DIAGNOSTIC AND PROGNOSTIC SIGNIFICANCE OF POISONING SEVERITY SCORE, GLASGOW COMA SCALE, VITAL SIGNS, AND ENZYMATIC BIOMARKERS IN CASES WITH ACUTE ANTICHOLINESTERASE INSECTICIDES POISONING

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ABSTRACT

Introduction: Organophosphate (OPC) and carbamate (CMC) compounds are highly toxic anticholinesterase (AntiChE) pesticides, extensively used worldwide, and still responsible for poisoning epidemics. Multi-organ dysfunctions have been reported following AntiChE poisoning. **Aim:** This study aimed to assess the correlation between poisoning severity score (PSS), Glasgow coma scale (GCS), and butyrylcholinesterase (BuChE) levels with clinical and laboratory changes in adult patients with acute AntiChE intoxication. **Subjects and methods:** This study included 25 individuals in healthy-control-group (HC-group) and 75 patients in AntiChE-group. According to poisoning severity criteria, patients were allocated into mild, moderate, or severe intoxicated-group. Gender, age, and causative substance data were reported. Clinical parameters like grade of PSS, delay in hospital arrival time (DHA), vital signs functions, GCS score, and length of hospital stay (LHS) were measured. The blood levels of biochemical parameters (pH, sodium, potassium, and random blood glucose) and enzymatic biomarkers (BuChE, cardiac creatine kinase-myocardial band and cardiac troponin I, pancreatic amylase and lipase, hepatic aspartate and alanine aminotransferases, and kidney urea and creatinine) were estimated. **Results:** Cases were mostly females, aged 23.82±0.82-year, and intoxicated by OPC, especially malathion. The commonest clinical findings were minor manifestations of PSS, DHA for 2-hour, drowsy GCS level, and relatively short LHS for ≤24-hour, while vital signs abnormalities predominantly included tachycardia, hypertension, tachypnea, and hyperthermia. The main biochemical abnormalities were metabolic acidosis, hypernatremia, hypokalemia, and hyperglycemia. Low BuChE levels were detected in all cases, whereas increased enzymatic biomarkers levels were noticed in some cases. On admission, the proportions and mean values of overall clinical and laboratory parameters showed statistically significant differences among the three intoxicated-groups and between AntiChE-group and HC-group. Additionally, PSS grade, GCS score, and degree of BuChE inhibition significantly correlated with DHA, all vital signs, LHS, and overall laboratory parameters as well as between each other. At discharge, the initial clinical and laboratory abnormalities were markedly improved and showed statistically insignificant differences from HC-group except for BuChE levels remained significantly low. **Conclusion:** Although the PSS, GCS, and BuChE seem similarly useful clinical indices at predicting severity of AntiChE poisoning, however, the efficacy of PSS outperform the GCS and BuChE effectiveness.

Keywords: Anticholinesterase poisoning**,** Organophosphate and Carbamate Intoxication, Clinical and Laboratory Abnormalities, Poisoning Severity Score, Glasgow Coma Scale, Predicting Severity of AntiChE Poisoning

INTRODUCTION

Pesticide compounds include several diverse groups or mixtures of chemicals which play an important economic role in preventing, destroying, or controlling a broad spectrum of potentially hazardous pests. The most widely used classes of pesticides are organophosphates, carbamates, and pyrethrins or pyrethroids compounds **(Erdman, 2004).**

Organophosphate compounds (OPC) and carbamate compounds (CMC) are spontaneously degradable anticholinesterase (AntiChE) pesticides synthesized to replace the more toxic organochlorine substances which persist and accumulate in the environment **(Colović et al., 2013).**

The OPC and CMC are extensively utilized all over the world for different agricultural, industrial, and domestic purposes. Despite the development of the less toxic classes of pesticides such as pyrethroids and neonicotinoids, the uses of OPC and CMC in urban settings are still high due to their great efficacy, relatively low cost, and lack of bioaccumulation in the environment **(Costa, 2006).**

The OPC and CMC are highly toxic acetylcholinesterase-inhibiting pesticides that continue to be intensively responsible for poisoning epidemics, particularly in developing countries **(Aslan et al., 2011).** The easy availability of such substances commonly produces human toxicity by accidental and suicidal manner **(Sungur and Güven, 2001).**

Acute poisonings by the AntiChE agents, particularly OPC and CMC, are an important life-threatening clinical problem and commonly responsible for considerable hospital-related admission, morbidity, and mortality worldwide **(Sam et al., 2009; Muley et al., 2014).** According to the estimated data of the World Health Organization, around three million cases with pesticide poisonings occur each year, resulting in approximately 250,000 to 370,000 annual deaths throughout the world **(Goldsmith et al., 2016).** In Egypt, a total of 20,300 AntiChE-poisoned cases were recorded over a period of 17-year (1966-1982) with an average of 1194 AntiChE-poisoned individuals/year, while in year 2006 the number of cases was increased; reached approximately 3 times (3564 persons) those occurred annually during the past decades **(Mansour and Gamalludin, 2008).**

The OPC and CMC act as irreversible and reversible cholinesterase inhibitors, respectively, causing excessive accumulation of the neurotransmitter acetylcholine within the synaptic cleft with subsequent overstimulation of muscarinic and nicotinic receptors present in the peripheral and central nervous system, producing cholinergic toxidrome **(Colović et al., 2013; Lee et al., 2015).**

Clinical manifestations of acute toxicity appear within few hours post exposure to AntiChE substances. The classic muscarinic effects comprise sweating, lacrimation, salivation, diarrhea, urination, gastrointestinal discomfort, pin-point pupils, emesis, rhinorrhea, increased bronchial secretion, bronchoconstriction, and dyspnea. The nicotinic effects include

muscle fasciculations, twitching, weakness and/or paralysis in severe cases. Central nervous system signs include restlessness, tremor, confusion, convulsions, respiratory depression, and coma **(Rubinshtein et al., 2002).**

Other numerous complications such as disturbances of acid-base equilibrium, electrolyte values, and blood glucose levels have been documented in several cases poisoned with AntiChE family **(Kara et al., 2002; Liu et al., 2008; Gündüz et al., 2015).**

Cardiovascular toxicity often accompanies poisoning by AntiChE compounds, which may be serious or fatal. Several studies have recorded different patterns of cardiotoxic manifestations and electrocardiographic abnormalities in patients with AntiChE poisoning including disturbances in vital functions and conduction defects. These cardiac disorders commonly occur within few hours after exposure and many physicians may not fully recognize them. Hence, early detection and adequate treatment can potentially prevent this life-threatening condition and improve outcome **(Kara et al., 2002; Karki et al., 2004).**

Acute pancreatitis is also recognized as an important complication of intoxication by AntiChE agents and has been reported in experimental and humans studies **(Singh et al., 2007; Aslan et al., 2011).** However, the true incidence of pancreatic injury is unknown and may be more frequent than clinically suspected. Additionally, patients with AntiChE poisonings may present with subclinical traits of acute pancreatitis. Thus, proper clinical and biochemical investigations can help in diagnosis **(Sahin et al., 2002; Brahmi et al., 2005).**

Although liver has an important role in degradation of AntiChE substances, the effects of these compounds on the liver functions are not well understood. Acute hepatotoxicity as a complication of exposure to these agents has been frequently reported in several studies **(Sahin et al., 2002; Singh et al., 2011).**

The association between AntiChE poisoning and subsequent risk of development of acute kidney injury has been mentioned in a few numbers of the articles. Misidentification or underestimation of AntiChE-related nephrotoxicity may cause severe adverse outcomes. In a population-based retrospective cohort study, the incidence of AntiChE-related nephrotoxicity has been higher than in the non-AntiChE poisoned cohort **(Lee et al., 2015).** Multiple organs distress syndrome, impairment of renal function, and acute renal failure have been reported in human following acute exposure to AntiChE substances that widely correlated with death **(Agostini and Bianchin, 2003; Kozacı et al., 2012).** Also, exposures to different types of AntiChE substances have been associated with nephrotoxicity in occupational workers **(Singh et al., 2011).**

AIM OF THE WORK

The present study was designed to evaluate the correlation between poisoning severity score grades, Glasgow coma scale scores, and butyrylcholinesterase levels with clinical and laboratory findings among acute AntiChE intoxicated patients who admitted to Benha Poison Control Unite. Also, the validity of these parameters in predicting the prognosis and outcome of acute AntiChE poisoning were assessed.

