## Predictive Value of Triage Vital Signs and Conscious Level for Outcome Evaluation in Acutely Organophosphate Poisoned Patients

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#### Abstract

Triage in Emergency departments requires emergency doctors to make rapid decisions based on their knowledge and experiences. Triage of patients is critical to patient safety, yet no clear information exists by the utility of initial vital signs and conscious level in identifying critically ill acutely organophosphate (OP) poisoned patients. The objective of this study is to determine the relationship between triage vital signs and conscious level in predicting outcome of acutely organophosphate poisoned patients. A retrospective and prospective study was carried out on 200 patients of acute OP poisoning admitted to the Poison Control Center (PCC), Ain Shams University Hospitals. Information including vital signs and Glasgow coma scale (GCS) on admission after obtaining the permission of the director of PCC and the regional ethics committee was collected from the sheets and computerized data base of the patients, an informed written consent has been obtained from each patient or from his/her caregiver for inclusion in the prospective part of the study. The results were revised, coded and organized for statistical analysis.

The study results revealed 180 (90%) patients discharged and 20 (10%) patients died. The study also showed that 8% of patients had fever, 9.5% had tachycardia, 1.5% had bradycardia, 5% had hypotension, 2% had hypertension, 2.5% had tachypnea and 3.5% showed bradypnea. Also 89% of patients had GCS > 8, while 11% of patients had GCS  $\leq$  8. The study showed statistically significant difference between discharged and died patients as regards heart rate, blood pressure, respiratory rate, body temperature and GCS

It could be concluded that heart rate, respiratory rate and coma scale can serve as easily measurable tools for outcome prediction in acutely OP poisoned patients. From the previous results, our study recommends to use these parameters to help emergency physicians to quickly detect poisoned patients with poor outcomes.

#### **Keywords** Triage, Vital signs, Glasgow, Outcome

#### Introduction

rganophosphates (OP) are used as insecticides in agricultural and domestic settings throughout the world (Eddleston et al., 2008).

Poisoning with OP compounds is responsible for great morbidity and mortality in developing countries. (*Sivangnanam*, 2002).

Insufficient control on the importation, production, storage and unsafe use of OP pesticides are the common reasons of poisoning (Suleman et al., 2006). The mortality rate of OP in developing countries is high and is often related to delay in diagnosis or improper management (Eizadi-Mood et al., 2007).

OP poisoning remains an important cause of morbidity and mortality, but no definite parameters have been identified as predictors of outcome. Prediction of morbidity at presentation might help in decision making in places of limited resources like rural settings in developing countries (*Muley et al.*, 2014).

Vital signs are used to measure the body's basic functions. These measurements are taken to help assessment of the general physical health of a person. There are four primary vital signs including body temperature, blood pressure, pulse (heart rate), and breathing rate (respiratory rate) (Swartz,

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2009).

Vital signs play an important role in diagnosis of intoxicated patients since they are the key components of toxic syndromes. However, their role in assessing the severity of poisoning has still lack of evidence (*Lee et al.*, 2008).

The Glasgow coma scale (GCS) remains the most widely used scale to describe the level of consciousness of the victim and is reliable for assessment and prognosis of patients with non-traumatic coma (*Deshpande et al.*, 2012).

The GCS has been shown to be an effective clinical parameter that helps clinicians to predict the outcome of OP poisoning cases in the initial assessment (Forsberg et al., 2010).

#### Aim of the work

The aim of this work is to determine the value of triaging the vital signs and conscious level in predicting outcome of acutely organophosphate poisoned patients presented to the PCC of Ain Shams University Hospitals which may help in improving the course of management and deciding the pathway of care.

#### **Patients and methods**

This is a retrospective and prospective study of 200 acutely OP intoxicated patients admitted to PCC of Ain Shams University Hospitals. An approval from the ethical committee was taken. Information was collected from the sheets and computerized data base of the patients, an informed written consent has been obtained from each patient or from his/her caregiver for inclusion in the prospective part of the study.

