058-1070.*

39.**Vernekar, P.V. and Shivaraj, K. (2017):** Peradeniya organophosphorus poisoning scale (POP) as a predictor of respiratory failure and mortality in organophosphorus poisoning. *Age (in years)., 18(30):22.*

40.**Vijayan, A. L.; Vanimaya, N.; Ravindran, S. et al. (2017):** Procalcitonin: a promising diagnostic marker for sepsis and antibiotic therapy. *J. Intensive Care., 5:51.*

41.**Xu, B.; Zeng, W.; Chen, F. et al. (2023):** Clinical characteristics and early prediction of mortality risk in patients with acute organophosphate poisoning-induced shock. *Front Med., 9:990934.*

الدور المحتمل لمستوى البروكالسيتونين في الدم في التنبؤ بالحاجة إلى التنفس الصناعي في حالات التسمم الحاد بالمبيدات الفسفورية العضوية

سماح ماهر البسطويسي[١] ، طارق عبدالهادي مصطفى[٢] ، سهير فؤاد حسن[٣] ، أسماء عبد المطلب غلوش[٤] ، رشا محمد علي ندا[١]

[١] قسم الطب الشرعي والسموم الاكلينكية، كلية الطب البشري، جامعة طنطا، مصر.
[٢] قسم التخدير والعناية المركزة، كلية الطب البشري، جامعة طنطا، مصر.
[٣] قسم الطوارئ والإصابات، كلية الطب البشري، جامعة طنطا، مصر.
[٤] قسم الكيمياء الحيوية ، كلية الطب البشري، جامعة طنطا، مصر.

الملخص العربي

المقدمة: يعد التسمم بالمبيدات الفسفورية العضوية مشكلة صحية عالمية تؤدي إلى آلاف الوفيات والأمراض سنويًا. ويظل فشل الجهاز التنفسي هو الآلية الرئيسية للوفاة. ولكن مع انتشار استخدام أجهزة التنفس الصناعي، تم علاج فشل الجهاز التنفسي بشكل فعال فى الكثير من الحالات.

الهدف من الدراسة: كان الهدف من هذه الدراسة هو قياس مستوى البروكالسيتونين في الدم في حالات التسمم الحاد بالمبيدات الفسفورية العضوية وتقييم دوره المحتمل كمؤشر مبكر للحاجة الي التنفس الصناعي لهؤلاء المرضى.

المرضى وطرق البحث: أجريت الدراسة على ٤٢ مريضا مصابين بتسمم حاد بالمبيدات الفسفورية العضوية. وبالإضافة إلى اتباع بروتكول التشخيص و العلاج لمرضى التسمم الحادة بالمبيدات الفسفورية العضوية ، فقد تم قياس مستوى البروكالسيتونين في الدم لدى جميع المرضى خلال ٢-٤ ساعات من التعرض للتسمم ، وتم تسجيل نواتج التسمم في جميع المرضى. تم تصنيف المرضى إلى مجموعتين حسب الحاجة إلى التنفس الصناعي، المجموعة ١ تحتاج إلى تنفس صناعي والمجموعة ٢ لم تحتاج الي تنفس صناعي أثناء علاجهم.

النتائج: ما يقرب من ٢٩٪ من المرضى الذين يعانون من التسمم الحاد بالمبيدات الفسفورية العضوية احتاجوا إلى تنفس صناعي. كان المرضى الذين يعانون من انخفاض درجات الوعي وظهرت عليهم الاعراض المسكارينية والنيكوتينية أكثر عرضة للحاجة إلى العناية المركزة والتنفس الصناعي. أظهر متوسط مستوى البروكالسيتونين وجود فرقا ذا دلالة إحصائية بين المجموعات التي احتاجت إلى تنفس صناعي من أولئك الذين لم يحتاجوا. كان مستوى البروكالسيتونين قادرًا على التنبؤ بالحاجة إلى التنفس الصناعي في المرضى الذين تسمموا بـالمبيدات الفسفورية العضوية بقيمة قطعية> ٠.٤ نانوجرام / مل، وبحساسية = ١٠٠.٠٪، ونوعية = ٨٣.٣٪، وبدقة الإجمالية = ٨٨.١٪.

الخلاصة والتوصيات: بالاضافة الى دور البروكالسيتونين كمؤشر فى حدوث العدوى البكتيرية فقد يكون لمستوى البروكالسيتونين قيم تنبؤية أخرى. حيث يمكن استخدامه في التنبؤ المبكر باحتياجات المرضى المصابين بالتسمم بالمبيدات الفسفورية العضوية الي أجهزة التنفس الصناعي.