SUBJECTS & METHODS

This prospective study was carried out over a period of 12-month from the 1st of March 2016 to the end of February 2017 at BPCU, Benha University Hospitals, El-Qalyubia, Egypt, after approval from the Research Ethical Committee of Benha Faculty of Medicine. Prior to starting the study, all proposed procedures were explained to the patients or relatives and all participants provided informed verbal consent. All selected patients with only acute AntiChE intoxication that fulfilled the inclusion criteria were enrolled in the present study.

A. Inclusion criteria:

1- History of acute AntiChE ingestion only (OPC and CMC).

2- Characteristic cholinergic toxidrome of cholinesterase inhibitors such as excessive salivation, lacrimation, and sweating, pinpoint pupils, chest wheezes and crepitations or rales, bradycardia, stool and urine incontinence, abdominal pain, nausea, and vomiting, muscular fasciculations or weakness, tachycardia, and manifestations of central nervous system stimulation or depression.

3- Improvement of the cholinergic toxidrome after administration of atropine and oximes.

4- Low BuChE level or activity.

5- Survived patients.

6- Patients or their relatives who gave informed consent.

B. Exclusion criteria:

1- All patients with co-ingestions.

2- All patients who received anticholinergic therapy before commencement of the study.

3- All patients with past medical history of special habits (smoking and intake of drugs, alcohol, or addiction), chronic exposure to anticholinesterase compounds, concomitant chronic systemic diseases (hepatitis, diabetes mellitus, hypertension, psychiatric, epilepsy, immunological, and neuromuscular), and/or any organ dysfunction.

4- All asymptomatic patients with normal BuChE levels and deceased cases.

5- Non-cooperative patients who refused to participate in the study.

Initially, all patients were rapidly stabilized, evaluated clinically, blood sampled, and managed on admission according to a standard clinical protocol of **Eddleston et al. (2004).**

Subjects:

All participated individuals were divided as follows:

(I) Healthy control group (HC-group): The individuals who kindly accepted to participate in this study were recruited randomly from the surrounding community with matched age and gender and matched as closely as possible with the intoxicated cases. They were non-smokers and free of any acute or chronic diseases with normal endocrine, cardio-pulmonary, and hepato-renal systems on clinical examination as well as they had not exposed to cholinesterase inhibitors and did not receive any drug during the last 2 weeks.

(II) Anticholinesterase group (AntiChE-group): Included the total number of cases with both OPC and CMC poisonings. Moreover, all patients were allocated into three intoxicated-groups according to their severity grades of poisoning namely mild, moderate, or severe.

Methods:

All suggested procedures were accomplished after stabilization of any life-threatening conditions and before initiation of therapy.

I- The epidemiological data:

The gender and age of all participants were recorded. Additionally, the subtypes of AntiChE causative compounds were reported.

II- The clinical data:

Clinical data in terms of the following parameters were carried out for all selected patients:

1- Delay in hospital arrival (DHA) time: time lapse or lags between exposures to AntiChE substances and the time of arrival to the unit.

2- Length of hospital stay (LHS) or the hospitalization period.

3- Vital signs: Each of heart rate (HR), blood pressure (BP), respiratory rate (RR), and body temperature (BT) was measured and documented. The abnormalities of vital functions in adults included bradycardia (HR <60 beats/minute), tachycardia (HR >100 beats/minute), hypotension (systolic blood pressure; SBP <90 mmHg and/or diastolic blood pressure; DBP <60 mmHg), hypertension (SBP >160 mmHg and/or DBP >95 mmHg), bradypnea (RR <12 breaths/minute), tachypnea (RR >25 breaths/minute), hypothermia (BT <36.8 ^{o}C), and hyperthermia (BT >37.2 ^{o}C) **(Nelson et al., 2011; Vijayakumar et al., 2011).**

4- Severity of poisoning: The clinical severity of each enrolled case was evaluated according to PSS criteria of the European Association of Poisons Centres and Clinical Toxicologists (EAPCCT) **(Persson et al., 1998).** According to severity grades, the selected patients were classified into 3 groups as follows:

A. Mild (grade 1): Cases with minor, transient, and spontaneously resolving symptoms within 24 hours of exposure.

B. Moderate (grade 2): Cases with pronounced more prolonged symptoms for 24 hours or more after exposure.

C. Severe (grade 3): Cases with life-threatening manifestations.

5- Level of consciousness: The conscious states of all patients were assessed against the criteria of GCS. The scale is composed of three tests: eye (score: 1-4), verbal (score: 1-5), and motor (score: 1-6) responses. The resulting sum of the three points gives each patient a score in the range of 3-15. The patients' neurologic status were scored as fully awake (15), drowsy (12-14), and stupor or coma (3-11) **(Baydin et al., 2007).**

III- Laboratory investigations:

During clinical examination, two venous and two arterial blood samples (5 ml each) were withdrawn from each selected patient, one at admission and the other at discharge. While, in the HC-group, a single venous and single arterial blood samples were taken from each individual. Venous blood samples were withdrawn directly into vacutainers containing coagulants, incubated at 37˚C for 15 minutes until blood clotted, and then centrifuged for 15 minutes at 3000 revolutions per minute to separate the sera. The clear non-haemolysed supernatant sera were quickly removed, collected in tubes, and immediately stored at -20°C till been investigated for the proposed biochemical parameters. Whereas, arterial blood samples were collected directly into vacutainers containing heparin anticoagulant and sent to the laboratory as soon as possible for pH analysis. The biochemical levels of pH of acid-base balance, sodium, potassium, and random glucose as well as the enzymatic biomarkers values of BuChE and cardiac (creatine kinase-myocardial band and cardiac troponin I), pancreatic (amylase and lipase), hepatic (aspartate aminotransferase and

alanine transaminase), and kidney (blood urea and creatinine) organs were estimated as follows:

A. Arterial blood pH level was measured spectrophotometrically according to the method of **Scott et al. (2006)** using EliTech UK IRMA TRUpoint blood gas analyzer (reference value: 7.35-7.45).

B. Serum sodium (Na) and potassium (K) levels were estimated according to the atomic absorption flame photometric method of **Pincus and Lifshitz (2007)** using the NA-p CAL and K-p CAL reagent kits, respectively, obtained from Spinreact Company (Spain) (reference value of Na: 135-145 mEq/L; reference value of K: 3.5-5.5 mEq/L).

C. Serum random blood glucose (BLG) level was assayed spectrophotometrically according to the method of **Kunsst (1994)** using hexokinase enzymatic reagent kits purchased from Spinreact Company (Spain) (reference value: 70-140 mg/dl).

D. Serum BuChE enzyme activity was evaluated spectrophotometrically according to the method of **Whittaker et al. (1983)** using cholinesterase reagent kits obtained from Spinreact Company (Spain) (reference value: 4850-12.000 U/L).

E. Serum creatine kinase-myocardial band (CK-MB) enzyme activity was quantitatively measured spectrophotometrically according to the procedure of **Gerhardt and Waldenström (1979)** using anti CK-M immune-inhibition reagent kits obtained from Spinreact Company (Spain) (reference value: <24 U/L).

F. Cardiac troponin I (cTnI) enzyme activity was quantitatively measured according to the method of **Adams et al. (1994)** using Accu-Bind cTnI Microplate Enzyme Linked Immunosorbent Assay (ELISA) commercial kits provided from Monobind Inc, Lack Forest (USA) (reference value: ≤1.3 ng/ml).

G. Serum amylase enzyme activity was estimated according to the amylase-colorimetric method of **Foo and Bais (1998)** using the chloro-nitrophenol maltotriose glucose kinetic reagent kits supplied from Spinreact Company (Spain) (reference value: up to 90 U/L).