#### **Patient 's Selection Criteria:**

The selected patients were of both sexes with acute OP exposure. The diagnosis of OP poisoning was based on the following criteria as guided by *Lee*, (2001):

- History of exposure to OP compounds.
- Characteristic toxidrome of OP toxicity
- Improvement of Muscarinic symptoms and signs after atropine administration.
- Low serum pseudo-cholinestrase activity.

#### **Exclusion Criteria:**

- Patients with history of sever cardiac, pulmonary, renal impairment or nephritic syndrome.
- Patients with any of the following which reduce pseudocholinestrase activity (Parenchymal liver disease, acute infection, metastatic carcinoma, malnutrition, iron deficiency anemia, dermatomyositis, early pregnancy and Patients taking

toxicants (cocaine, carbon disulfide, benzalkonium salts, organic mercury compounds, ciguatoxins, and solanines) or drugs (birth control pills and metoclopramide).

# The following data were collected from the sheet of each patient:

- (A) Sociodemographic data including age and sex of the patients.
- (B) History including mode of poisoning, route of intake and delay time.
- (C) Physical findings on admission:
  - a. Vital signs.
  - b. Neurological assessment according to Glasgow Coma Scale (GCS).
- (D) Outcome of the patients:

The patients were classified according to the outcome into discharged and died patients

#### **Statistical Analysis:**

The obtained results were revised, coded and organized for statistical analysis using SPSS (Statistical Package for Social Science) version 20 software. Mean, standard deviation (±SD) was done for numerical data. Frequency and percentage were obtained for non-numerical data. Comparison between outcome groups was tested by using Chisquare test for qualitative data, and by using Independent t-test for quantitative data. Linear regression analysis was used to identify significant predictors of outcomes. P-value less than 0.05 was considered statistically significant.

#### **Results**

A retrospective and prospective study was carried out on 200 patients of acute OP poisoning admitted to the Poison Control Center (PCC), Ain Shams University Hospitals. In the current study, the mean age was 23.4. There were 44.5% of patients in the age group between 18 to < 40 years and 37% of patients were in the age group < 18 years old, 53% of patients were females and 47% were males (table, 1).

The outcome of the patients as shown in **(table, 2)** was as the following: 180 patients were discharged representing about (90%) and 20 patients were died representing about (10%). As regard mode of poisoning, route of exposure and delay time.

**Table (3)** showed that 66% of patients were intoxicated due to suicidal attempt, while accidental exposure occurred in 34% of patients. Most of patients were exposed to the OP compounds through oral route by ingestion (96.5%), 2% through dermal exposure and only 1.5% through inhalation. The mean delay time of patients was 4.84 hours. The delay time taken between the exposure and asking medical advice ranged from 0.5 up to 36 hours. delay time < 2 hours was in 10.5% of patients, while 74% of

patients showed delay time 2 - 6 hours and 15.5% of patients showed delay time > 6 hours. As regards physical signs,

**Table (4)** showed that 8% of patients presented with fever. There were 9.5% of patients presented with tachycardia, 1.5% of patients presented with bradycardia, 5% of patients had hypotension, 2% of patients had hypotension. Shock observed in 2.5% of patients of the present study. Also respiratory rate abnormality observed in 6% of patients (2.5% had tachypnea and 3.5%

showed bradypnea). Moreover, **table (4)** showed also that 89% of patients had GCS > 8, while 11% of patients had GCS $\le 8$ .

Statistical analysis showed that there was significant difference between discharged and died patients as regards blood pressure, shock development, heart rate, respiratory rate, body temperature and GCS (**Table**, **5**). **Table**, **(6**) showed that predictors of outcome were heart rate, respiratory rate and GCS.

Table (1): Number and percentage of the studied patients as regard sociodemographic data (age and sex)

		Number	Percentage %		
Age in years	< 18	74	37%		
	[18, 40[	89	44.5%		
	[40, 60]	33	16.5%		
	> 60	4	2%		
	Mean ± SD	$23.40 \pm 9.97$			
	Range	0.08 – 63			
Sex	Male	94	47%		
	Female	106	53%		

SD: Standard deviation

Table (2): Number and percentage of the outcome of the studied patients.

Outcome	Frequency	Percentage
Discharged	180	90%
Died	20	10%
Total	200	100%

Table (3): Number and percentage of the studied patients as regard mode of poisoning, route of intake, and delay time.

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History		Number	Percentage %	
Mode of poisoning	Suicidal	132	66%	
	Accidental	68	34%	
Route of intake	Oral	193	96.5%	
	Inhalation	3	1.5%	
	Dermal	4	2%	
Delay time / hours	<2	21	10.5%	
	2-6	148	74%	
	>6	31	15.5%	
	Mean ± SD	$4.84 \pm 1.62$		
	Range	0.5 - 36		

SD: Standard deviation

Table (4): Number and percentage of the studied patients as regard vital signs and conscious level

	ital signs	Number	Percentage %		
•	Normal (120/80)	186	93%		
Blood pressure	(10111111 (120/00)	100	7070		
(mmHg)	Hypertension (>140/90)	4	2%		
\ <b>8</b> /	Hypotension (<90/60)	10	5%		
		Mean ± SD	Range		
	Systolic blood pressure	$112.52 \pm 15.91$	60 - 180		
	Diastolic blood pressure	$70.75 \pm 10.51$	40 – 110		
Shock	Negative	195	97.5%		
	Positive	5	2.5%		
1	Normal (60 – 100)	178	89%		
Heart rate	Tachycardia (>100)	19	9.5%		
(beat/minute)	Bradycardia (<60)	3	1.5%		
	Mean ± SD	$90.98 \pm 26.08$			
	Range	40– 194			
Respiratory rate	Normal (14-16)	188	94%		
(breath/ minute)	Tachypnea (>16)	5	2.5%		
	Bradypnea (<14)	7	3.5%		
	Mean ± SD	$16.13 \pm 5.07$			
	Range	5 – 56			
Temperature	Normal $(37 \pm 0.4^{\circ}C)$	184	92%		
(°C)	Fever (>37.5°C)	16	8%		
	Hypothermia (<35°C)	0	0.0%		
	Mean ± SD	$37.22 \pm 0.38$ $36.5 - 38.8$			
	Range				
	GCS ≤ 8	22			
GCS	GCS > 8	178			
	Mean ± SD	$12.98 \pm 3.50$			
	Range	3 – 15			

GCS: Glasgow Coma Scale

Table (5): Chi- square statistical analysis of vital signs and GCS in relation to outcome.

Vital signs		Discharged N=180		Died N=20		Chi-square test	
		N	%	N	%	X <sup>2</sup>	P-value
Blood pressure	Normal (120/80)	171	95%	15	75%	18.996	< 0.001
(mmHg)	Hypertension (>140/90)	4	2.2%	0	0%		
	Hypotension (<90/60)	5	2.8%	5	25%		
Shock	Negative	178	98.9%	17	85%	14.245	< 0.001
	Positive	2	1.1%	3	15%		
Heart rate beat/min	Normal (60 – 100)	161	89.4%	17	85%	29.151	< 0.001
	Tachycardia (>100)	19	10.6%	0	0%		
	Bradycardia (<60)	0	0%	3	15%		
Respiratory rate breath/min	Normal (14-16)	177	98.4%	11	55%	60.167	< 0.001
	Tachypnea (>16)	1	0.5%	4	20%		
	Bradypnea (<14)	2	1.1%	5	25%		
Temperature	Normal $(37 \pm 0.4^{\circ}C)$	168	93.35	16	80%	4.348	0.037
(°C)	Fever (>37.5°C)	12	6.7%	4	20%		
	Hypothermia (<35°C)	0	0%	0	0%		
GCS	≤8	5	2.8%	17	85%	124.29	< 0.001
	> 8	175	97.2%	3	15%		
	Mean $\pm$ SD	13.850± 2.298		350± 2.298   5.200± 2.802			