H. Serum lipase enzyme activity was determined according to the lipase-colorimetric kinetic method of **Lorentz (1998)** using the lipase-LS dilauryl-rac-glycero-3-glutaric acid-(6-methylresorufin)-ester reagent kits supplied from Spectrum Diagnostics - The Egyptian Company for Biotechnology (S.A.E) (reference value: up to 60 U/L).

I. Serum aspartate aminotransferase (AST) and alanine transaminase (ALT) hepatic enzymes activities were measured spectrophotometrically according to the method of **Pincus et al. (2007)** using the AST and ALT NADH kinetic UV IFCC reagent kits, respectively, provided from Spinreact Company (Spain) (reference value of AST: 38 U/L; reference value of ALT: 40 U/L).

J. Serum blood urea (BLU) and creatinine (CRE) renal enzymes activities were estimated, respectively, according to urease- and creatinine-colorimetric methods of **Lamb and Price (2008)** using the urease-glutamate dehydrogenase and Jaffe's sodium picrate reagent kits, respectively, provided from Spinreact Company (Spain) (reference value of BLU: 15-45 mg/dL; reference value of CRE: 0.7-1.4 mg/dL in males and 0.6-1.1 mg/dL in females).

Statistical Methods

The collected data were defined, coded, and analyzed using the statistical package of social science software version 16 (SPSS Inc, Chicago, IL Company). Categorical data were presented as number and percentages while quantitative data were expressed as mean ± standard error of measurement (SEM). Chi square test (X^2), one-way analysis of variance (ANOVA), and Pearson's correlation coefficient were used as tests of significance. Chi square (X^2) was used as test for comparison between number and percentage of two or more groups to compare frequencies. ANOVA was used to compare between more than two continuous variables and significant results followed by Tukey HSD post-hoc multiple comparisons test to detect which pairs were significant. Pearson's correlation coefficient was used to evaluate the linear association between two quantitative variables, where positive and negative results indicating direct and inverse relationship, respectively. A P-value of <0.05 ($P<0.05$) was considered as significant level.

RESULTS

During this one year prospective study, 75 patients of both sexes with AntiChE poisoning clearly fulfilled the inclusion criteria and accepted study participation.

As shown in Table (1), HC-group included 25 individuals (9 males and 16 females with a sex ratio of 1:1.77) and the AntiChE-group comprised 75 patients (30 males and 45 females with a sex ratio of 1:1.5). There was more prevalence of acute poisoning among female than male and the mean age of the overall patients was 23.82±0.82 years. Statistical analysis showed non-significant differences between HC-group, AntiChE-group, and Intoxicated-groups regarding the proportions of gender and age variables.

As noted in Table (2), poisoning by OPC was more common than by CMC with malathion and aldicarb were the most frequently utilized subtypes among both substances, respectively.

As illustrated in Table (3), the main bulk of patients in AntiChE-group experienced mild toxic manifestations (grade 1) of PSS, followed in order of frequency by those with moderate (grade 2) and severe (grade 3) toxicities, DHA for 2-hour followed by those with ≥ 5-hour post exposure, and relatively short LHS for ≤24-hour then for 48-hour.

The vast majority of cases in AntiChE-group showed abnormalities in their HR, BP, RR, and conscious status, while unusual BT were less frequently observed.

The most common findings of clinical abnormalities were tachycardia, hypertension, tachypnea, hyperthermia, and drowsy conscious score of GCS. Chi-square statistical analysis between different proportions of the overall clinical parameters showed significant differences among the intoxicated-groups.

As observed in Table (4), the mean values of PSS, DHA, GCS, and LHS in AntiChE-group were 1.69±0.09, 3.5±0.32 hours, 13.1±0.25, and 45.65±4.03 hours, respectively. Among the Intoxicated-groups, patients with moderate and severe grades of poisoning depicted statistically significant increases in the mean values of DHA and LHS, while their mean values of GCS exhibited statistically significant decreases when compared to the corresponding values of cases with

mild toxicity grade. Also, both mean values of DHA and LHS were statistically significantly higher and that of GCS was statistically significantly lower in severe than moderate cases.

Moreover, the mean values of HR, diastolic BP (DBP), systolic BP (SBP), RR, and BT in AntiChE-group were 87.32±3.77 beats/minute, 74.91± 2.57 mmHg, 118.69±3.83 mmHg, 21.37±0.84 breaths/minute, and 37.2±0.08 ^{o}C, respectively. The mean values of all vital signs were statistically significantly increased and decreased in moderate and severe poisonings, respectively, as compared to the corresponding values of mild toxicity grade. Also, the differences in vital signs were statistically significantly reduced in severe than moderate poisonings.

As recognized in Table (5), statistical analysis revealed significant elevations in the total mean values of HR and RR and significant reduction in the total mean value of GCS score of AntiChE-group on admission when matched with those of HC-group. Also, statistically significant differences in the mean values of all disturbed vital signs and conscious status were observed between AntiChE-group on admission and HC-group.

On the other hand, these initial changes in the mean values of the total and abnormal clinical findings showed marked improvement at discharge as evidenced by statistically insignificant and significant distinctions from the mean values of HC-group and AntiChE-group at admission, respectively.

As detected in Table (6), the r-values of DHA and LHS showed statistically significant strong positive correlation with PSS grades, however, significant strong negative correlation were observed between PSS grades and the total and slow HR, total, low, and high DBP, low and high SBP, low BT, GCS score, and BuChE enzyme. Moreover, there were significant moderate negative correlation between PSS grades and the other statistically analyzed variables.

The r-values of DHA and LHS showed statistically significant strong negative association with GCS scores; nonetheless, significant strong positive association were noticed between GCS scores and slow and rapid HR, low DBP, low SBP, slow and rapid RR, low BT, and BuChE enzyme. Additionally, there were significant moderate positive association between GCS scores and the other statistically analyzed variables.

The r-values of DHA and LHS depicted statistically significant strong negative relationship with BuChE enzyme, additionally; there were statistically significant strong positive relationship between BuChE enzyme and total HR and total and slow RR. Furthermore, there were significant moderate positive relationship between BuChE enzyme and the other statistically analyzed variables.

As manifested in Table (7), the main reported abnormal biochemical findings in AntiChE-group were low pH (30; 40%), hypernatremia (21; 28%), hypokalemia (42; 56%), and hyperglycaemia (43; 57.33%), whereas the chief recorded abnormal laboratory findings of enzymatic biomarkers were low BuChE (75; 100% and the activity was decreased by 50% in 32; 42.67% cases) and high serum levels of CK-MB (28; 49.12%), cTnI (18; 24%), amylase (25; 33.33%), lipase (20; 26.67%), AST (41; 54.67%), ALT (33; 44%), BLU (33; 44%), and CRE (29; 38.67%). Chi-square statistical analysis between

different proportions of the overall laboratory parameters showed significant differences among the intoxicated-groups.

As delineated in Table (8), in moderate and severe intoxicated-groups on admission, the mean values of pH, K, and BuChE enzyme were statistically significantly decreased, while those of Na, BLG, CK-MB, cTnI, amylase, lipase, AST, ALT, BLU, and CRE were statistically significantly increased when compared with the corresponding values of mild intoxicated-group.

As evidenced in table (9), in AntiChE-group on admission, statistical analysis depicted significant decreases in the mean values of pH, K, and BuChE enzyme, whereas those of Na, BLG, CK-MB, cTnI, amylase, lipase, AST, ALT, BLU, and CRE exhibited significant increases when compared with the corresponding values of HC-group.

Otherwise, these initial alterations in the mean values of almost all investigated biochemical and enzymatic biomarkers were remarkably returned back to normal levels at discharge as evidenced by statistically insignificant and significant disparities from the mean values of HC-group and AntiChE-group at admission, respectively. However, comparison between the mean values of BuChE enzyme of AntiChE-group at discharge and at admission revealed statistically significant increase, but still significantly lowers than HC-group value.