<sup>\*</sup>P is considered statistically significant if  $\leq 0.05$ . GCS: Glasgow Coma Scale

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Model	Unstandardized Coefficients		Standardized Coefficients	T	P- value		
	В	Std. Error	Beta				
<b>Blood Pressure</b>	-0.001	0.001	-0.037	-0.760	0.448		
Heart rate	-0.002	0.001	-0.197	-3.564	< 0.001		
Respiratory rate	0.008	0.003	0.128	2.326	0.021		
<b>Body temperature</b>	0.013	0.041	0.016	0.306	0.760		
GCS	-0.062	0.004	-0.725	-14.543	< 0.001		

Table (6): Linear regression analysis of vital signs and coma in relation to outcome.

#### GCS: Glasgow Coma Scale

#### Discussion

Acute OP poisoning may be a cause of morbidity and mortality across many countries including Egypt. This study aimed at determining the value of triage vital signs and conscious level in predicting the outcome of patients with acute OP poisoning.

In this study 180 patients (90%) improved and were discharged and 20 patients (10%) died during treatment in the PCC. This was in agreement with results reported by Bilal et al. (2014) where the recovery in their results was observed in 90% of cases and death was observed in 10% cases. This ratio was close to this obtained by Godhwani et al. (2004) where it was 12%. Yamashita et al. (1997) reported a higher rate of 25%, while Dharmani and Jaga, (2005) reported fatality in hospital-based surveys of OP poisoning as high as 46%. Deaths due to OP poisoning remain high despite optimal respiratory care with mechanical ventilation and oxygen supplementation, mortality rate in different studies ranged from 12 - 27.6 % (Johnson et al., 2000). In cases of heavy OP exposure death is usually related to respiratory failure, death may also result from cardiac arrest, due to cardiac dysrhythmias and various degrees of heart block (Asari et al., 2004).

As regards the vital signs 9.5% of patients presented with tachycardia and 1.5% of patients presented with bradycardia. Concerning the blood pressure 5% of patients had hypotension, 2% of patients had hypertension, Vijayakumar et al. (2011) reported in their study that "tachycardia was found in 60% of patients while bradycardia was found in 10%. Hypertension was found in 35% of cases and hypotension was found in 10% of cases". Banerjee et al. (2012) reported occurrence of hypertension in 9%, bradycardia in 38% and tachycardia in 8%. The variations in the frequency of tachycardia and bradycardia between the studies may be due to differences in delay time. Following the exposure to OP, initially tachycardia occurs due to sympathetic stimulation, and then followed by by parasympathetic bradycardia stimulation (Gündüz et al., 2015).

In the current study respiratory rate abnormalities observed in 6% of patients (2.5% had tachypnea and 3.5% showed bradypnea). In study

done by *Banerjee et al.* (2012) tachypnea was recorded in 35% of patients.

In the current study most of patients (92%) had normal temperature and 8% had hyperthermia. This was in contrast to the study done by *Moffatt et al.* (2010) in which there was initial hypothermia affecting most patients under the study and the study concluded that OP poisoning causes an initial hypothermia, and this is followed by a period of normal to high body temperature. Many factors take part in this fever like lower respiratory tract infection, drug administration as atropine, convulsion or agitation and the use of physical restraint.

The present study showed that there was statistically significant difference between the discharged and died patients as regards blood pressure, incidence of shock, heart rate, respiratory rate and body temperature where hypotension, shock, bradycardia, bradypnea and fever observed more in died patients than discharged patients. This was similar to the results observed by Lee et al. (2008) and Yu et al. (2012) who mentioned that there were significant differences in heart rate, systolic blood pressure, respiratory rate and body temperature between survivors and non-survivors. Also, this was in agreement with Lee et al. (2013) study in which there was significant association between the mortality and decrease in respiratory rate. Severe and refractory hypotension is a leading cause of death in OP poisoning. Although shock is mainly due to vasodilatation, cardiac ischemia with cardiogenic shock was also recorded as a leading cause of death after malathion toxicity (Peter et al., 2014). Also Tintinalli (2010) reported that shock causes hypoxia resulting in accumulation of pyruvate which converted to lactate causing lactic acidosis. Hyperthermia lead to tachycardia and tachypnea as blood pressure drops and the heart attempts to maintain adequate circulation. The decrease in blood pressure can then cause blood vessels to contract resulting in a pale or bluish skin color and seizures in advanced cases with organ failure, unconsciousness and death Hildebrandt et al. (2002). Also, Baumann and Strout, (2007) found that the Emergency Severity Index triage

<sup>\*</sup>P is considered statistically significant if  $\leq 0.05$ .

score, which incorporates vital signs into its algorithm, accurately assesses the risk of patients for hospitalization and mortality. Lam et al. (2010) reported that the heart does not pump enough blood during bradycardia and can be life-threatening with resultant heart failure, sudden cardiac arrest or sudden death. While tachycardia vary in severity depending on the rate and duration. Possible complications include blood clots that can cause a stroke or heart attack, heart failure and sudden associated ventricular death, usually with tachycardia or ventricular fibrillation. While Edmonds et al. (2002) found that in the ED, considerable interobserver variability exists in the measurement of vital signs, with that by sequential observers differing as little as 10 to 15% for heart rate and as much as 35% for respiratory rate which made us must be very careful in evaluation the poisoned cases severity by using vital signs only.

According to the current study there were 89% of patients had GCS more than 8, while 11% of patients with GCS less than or equal to 8. These results go with those of Unverir et al. (2006) and Lee et al. (2008). While Cook et al. (1998) reported that 64% of the patients referring to the emergency department due to poisoning had GCS less than or equal to 8. GCS is now used in several emergency departments as an indicator for the status of the central nervous system regardless of their primary etiology. Poisoning with drugs influences the biochemical substances of the brain and causes brain damage. This may change the level of consciousness as well (Mood et al., 2011). In the current study there was statistically significant difference between discharged and died patients as regard GCS. There were 85% of died had GCS less than or equal to 8. This was similar to results observed by Lee et al. (2008), Yu et al. (2012) and Muley et al. (2014) who mentioned that there was significant difference in GCS between survivors and non-survivors. Also Budhathoki et al. (2009) mentioned that GCS less than 8 had been more associated with mortality in children presenting with poisoning more frequently. Moreover Russell and Shobhan, (2009) found that a GCS of 8 or less is a useful guide for the requirement of endotracheal intubation where the cause of coma is poisoning.

In the present study the mean of GCS values were 5.2 for patients who died and 13.8 for discharged patients. This was similar to results observed by *Basar et al.* (2011) where they studied acutely organophosphrous (OP)-poisoned patients admitted within 24 hours after exposure and found that the mean GCS values were 4 for patients who had died and 13 for discharged patients and there was a statistical correlation between GCS values and mortality, and low values of GCS indicate the

potential for development of respiratory insufficiency and bad prognosis. Also *Kennon and Vikhyat*, (2004) had studied 39 poisoned patients and found that GCS is a reliable tool for the evaluation of mental status of poisoning patients in the emergency department.

In the current study, linear regression analysis identified that the heart rate, respiratory rate and coma scale are significant predictors of outcome while blood pressure and body temperature failed to be a predictor. This result coincides with those of Hsin et al. (2008) who reported that abnormal vital signs were significant predictors of poisoning-related fatalities. Also Yu et al. (2012) denoted that the patients with extremely abnormal vital signs had the greatest risk of inhospital mortality. Bradycardia is recorded more frequently in patients with delayed presentation to the hospital and this finding is strongly associated with mortality (Gündüz et al., 2015). In Lee et al. (2013) study there was significant association between the mortality and decrease in respiratory rate which reflect that the respiratory insufficiency is important leading cause of death in OP poisoning. Similarly, Davies et al. (2008) reported that GCS was effective at predicting outcome in acute organophosphate poisoning and patients presenting with a GCS  $\leq$  13 need intensive monitoring and treatment. Sam et al. (2009) reported that GCS can be applied in predicting mortality in OP poisoning. Muley et al. (2014) mentioned that GCS  $\leq$  12 had been significantly associated with morbidity.