As demonstrated in Table (10), the r-values of all investigated biochemical and enzymatic biomarkers of AntiChE-group were statistically significantly correlated with PSS grades. There was a statistically significant strong negative correlation between pH and PSS scores, whereas the r-values of Na, K, BLG, CK-MB, cTnI, AST, and ALT showed statistically significant strong positive correlations with PSS scores. In addition, the r-values of amylase, lipase, BLU, and CRE showed statistically significant moderate positive correlations with PSS scores.

The r-values of all estimated biochemical and enzymatic biomarkers of AntiChE-group were statistically significantly associated with GCS scores, wherein the r-values of pH and K showed statistically significant moderate positive associations, those r-values of Na, BLG, CK-MB, cTnI, AST, ALT, BLU, and CRE depicted statistically significant moderate negative associations, and those r-values of amylase and lipase showed statistically significant weak negative associations.

The r-values of all assayed biochemical and enzymatic biomarkers of AntiChE-group were statistically significantly associated with BuChE enzyme, wherein the r-values of CK-MB and cTnI revealed statistically significant strong negative relationships, those r-values of pH and K showed statistically significant moderate positive relationships, and those r-values of Na, BLG, amylase, lipase AST, ALT, BLU, and CRE depicted statistically significant moderate negative relationships.

Table (1): Distribution pattern and statistical differences between genders and age among HC-group, AntiChE-group, and Intoxicated-groups.

Variables	HC-group	AntiChE	Intoxicated-groups		
			Mild	Moderate	Severe
Gender					
Male[@]	9 (36)	30 (40)	17 (22.67)	8 (10.67)	5 (6.67)
Female[@]	16 (64)	45 (60)	21 (28.00)	14 (18.67)	10 (13.33)
Sex ratio	1:1.77	1:1.5	1:1.24	1:1.75	1:2
X^2 score	0.902				
P-value	0.924^{NS}				
Age (Year)					
Mean ± SEM	25.16±1.37	23.82±0.82	22.73±1.16	24.15±1.52	26.1±1.75
F-statistic[#]	0.8291				
P-value	0.5082^{NS}				

HC=Healthy control; AntiChE=Anticholinesterase; @=Number and percentage; X^2=Chi-square; #=F-test result of one-way analysis of variance (ANOVA); P=Probability; NS=Non-significant difference. P-value of >0.05 was considered non-significant.

Table (2): Distribution pattern of the AntiChE causative compounds according to their different subtypes.

Compounds	Subtypes	Number (%)	Total (%)	X^2 score	P-value
OPC	Malathion (Carbophos)	32 (42.67)	53 (70.67)	2.38	0.497^{NS}
	Dimethoate (Rogor)	9 (12.00)			
	Diazinon (Basudin)	7 (9.33)			
	Chloropyrifos (Lorsban)	5 (6.67)			
CMC	Aldicarb (Temik)	14 (18.67)	22 (29.33)		
	Methomyl (Lannate)	5 (6.67)			
	Carbofuran (Furadan)	3 (4.00)			

AntiChE=Anticholinesterase; OPC=Organophosphate compounds; CMC=Carbamate compounds; %=Percentage; X^2=Chi-square; P=Probability; NS=Non-significant difference. P-value of >0.05 was considered non-significant.

Table (3): Distribution pattern and chi-square statistical differences between the three Intoxicated-groups concerning the PSS, DHA, vital signs, GCS, and LHS at the time of admission.

Variables		Number (Percentage)				X^2 score	P-value
		AntiChE	Intoxicated-groups				
			Mild	Moderate	Severe		
PSS		75 (100)	38 (50.67)	22 (29.33)	15 (20.00)	-----	-----
DHA	≤ 2-hour	38 (50.67)	30 (40.00)	6 (8.00)	2 (2.67)	26.63	0.0002*
	3-hour	10 (13.33)	2 (2.67)	5 (6.67)	3 (4.00)		
	4-hour	10 (13.33)	3 (4.00)	3 (4.00)	4 (5.33)		
	≥ 5-hour	17 (22.67)	3 (4.00)	8 (10.67)	6 (8.00)		
HR	Normal	20 (26.67)	14 (18.67)	4 (5.33)	2 (2.67)	4.19	0.0408*
	Abnormal	55 (73.33)	24 (32.00)	18 (24.00)	13 (17.33)		
	Bradycardia	23 (30.67)	11 (14.67)	2 (2.67)	10 (13.33)	13.72	0.001*
	Tachycardia	32 (42.67)	13 (17.33)	16 (21.33)	3 (4.00)		
BP	Normal	30 (40.00)	25 (33.33)	4 (5.33)	1 (1.33)	21.84	0.0001*
	Abnormal	45 (60.00)	13 (17.33)	18 (24.00)	14 (18.67)		
	Hypotension	18 (24.00)	5 (6.67)	1 (1.33)	12 (16.00)	21.1	0.0001*
	Hypertension	27 (36.00)	8 (10.67)	17 (22.67)	2 (2.67)		
RR	Normal	36 (48.00)	31 (41.33)	2 (2.67)	3 (4.00)	35.22	0.0001*
	Abnormal	39 (52.00)	7 (9.33)	20 (26.67)	12 (16.00)		
	Bradypnea	13 (17.33)	0 (0.00)	3 (4.00)	10 (13.33)	20.03	0.0001*
	Tachypnea	26 (34.67)	7 (9.33)	17 (22.67)	2 (2.67)		
BT	Normal	45 (60.00)	33 (44.00)	5 (6.67)	7 (9.33)	25.25	0.0001*
	Abnormal	30 (40.00)	5 (6.67)	17 (22.67)	8 (10.67)		
	Hypothermia	9 (12.00)	2 (2.67)	1 (1.33)	6 (8.00)	12.66	0.0018*
	Hyperthermia	21 (28.00)	3 (4.00)	16 (21.33)	2 (2.67)		
GCS	Full awake (15)	35 (46.67)	28 (37.33)	7 (9.33)	0 (0.00)	26.22	0.0001*
	AMS	40 (53.33)	10 (13.33)	15 (20.00)	15 (20.00)		
	Drowsy	23 (30.67)	10 (13.33)	9 (12.00)	4 (5.33)	13.27	0.0013*
	Coma	17 (22.67)	0 (0.00)	6 (8.00)	11 (14.67)		
LHS	≤24-hour	30 (40.00)	30 (40.00)	0 (0.00)	0 (0.00)	116.5	0.0001*
	48-hour	20 (26.67)	8 (10.67)	12 (16.00)	0 (0.00)		
	72-hour	11 (14.67)	0 (0.00)	10 (13.33)	1 (1.33)		
	>72-hour	14 (18.67)	0 (0.00)	0 (0.00)	14 (18.67)		

AntiChE=Anticholinesterase; PSS=Poisoning severity score; DHA=Delay in hospital arrival (hours); HR=Heart rate (beats/minute); DBP=Diastolic blood pressure (mmHg); SBP=Systolic blood pressure (mmHg); RR=Respiratory rate (breaths/minute); BT=Body temperature (ºC); GCS=Glasgow Coma Scale; AMS=Altered mental status; LHS=Length of hospital stay (hours); X^2=Chi-square; P=Probability; *=Significant difference. P-value of <0.05 was considered significant

Table (4): One-way analysis of variance (ANOVA) statistical differences between the Intoxicated-groups concerning the clinical findings at the time of admission.

Variables		AntiChE-group	Intoxicated-groups			
			Mild	**Moderate**	**Severe**	**Severe vs Moderate**
PSS grade	Mean ± SEM	1.69±0.09	1±0.00	2±0.00	3±0.00	-----
	P-value	-----	-----	0.0001* (↑)	0.0001* (↑)	0.0001* (↑)
DHA	Mean ± SEM	3.5±0.32	1.71±0.13	4.14±0.33	7.1±0.91	-----
	P-value	-----	-----	0.001* (↑)	0.0001* (↑)	0.002* (↑)
HR	Mean ± SEM	87.32±3.77	82±3.38	116.6±6.9	57.87±6.1	-----
	P-value	-----	-----	0.0001* (↑)	0.007* (↓)	0.0001* (↓)
DBP	Mean ± SEM	74.91± 2.57	71.53±2.7	94.1±3.94	55.33±4.79	-----
	P-value	-----	-----	0.0001* (↑)	0.010* (↓)	0.0001* (↓)
SBP	Mean ± SEM	118.69±3.83	116.76±3.12	147.95±5.98	80.67±7.02	-----
	P-value	-----	-----	0.0001* (↑)	0.0001* (↓)	0.0001* (↓)
RR	Mean ± SEM	23.41±1.1	23.71±0.82	30.32±2.1	12.53±1.46	-----
	P-value	-----	-----	0.002* (↑)	0.0001* (↓)	0.0001* (↓)
BT	Mean ± SEM	37.2±0.08	37.06±0.3	37.86±0.16	36.55±0.15	-----
	P-value	-----	-----	0.0001* (↑)	0.003* (↓)	0.0001* (↓)
GCS	Mean ± SEM	13.1±0.25	14.55±0.14	12.68±0.38	9.93±0.34	-----
	P-value	-----	-----	0.0001* (↓)	0.0001* (↓)	0.0001* (↓)
LHS	Mean ± SEM	46.97±4.1	20.76±1.16	51.68±3.33	106.5±5.62	-----
	P-value	-----	-----	0.0001* (↑)	0.0001* (↑)	0.0001* (↑)

AntiChE=Anticholinesterase; PSS=Poisoning severity score; DHA=Delay in hospital arrival (hours); HR=Heart rate (beats/minute); DBP=Diastolic blood pressure (mmHg); SBP=Systolic blood pressure (mmHg); RR=Respiratory rate (breaths/minute); BT=Body temperature (ºC); GCS=Glasgow Coma Scale; LHS=Length of hospital stay (hours); SEM=Standard error of measurement; P=Probability; *=Significant difference; ↑=Increase; ↓=Decrease. P-value of <0.05 was considered significant.

Table (5): One-way analysis of variance (ANOVA) statistical comparison between HC-group and AntiChE-group at admission and discharge regarding to the mean values of the total and abnormal clinical findings.

Variables		HC-group	AntiChE-group		
			Admission	Discharge	Discharge vs Admission
PSS grades	Mean ± SEM	0±0.00	1.69±0.09	0±0.00	-----
	P-value	-----	0.0001*	-----	0.0001* (↓)
Total HR	Mean ± SEM	72.24±0.85	87.32±3.77	73.12±0.65	-----
	P-value	-----	0.009* (↑)	0.89NS	0.0001* (↓)
Slow HR	Mean ± SEM	-----	49.13±1.1	70.22±1	-----
	P-value	-----	0.0001* (↓)	0.33NS	0.0001* (↑)
Rapid HR	Mean ± SEM	-----	119±3.21	75.5±0.46	-----
	P-value	-----	0.0001* (↑)	0.60NS	0.0001* (↓)
Total DBP	Mean ± SEM	75.88± 0.87	74.91± 2.57	70.24±0.58	-----
	P-value	-----	0. 0.89NS	0. 0.31NS	0. 0.18NS
Low DBP	Mean ± SEM	-----	50.17±1.1	73.16±1.1	-----
	P-value	-----	0.0001* (↓)	0.10NS	0.0001* (↑)
High DBP	Mean ± SEM	-----	102.78±0.82	74.11±0.83	-----
	P-value	-----	0.0001* (↑)	0.33NS	0.0001* (↓)
Total SBP	Mean ± SEM	115.2±0.87	118.69±3.83	108.97±0.72	-----
	P-value	-----	0.88NS	0.56NS	0.024* (↓)
Low SBP	Mean ± SEM	-----	73.78±2.26	110.89±1.39	-----
	P-value	-----	0.0001* (↓)	0.072NS	0.0001* (↑)
High SBP	Mean ± SEM	-----	156.44±1.77	112.93±1.03	
	P-value	-----	0.0001* (↑)	0.49NS	0.0001* (↓)
Total RR	Mean ± SEM	19.44±0.46	23.41±1.1	19.84±0.25	-----
	P-value	-----	0.017* (↑)	0.958NS	0.002* (↓)
Slow RR	Mean ± SEM	-----	10.54±0.18	19.31±0.35	-----
	P-value	-----	0.0001* (↓)	0.89NS	0.0001* (↑)
Rapid RR	Mean ± SEM	-----	29.77±0.52	18.04±0.33	-----
	P-value	-----	0.0001* (↑)	0.055NS	0.0001* (↓)
Total BT	Mean ± SEM	37.07±0.01	37.2±0.08	37.04±0.01	-----
	P-value	-----	0.57NS	0.89NS	0.10NS
Low BT	Mean ± SEM	-----	36.17±0.13	37.1±0.03	-----
	P-value	-----	0.0001* (↓)	0.90NS	0.0001* (↑)
High BT	Mean ± SEM	-----	38±0.14	37.08±0.02	
	P-value	-----	0.0001* (↑)	0.90NS	0.0001* (↓)
Total GCS	Mean ± SEM	15±0.00	13.08±0.25	14.63±0.06	-----
	P-value	-----	0.0001* (↓)	0.63NS	0.0001* (↑)
Altered GCS	Mean ± SEM	-----	11.4±0.27	14.38±0.08	-----
	P-value	-----	0.0001* (↓)	0.065NS	0.0001* (↑)

HC=Healthy control; AntiChE=Anticholinesterase; PSS=Poisoning severity score; HR=Heart rate (beats/minute); DBP=Diastolic blood pressure (mmHg); SBP=Systolic blood pressure (mmHg); RR=Respiratory rate (breaths/minute); BT=Body temperature (ºC); GCS=Glasgow Coma Scale; SEM=Standard error of measurement; P=Probability; NS=Non-significant difference; *=Significant difference; ↑=Increase; ↓=Decrease. P-value of >0.05 was considered non-significant; P-value of <0.05 was considered significant.

Table (6): Pearson's correlation analysis between PSS, GCS, and BuChE with the clinical findings of AntiChE-group at the time of admission.

Variables	PSS		GCS		BuChE	
	r-value	P-value	r-value	P-value	r-value	P-value
DHA	0.8595$	0.0001*	-0.8839$	0.0001*	-0.8037$	0.0001*
Total HR	-0.7729$	0.0001*	0.5311&	0.0001*	0.7848$	0.0001*
Slow HR	-0.8902$	0.0001*	0.9482$	0.0001*	0.6081&	0.0035*
Rapid HR	-0.5771&	0.0007*	0.7887$	0.0001*	0.5397&	0.0018*
Total DBP	-0.8385$	0.0001*	0.6666&	0.0001*	0.6274&	0.0001*
Low DBP	-0.8511$	0.0001*	0.8254$	0.0001*	0.527&	0.0246*
High DBP	-0.7915$	0.0001*	0.5692&	0.0019*	0.6277&	0.0005*
Total SBP	-0.7227&	0.0001*	0.5703&	0.0001*	0.6396&	0.0001*
Low SBP	-0.7651$	0.0002*	0.843$	0.0001*	0.664&	0.0027*
High SBP	-0.7693$	0.0001*	0.5417&	0.0035*	0.5944&	0.0011*
Total RR	-0.5584&	0.0001*	0.5302&	0.0001*	0.7611$	0.0001*
Slow RR	-0.5752&	0.0397*	0.8564$	0.0001*	0.7793$	0.0017*
Rapid RR	-0.6825&	0.0001*	0.7862$	0.0001*	0.7459&	0.0001*
Total BT	-0.7035&	0.0001*	0.7239&	0.0001*	0.6083&	0.0001*
Low BT	-0.7501$	0.0199*	0.8355$	0.005*	0.6841&	0.0421*
High BT	-0.6592&	0.0012*	0.7231&	0.0002*	0.5132&	0.0173*
PSS	-----	-----	-0.8094$	0.0001*	-0.9173$	0.0001*
GCS	-----	-----	-----	-----	0.8883$	0.0001*
LHS	0.912$	0.0001*	-0.7751$	0.0001*	-0.9433$	0.0001*

PSS=Poisoning severity score; GCS=Glasgow Coma Scale; BuChE=Butyrylcholinesterase; AntiChE=Anticholinesterase; DHA=Delay in hospital arrival (hours); HR=Heart rate (beats/minute); DBP=Diastolic blood pressure (mmHg); SBP=Systolic blood pressure (mmHg); RR=Respiratory rate (breaths/minute); BT=Body temperature (ºC); LHS=Length of hospital stay (hours); *=Significant difference. P-value of <0.05 was considered significant. $=Strong correlation; &=Moderate correlation.