#### Conclusion

Heart rate, respiratory rate and GCS are easily measurable and useful parameters for predicting OP poisoning-related fatality. Such objective information is routinely collected in the emergency department and can help emergency physicians to quickly detect the severity of OP poisoned patients and predict the poor outcomes of those who need intensive care admission.

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#### References

Asari Y, Kamijyo T and Soma K (2004): Changes in the heamodynamic state of patients with acute lethal organophosphate poisoning. Vet Hum Toxicol. 46(1): 5-9.

Banerjee I, Tripathi S and Roy A (2012): Clinicoepidemiological characteristics of patients presenting with organophosphorus poisoning. North American Journal of Medical Sciences. 4 (3): 147-150, doi: 10.4103/1947-2714.93884

Basar C, Ali D, Mesut Y et al., (2011):"The prognostic value of the Glasgow coma scale, serum

- acetylcholinesterase and leukocyte levels in acute organophosphorus poisoning."Ann. Saudi Med. 31(2): 163–166.
- Baumann M and Strout T (2007): "Triage of geriatric patients in the emergency department: validity and survival with the Emergency Severity Index." Ann. Emerg. Med. 49(2):234-240.
- Bilal M, Khan Y, Ali S et al., (2014): The pattern of organophosphorus poisoning and its short term outcomes in various socioeconomic groups. KJMS.7 (1): 11-16.
- Budhathoki S, Poudel P, Shah D et al., (2009): "Clinical profile and outcome of children presenting with poisoning or intoxication: a hospital based study." Nepal. Medical College Journal. 11(3): 170–175.
- Cook S, Moeschler O, Michaud K et al., (1998): Acute opiate overdose: Characteristics of 190 consecutive cases. Addiction. 93: 1559–1565.
- Davies J, Eddleston M, Buckley N et al., (2008): Predicting Outcome in Acute Organophosphorus Poisoning with a Poison Severity Score or the Glasgow Coma Scale QJM. 101(5): 371–379. doi:10.1093/qjmed/hcn014.
- Deshpande A, Gaikwad N and Deshpande S (2012): Study of Glasgow Coma Scale Score and QTc Interval in Prognosis of Organophosphate Compound Poisoning. Indian Journal of Clinical Medicine.3: 25–31.
- Dharmani C and Jaga K (2005): Epidemiology of acute organophosphate poisoning in hospital emergency room patients. Rev Environ Health. 20(3): 215-247.
- Eddleston M, Buckley N, Eyer P et al., (2008): Medical management of acute organophosphorus pesticide self-poisoning. Lancet. 371:597–607.
- Edmonds Z, Mower W, Lovato L et al., (2002):"The reliability of vital sign measurements."Ann Emerg Med. 39(3):233-237.
- Eizadi-Mood E, Saghaei M and Jabalameli M (2007):"Predicting outcomes in organophosphate poisoning based on APACHE II and modified APACHE II scores". Hum. Exp. Toxicol. 26(7): 573-578.
- Forsberg S, Hojer J and Ludwigs U (2010): Prognosis in patients presenting with nontraumatic coma. Crit Care. 14:333.
- Godhwani S, Godhwani S and Tulsiani K (2004):

  Management of organic insecticide poisining in intensive care unit (I.C.U).Indian J. Anaesth. 48(4): 295-393.
- Gündüz E, Dursun R, Icer M et al., (2015): Factors affecting mortality in patients with organophosphate poisoning. Journal of Pakistan Medical Association. 65(9): 967-972.
- Hildebrandt B, Wust P, Ahlers O et al., (2002): "The cellular and molecular basis of hyperthermia." Critical Reviews in Oncology/Hematology. 43(1):33–56.
- Hsin L, Hung L, Steve Y et al., (2008): "Presentations of patients of poisoning and predictors of poisoning related fatality: finding from a hospital based prospective study." BMC Public Health. 8:7-9.