Table (7): The distribution patterns and chi-square statistical differences between the intoxicated study groups concerning the normal and abnormal laboratory findings of the investigated biochemical and enzymatic biomarkers at the time of admission.

Variables		Number (Percentage) Groups				X^2 score	P-value
		AntiChE	Mild	Moderate	Severe		
pH	Normal	45 (60)	33 (44)	6 (8)	6 (8)	23.73	0.0001*
	Decreased	30 (40)	5 (6.67)	16 (21.33)	9 (12)		
Na	Normal	54 (72)	36 (48)	14 (18.67)	4 (5.33)	25.8	0.0001*
	Increased	21 (28)	2 (2.67)	8 (10.67)	11 (14.67)		
K	Normal	33 (44)	30 (40)	2 (2.67)	1 (1.33)	38.2	0.0001*
	Decreased	42 (56)	8 (10.67)	20 (26.67)	14 (18.67)		
BLG	Normal	25 (33.33)	22 (29.33)	2 (2.67)	1 (1.33)	23.1	0.0001*
	Increased	43 (57.33)	12 (16.00)	18 (24.00)	13 (17.33)		
	Decreased	7 (9.33)	4 (5.33)	2 (2.67)	1 (1.33)		
BuChE	50%	32 (42.67)	32 (42.67)	0 (0.00)	0 (0.00)	111.36	0.0001*
	20-50%	24 (32.00)	6 (8.00)	18 (24.00)	0 (0.00)		
	10-20%	7 (9.33)	0 (0.00)	4 (5.33)	3 (4.00)		
	<10%	12 (16.00)	0 (0.00)	0 (0.00)	12 (16.00)		
CK-MB	Normal	47 (82.46)	38 (66.67)	7 (12.28)	2 (3.51)	47.19	0.0001*
	Increased	28 (49.12)	0 (0.00)	15 (26.32)	13 (22.81)		
cTnI	Normal	57 (76.00)	38 (50.67)	14 (18.67)	5 (6.67)	28.81	0.0001*
	Increased	18 (24.00)	0 (0.00)	8 (10.67)	10 (13.33)		
Amylase	Normal	50 (66.67)	33 (44.00)	11 (14.67)	6 (8.00)	14.51	0.0007*
	Increased	25 (33.33)	5 (6.67)	11 (14.67)	9 (12.00)		
Lipase	Normal	55 (73.33)	36 (48.00)	12 (16.00)	7 (9.33)	18.33	0.0001*
	Increased	20 (26.67)	2 (2.67)	10 (13.33)	8 (10.67)		
AST	Normal	34 (45.33)	32 (42.67)	1 (1.33)	1 (1.33)	46.99	0.0001*
	Increased	41 (54.67)	6 (8.00)	21 (28.00)	14 (18.67)		
ALT	Normal	42 (56.00)	35 (46.67)	4 (5.33)	3 (4.00)	40.76	0.0001*
	Increased	33 (44.00)	3 (4.00)	18 (24.00)	12 (16.00)		
BLU	Normal	42 (56.00)	35 (46.67)	5 (6.67)	2 (2.67)	41.1	0.0001*
	Increased	33 (44.00)	3 (4.00)	17 (22.67)	13 (17.33)		
CRE	Normal	46 (61.33)	34 (45.33)	8 (10.67)	4 (5.33)	26.1	0.0001*
	Increased	29 (38.67)	4 (5.33)	14 (18.67)	11 (14.67)		

AntiChE=Anticholinesterase; Na=Sodium; K=Potassium; BLG=Blood glucose; BuChE=Butyrylcholinesterase; CK-MB=Creatine kinase-myocardial band; cTnI=Cardiac troponin I; AST=Aspartate aminotransferase; ALT=Alanine transaminase; BLU=Blood urea; CRE=Creatinine; X^2=Chi-square; P=Probability; *=Significant difference. P-value of <0.05 was considered significant.

Table (8): One-way analysis of variance (ANOVA) statistical differences between the Intoxicated-groups concerning the mean values of the investigated biochemical and enzymatic biomarkers at the time of admission.

Variables		AntiChE-groups			
		Mild	**Moderate**	**Severe**	**Severe vs Moderate**
pH	Mean ± SEM	7.36±0.003	7.18±0.039	7.02±0.085	-----
	P-value	-----	0.0001* (↓)	0.0001* (↓)	0.025* (↓)
Na (mEq/L)	Mean ± SEM	137.16±0.49	144.64±1.64	155.1±2.89	-----
	P-value	-----	0.0001* (↑)	0.0001* (↑)	0.0001* (↑)
K (mEq/L)	Mean ± SEM	4.1±0.1	3.1±0.08	2.67±0.09	-----
	P-value	-----	0.0001* (↓)	0.0001* (↓)	0.044* (↓)
BLG (mg⁄dl)	Mean ± SEM	109.47±6.02	173.14±9.2	216.87±14.1	-----
	P-value	-----	0.0001* (↑)	0.0001* (↑)	0.009* (↑)
BuChE (U/L)	Mean ± SEM	3851.76±146.1	2145.77±139.5	779.2±43.6	-----
	P-value	-----	0.0001* (↓)	0.0001* (↓)	0.0001* (↓)
CK-MB (U/L)	Mean ± SEM	22.19±0.25	37.42±3.32	58.12±4.74	-----
	P-value	-----	0.0001* (↑)	0.0001* (↑)	0.0001* (↑)
cTnI (ng/ml)	Mean ± SEM	1.14±0.03	9.2±2.51	22.83±4.42	-----
	P-value	-----	0.009* (↑)	0.0001* (↑)	0.0001* (↑)
Amylase (U/L)	Mean ± SEM	56.87±4.5	126.77±10.89	189.73±24.71	-----
	P-value	-----	0.0001* (↑)	0.0001* (↑)	0.003* (↑)
Lipase (U/L)	Mean ± SEM	43.82±2.13	92.32±9.46	145.6±22.97	-----
	P-value	-----	0.0001* (↑)	0.0001* (↑)	0.003* (↑)
AST (U/L)	Mean ± SEM	30.71±1.65	103.27±7.17	161.4±20.29	-----
	P-value	-----	0.0001* (↑)	0.0001* (↑)	0.0001* (↑)
ALT (U/L)	Mean ± SEM	29.81±1.55	83.95±6.59	143.1±18.39	-----
	P-value	-----	0.0001* (↑)	0.0001* (↑)	0.0001* (↑)
BLU (mg⁄dl)	Mean ± SEM	34.29±1.44	64.88±3.32	81.46±4.88	-----
	P-value	-----	0.0001* (↑)	0.0001* (↑)	0.001* (↑)
CRE (mg⁄dl)	Mean ± SEM	1.3±0.02	1.69±0.07	2.55±0.21	-----
	P-value	-----	0.0001* (↑)	0.0001* (↑)	0.0001* (↑)

AntiChE=Anticholinesterase; Na=Sodium; K=Potassium; BLG=Blood glucose; BuChE=Butyrylcholinesterase; CK-MB=Creatine kinase-myocardial band; cTnI=Cardiac troponin I; AST=Aspartate aminotransferase; ALT=Alanine transaminase; BLU=Blood urea; CRE=Creatinine; SEM=Standard error of measurement; P=Probability; *=Significant difference; ↑=Increase; ↓=Decrease. P-value of <0.05 was considered significant.

Table (9): One-way analysis of variance (ANOVA) statistical comparison between HC-group and AntiChE-groups at admission and discharge regarding to the mean values of the investigated biochemical and enzymatic biomarkers.

Variables		HC-group	AntiChE-groups		
			Admission	Discharge	Discharge vs Admission
pH	Mean ± SEM	7.37±0.006	7.24±0.025	7.36±0.002	-----
	P-value	-----	0.0001* (↓)	0.944NS	0.0001* (↑)
Na (mEq/L)	Mean ± SEM	138.12±0.59	142.93±1.11	137.51±0.29	-----
	P-value	-----	0.005* (↑)	0.915NS	0.0001* (↓)
K (mEq/L)	Mean ± SEM	4.31±0.12	3.49±0.09	4.04±0.05	-----
	P-value	-----	0.0001* (↓)	0.15NS	0.0001* (↑)
BLG (mg⁄dl)	Mean ± SEM	113.36±3.77	149.63±7	107.19±1.98	-----
	P-value	-----	0.0001* (↑)	0.78NS	0.0001* (↓)
BuChE (U/L)	Mean ± SEM	7403.7±324.7	2736.8±165.4	3975.3±170.4	-----
	P-value	-----	0.0001* (↓)	0.0001* (↓)	0.0001* (↑)
CK-MB (U/L)	Mean ± SEM	19.31±0.48	33.84±2.1	21.32±0.28	-----
	P-value	-----	0.0001* (↑)	0.73NS	0.0001* (↓)
cTnI (ng/ml)	Mean ± SEM	1.11±0.04	7.84±1.48	1.22±0.03	-----
	P-value	-----	0.002* (↑)	0.89NS	0.0001* (↓)
Amylase (U/L)	Mean ± SEM	39.28±2.38	100.16±9	49.17±2	-----
	P-value	-----	0.0001* (↑)	0.68NS	0.0001* (↓)
Lipase (U/L)	Mean ± SEM	35.45±1.93	78.4±7.1	42.29±1.41	-----
	P-value	-----	0.0001* (↑)	0.77NS	0.0001* (↓)
AST (U/L)	Mean ± SEM	26.48±1.28	78.13±7.56	31.55±0.83	-----
	P-value	-----	0.0001* (↑)	0.85NS	0.0001* (↓)
ALT (U/L)	Mean ± SEM	25.6±1.35	68.35±6.58	30.29±0.61	-----
	P-value	-----	0.0001* (↑)	0.84NS	0.0001* (↓)
BLU (mg⁄dl)	Mean ± SEM	29.53±1.15	52.7±2.74	33.2±0.72	-----
	P-value	-----	0.0001* (↑)	0.58NS	0.0001* (↓)
CRE (mg⁄dl)	Mean ± SEM	1.11±0.05	1.66±0.07	1.27±0.01	-----
	P-value	-----	0.0001* (↑)	0.21NS	0.0001* (↓)

HC=Healthy control; AntiChE=Anticholinesterase; Na=Sodium; K=Potassium; BLG=Blood glucose; BuChE=Butyrylcholinesterase; CK-MB=Creatine kinase-myocardial band; cTnI=Cardiac troponin I; AST=Aspartate aminotransferase; ALT=Alanine transaminase; BLU=Blood urea; CRE=Creatinine; SEM=Standard error of measurement; P=Probability; NS=Non-significant difference; *=Significant difference; ↑=Increase; ↓=Decrease. P-value of >0.05 was considered non-significant; P-value of <0.05 was considered significant.

Table (10): Pearson's correlation analysis between PSS, GCS, and BuChE with laboratory findings of the investigated biochemical and enzymatic biomarkers of AntiChE-groups at the time of admission.

Parameters	PSS		GCS		BuChE	
	r-value	P-value	r-value	P-value	r-value	P-value
pH	-0.8438$	0.0001*	0.5706&	0.0001*	0.6316&	0.0001*
Na (mEq/L)	0.7874$	0.0001*	-0.5124&	0.0001*	-0.6197&	0.0001*
K (mEq/L)	-0.8533$	0.0001*	0.5314&	0.0001*	0.5354&	0.0001*
BLG (mg⁄dl)	0.9149$	0.0001*	-0.6521&	0.0001*	-0.5842&	0.0001*
CK-MB (U/L)	0.7897$	0.0001*	-0.5136&	0.0001*	-0.8264$	0.0001*
cTnI (ng/ml)	0.7931$	0.0001*	-0.5959&	0.0001*	-0.7991$	0.0001*
Amylase (U/L)	0.6583&	0.0001*	-0.4314@	0.0001*	-0.6903&	0.0001*
Lipase (U/L)	0.5126&	0.0001*	-0.3615@	0.0014*	-0.6129&	0.0001*
AST (U/L)	0.7808$	0.0001*	-0.6866&	0.0001*	-0.7023&	0.0001*
ALT (U/L)	0.7733$	0.0001*	-0.6493&	0.0001*	-0.7495&	0.0001*
BLU (mg⁄dl)	0.6915&	0.0001*	-0.5598&	0.0001*	-0.6859&	0.0001*
CRE (mg⁄dl)	0.6291&	0.0001*	-0.5929&	0.0001*	-0.5334&	0.0001*

PSS=Poisoning severity score; GCS=Glasgow Coma Scale; BuChE=Butyrylcholinesterase; AntiChE=Anticholinesterase; Na=Sodium; K=Potassium; BLG=Blood glucose; CK-MB=Creatine kinase-myocardial band; cTnI=Cardiac troponin I; AST=Aspartate aminotransferase; ALT=Alanine transaminase; BLU=Blood urea; CRE=Creatinine; P=Probability; *=Significant difference. P-value of <0.05 was considered significant. $=Strong correlation; &=Moderate correlation; @=Weak correlation.

DISCUSSION

Pesticide substances are extensively utilized in both developed and developing countries all over the world. These compounds have the potential to induce serious harmful effects on the health and death in human beings **(Aslan et al., 2011).**

The common use of AntiChE insecticides, especially the OPC and CMC, for various purposes has been associated with intense hazards effects including severe acute and chronic poisonings in human and animal species **(Kumar et al., 2010).** Every year, millions of people worldwide are intentionally and unintentionally poisoned by pesticides, particularly OPC and CMC **(Goldsmith et al., 2016).**

In the current study, the number of intoxicated females was predominating over males with insignificant difference observed in the mean age between the study groups. These findings harmonize with those achieved by several authors who documenting that females outnumbered males **(Exner and Ayala, 2009; Aslan et al., 2011).** In contrast, other authors have reported predominance of AntiChE poisoning among males **(Soltaninejad et al., 2007; Sam et al., 2009)**. The higher incidence of AntiChE poisoning among females may be due to the prevalence of the suicidal attempts by these products and different socio-economic problems.

The present study illustrated predominance of intoxication by OPC over CMC, which agrees with the results of other studies conducted in Egypt **(Mansour and Gamalludin, 2008)**, Iran **(Soltaninejad et al., 2007),** and Turkey **(Aslan et al**., **2011).** On the contrary, other researchers found more

prevalence of poisoning by CMC than OPC **(Saadeh et al., 1996).** Uncontrolled sale and preferred use of the highly toxic OPC may be consider as the main reasons for this high number of poisoning by OPC **(Sungur and Güven, 2001).**

In this study, malathion and aldicarb were the most common causative agents involved in OPC and CMC poisoning, respectively. Similar to this finding, several authors have found predominant use of these agents in poisoning incidents among the intoxicated cases **(Soltaninejad et al., 2007; Ibrahim et al., 2011).** Contrariwise to this, other study revealed that dichlorvos and phenthoate OPC mostly involved in acute human poisoning **(Saadeh et al., 1996; Abd El Al et al., 2016).** The higher prevalence of these AntiChE subtypes may be attributed their wide availability, low mammalian toxicity, and global popularity in controlling arthropods, ectoparasites, human head and body lice, and household insects as well as they are very cheap, easily obtained, and sold in unregulated packets **(Al-Attar, 2010).**

The present results emphasized predominance of patients with mild grade of PSS. Likewise, other studies have reported that most AntiChE poisoned patients presented to the hospital with mild intoxication as evaluated by PSS **(Weissmann-Brenner et al., 2002; Akdur et al., 2010).** Paradoxical to this, **Sam et al. (2009)** and **Azab (2015)** have reported that the majority of AntiChE poisoned cases suffered from severe toxicity with higher PSS grades. The severity of poisoning by AntiChE compounds depends on the dose, the route of exposure, the type and pharmacokinetic properties of substance, and the previous health status of the affected individual **(Soysal et al., 2011).**

The current findings announced that the vast majority of intoxicated cases were admitted to the hospital within two-hour after toxic exposure, which is similar to the results of other articles **(Kara et al., 2002; Karki et al., 2004; Vijayakumar et al., 2011).** Contradiction to this, other studies have depicted that the minority of AntiChE poisoned cases presented to the hospital within two-hour or shown prolonged mean arrival time following toxic exposure **(Ozer et al., 2007; Exner and Ayala, 2009).** This discrepancy may be due to the fact that the greater numbers of patients came directly to the unit without transfers from another health care facility and their residency present nearby the hospital.