- Johnson M, Jacobsen D, Meredith Tet al., (2000): Evaluation of antidotes for poisoning in organophosphorus pesticides. Emerg Med. 12(1):22-37.
- Kennon H. and Vikhyat S (2004): "Reliability of the Glasgow Coma Scale for the emergency department evaluation of poisoned patients." Hum. Exp. Toxicol. 23(4): 197-200.
- Lam S, Lau A and Yan W (2010): "Over 8 years experience on severe acute poisoning requiring intensive care in Hong Kong. China." Hum. Exp. Toxicol. 29: 757-765.
- Lee H, Lin H, Yeh S et al., (2008): Presentations of patients of poisoning and predictors of poisoning-related fatality: Findings from a hospital-based prospective study. BMC Public Health. 8:7.
- Lee J, Lee Y, Park Y et al., (2013): The difference in C-reactive protein value between initial and 24 hours follow-up (D-CRP) data as a predictor of mortality in organophosphate poisoned patients, Clinical Toxicology. 51:29–34, doi: 10.3109/15563650.2012.745939
- Lee P (2001): Clinical features of patients with acute organophosphate poisoning requiring intensive care. ICU Med. 27:694-699.
- Moffatt A, Mohammed F, Eddleston M et al., (2010): Hypothermia and fever after organophosphorus poisoning in humans—a prospective case series, Journal of Medical Toxicology. 6(4): 379-385, doi: 10.1007/s13181-010-0012-y
- Mood N, Sabzghabaee A, Yadegarfar G et al., (2011): Glasgow Coma Scale and Its Components on Admission: Are They Valuable Prognostic Tools in AcuteMixed Drug Poisoning?. Critical Care Research and Practice, Article ID 952956, 5 pages doi:10.1155/2011/952956.
- Muley A, Shah C, Lakhani J et al., (2014): To identify morbidity and mortality predictors in acute organophosphate poisoning. Indian J Crit Care Med. 18:297-300.
- Peter J, Sudarsan T and Moran J (2014): Clinical features of organophosphate poisoning: A review of different classification systems and approaches, Indian J Crit Care Med. 18(11): 735–745, doi: 10.4103/0972-5229.144017
- Russell D and Shobhan T (2009): "Decreased Glasgow Coma Scale Score Does Not Mandate Endotracheal Intubation in the Emergency Department" J. Emerg. Med. 37(4):451-455.
- Sam K, Kondabolu K, Pati D et al., (2009): Poisoning severity score, APACHE II and GCS: Effective clinical indices for estimating severity and predicting outcome of acute organophosphorus and carbamate poisoning. J Forensic Leg Med.16:239-286.
- Sivangnanam S (2002): Potential therapeutic agents in the management of organophosphate poisoning. Crit Care. 6:260–1.
- Suleman M, Jibran R and Rai M (2006): The analysis of organophosphate poisoning case treated at Bhawal

- Victoria Hospital, Bahawalpur in 2000-2003. Pak J Med Sci. 22:244-249.
- Swartz M (2009): The physical examination, textbook of physical diagnosis, history and examination with student consult, 6th Edition. Chapter 7, Saunders Elsevier, Philadelphia: 129-131.
- Tintinalli E (2010): Shock In Emergency Medicine. New York: McGraw-Hill Companies. P.P. 168-170.
- Unverir P, Atilla R, Karcioglu O et al., (2006): "A retrospective analysis of antidepressant poisonings in the emergency department: 11-year experience," Human and Experimental Toxicology. 25(10): 605–612.
- Vijayakumar S, Fareedullah M, Kumar E et al., (2011): A prospective study on electrocardiographic findings of patients with organophosphorus poisoning, Cardiovasc Toxicol. 11: 113–117, doi: 10.1007/s12012-011-9104-4
- Yamashita M, Tanaka J and Ando Y (1997): Human mortality in organophosphate poisonings. Vet Hum Toxicol. 39(2):84-85.
- Yu J, Weng Y, Chen K et al., (2012): Triage vital signs predict in-hospital mortality among emergency department patients with acute poisoning: a case control study BMC Health Services Research. 12:262-8.

### الملخص العربي

# القيمة التنبؤية لفحص العلامات الحيوية ومستوى الوعى لتقييم النتائج في مرضى التسمم الحاد بالمركبات الفسفورية العضوية

منى القطب موسى ١ و سهير على محمد و مها عبدالحميد هلال و مروه أحمد حسب النبي ٢ و نايل عبدالحميد زكى ٣

الفحص في أقسام الطوارئ يتطلب من طبيب الطوارئ إتخاذ قرارات سريعة بناء على معرفتة وخبراته. فحص المرضى أمر بالغ الأهمية لسلامة المرضى، ولكن لا توجد معلومات واضحة فيما يتعلق بجدوى العلامات الحيوية الأولية في تحديد الحالات المتدهورة من حالات التسمم الحاد بالمركبات الفسفورية العضوية . في الدراسات الحديثة لعلم السموم الاكلينيكية، العلامات الحيوية تلعب دورا هاما في التشخيص لأنما هي المكونات الرئيسية لمتلازمات السمية. وبالرغم من ذلك يعد دورها في تقييم شدة المرض في حالات التسمم ما يزال غير كافي. يعد التشخيص المبكر وبدء العلاج المناسب السريع في غرفة الطوارئ من اجل خفض معدلات الاعتلال والوفيات في حالات التسمم الحاد بالمركبات الفسفورية العضوية . ولذلك تحدف هذة الدراسة الى تحديد العلاقة بين فحص العلامات الحيوية ومستوى الوعى في توقع النتائج للمرضى الذين يعانون من التسمم الحاد بالمركبات الفسفورية العضوية. تم عمل هذه الدراسة باثر رجعي ومستقبلي على ٢٠٠ مريض من حالات التسمم الحاد بالمركبات الفسفورية العضوية بمركز علاج التسمم بمستشفيات جامعة عين شمس. وتم تقسيم المرضى الى مجموعتين رئيسيتين (حالات تم خروجها و وفيات). وقد تم جمع البيانات من ملفات المرضى وقاعدة الملفات الالكترونية بالمركز والتي تشمل: العمر، الجنس، أسلوب التّسمّم, طريق التّعرّض, وقت الدّخول بعد التعرض للتسمم، النبض، و ضغط الدم، ودرجة الحرارة ومعدل التنفس و تقييم درجة وعي المريض وفقا الي مقياس جلاسكو. و قد كشفت نتائج البحث ان ١٨٠ (٩٠٪) من المرضي تم خروجهم و ٢٠ (١٠٪) من المرضى قد توفوا. وأظهرت الدراسة أيضا أن ٨٪ من المرضى كان لديهم ارتفاع في درجة حرارة الجسم، وكان ٩,٥٪ يعانوا من سرعة في دقات القلب، وكان ١,٥٪ بطء في ضربات القلب، وكان هناك انخفاض ضغط الدم في ٥٪، وكان ٢٪ يعاني من ارتفاع ضغط الدم، وأظهر ٢,٥٪ زيادة في معدل التنفس و ٣٠٥٪ كان لديهم بطء في معدل التنفس. أيضا ٨٩٪ من المرضى كانت درجة وعيهم اكبر من ٨٪ بمقياس جلاسكو، بينما ١١٪ فيما يتعلق بمعدل ضربات القلب وضغط الدم ، معدل التنفس،درجة حرارة الجسم ومقياس جلاسكو. وبناء على ذلك نستنتج انه من الممكن ان يكون معدل ضربات القلب و معدل التنفس ومقياس الغيبوبة بمثابة وسائل سهلة لقياس التنبؤ بنتائج حالات التسمم الحاد بالمركبات الفسفورية العضوية و تحديد درجة خطورتة .و توصى هذه الدراسة بإستخدام هذه الوسائل لمساعدة اطباء الطوارئ في تحديد مرضى التسمم بالنتائج الضعيفة.

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