In this study, the duration of hospital stay has been found to be shorter than that reported in the other studies **(Ozer et al., 2007; Kozacı et al., 2012)**. The length of hospital stay is directly proportional to the severity of poisoning, ingested dose, and arrival time to the hospital, which may contribute to this difference.

In the current study, various alternations in the vital signs and conscious level as evaluated by GCS were observed in some patients. Also, the mean values of vital signs and GCS scores of AntiChE-group on admission showed significant differences when compared with the corresponding values of both AntiChE-group at discharge and HC-group. However, these clinical changes were markedly improved at discharge as evidenced by insignificant differences from HC-group.

Analogous to the current observations of alternations in heart rate, many previous studies showed

more frequent occurrence of tachycardia than bradycardia among AntiChE intoxicated patients **(Yurumez et al., 2007; Aslan et al**., **2011; Vijayakumar et al., 2011).** Tachycardia may occur via nicotinic effects on adrenal medulla, sympathetic ganglionic stimulation, hypoxia, and/or dehydration (**Anand et al., 2009)**, while bradycardia results from the muscarinic effects of AntiChE compounds **(Yurumez et al., 2007)** or secondary to augmented vagal influence that shortens the atrial refractory period and increases the conduction time and the refractory period of sino-atrial and atrioventricular nodes **(Vijayakumar et al, 2011).**

Similar to the present results of abnormalities in blood pressure, preceding findings from many articles depicted more prevalent occurrence of hypertension than hypotension in AntiChE poisoned patients **(Yurumez et al., 2007; Vijayakumar et al., 2011).** In AntiChE poisoning, hypertension denotes nicotinic manifestation, whereas hypotension indicates cholinergic effect with vascular receptors overstimulation by circulating acetylcholine **(Eddleston and Clark, 2011; Vijayakumar et al., 2011)**.

Poisoning by AntiChE compounds can induce either central respiratory depression with a decreased respiratory rate or tachypnea **(Eddleston et al., 2006).** Parallel with the current results of respiratory dysfunction, other investigators reported various respiratory complications following AntiChE poisoning with more manifestations of tachypnea than bradypnea **(Bhattacharyya et al., 2011)**. On the contrary, the study of **Yurumez et al. (2007**) has shown that the majority of AntiChE poisoned cases experienced bradypnea. These complications may arise from acute stimulation of cholinergic receptors with subsequent development of bronchospasm and bronchoconstriction due to muscarinic effects, weakness and paralysis of respiratory diaphragm and intercostal muscles from nicotinic effects, and loss or cessation of central respiratory drive due to central effects, resulting in respiratory insufficiency, distress, dyspnea, failure, or apnea **(Satoh, 2006).**

Comparable with the current findings of changes in body temperature, several human reports have demonstrated considerable occurrence of hyperthermia than hypothermia after AntiChE poisoning. AntiChE compounds may interfere with the control of acetylcholine-regulated homeostasis, resulting in disturbance of normal thermoregulation mechanisms. Also, it has been suggested that changes in body temperature may be dose dependent. Generally, exposure to higher and lower doses of AntiChE compounds may be associated with hypothermia and hyperthermia, respectively **(Yurumez et al., 2007; Talaie et al., 2012).**

On the other hand, the results of several other publications regarding to AntiChE poisoning have illustrated either higher incidents of respiratory complications or less frequent occurrence of cardio-respiratory disturbances, tachycardia, tachypnea, and fall of blood pressure **(Ozer et al., 2007; Titlić et al., 2008),** which disagree with the present findings.

Acute AntiChE poisoning is one of the commonest causes of altered mental status. In the present study, clinical examination revealed that the main bulk of patients presented to the unit with different grades of altered mental

status as assessed by GCS. This finding is in agreement with several other literatures which illustrating that the vast majority of AntiChE intoxicated patients had a low GCS scores **(Sungur and Güven, 2001; Ozer et al., 2007; Kozacı et al., 2012).** Unlikely, other studies showed a lower incidence of comatosed patients due to AntiChE poisoning (**Baydin et al., 2007**). The neurological disturbances and decrease in GCS score can be explained by several factors such as direct impact of AntiChE compounds on peripheral and central nervous systems with subsequent accumulation of acetylcholine and overstimulation of synapses, hypoxia, and hemodynamic instability and hypoperfusion due to arrhythmia and hypotension **(Liu et al., 2008; Kozacı et al., 2012).**

In this study, PSS grades significantly correlated with the delay in hospital arrival and length of hospital stay periods as well as vital signs at the time of admission.

Similarly, **Sam et al. (2009)** have concluded that the delay in pre-hospitalization time can influence the severity of AntiChE poisoning as evidenced by a significant linear correlation between the pre-hospitalization period and PSS grades. On the other side, **Akdur et al. (2010) and Azab (2015)** have found insignificant correlation and no significant difference, respectively, between pre-hospitalization period and PSS grades.

In addition, **Rehiman et al. (2008)** and **Azab (2015)** have reported a significant positive association and a significant difference, respectively, between the severity of poisoning grades and the length of hospital stay, thus the hospitalization period being higher in grade 3 intoxicated patients on admission. On the contrary, **Sam et al. (2009)** have observed insignificant correlation between PSS and the hospitalization period.

Several authors have suggested that criteria such as cardio-respiratory rates can assess severity of AntichE intoxication and help in predicting possible outcome **(Senanayake et al., 1993).**

Also, the study of **Yu et al. (2012)** has showed significant association between altered vital signs and poisoning-related severity. The prior authors stated that vital signs may play an important role in diagnosis, assessing, and prediction of the clinical severity and outcome among poisoned patients. Unlikely, **Davies et al. (2008)** has proclaimed that the heart rate is of little value in predicting the outcome of AntiChE poisoning and weakly contributes to the predictive value of the PSS.

The present results demonstrated significant correlation between the GCS scores and the delay in hospital arrival and length of hospital stay periods as well as vital signs at the time of admission.

Likewise, the study of **Muley et al. (2014)** has shown markedly lower GCS score in concomitant with prolonged time elapsed after exposure to AntiChE substances in patients needed mechanical ventilation on admission. Also, the sensitivity and specificity of GCS at the time of admission to the medical facilities significantly contribute in predicting severity and outcome in dimethoate intoxicated patients **(Davies et al., 2008).** Furthermore, the study of **Muley et al. (2014)** has demonstrated a significant negative association between the duration of hospital stay and the GCS scores.

The current work illustrated significant correlation between the BuChE levels and the pre-hospitalization and hospitalization periods as well as vital signs at the time of admission. The same findings have been reported by **Muley et al. (2014)**, wherein great reduction in serum BuChE levels and considerable delay in pre-hospitalization periods are noticed in AntiChE poisoned patients, in addition, a moderate negative correlation is also seen between serum BuChE levels and hospital stay.

In general, the more delay in pre-hospitalization period, the more severity of poisoning degree including alternations in vital signs and mental status, and the more increase in hospitalization period. These abnormalities may be due to delay in early therapeutic intervention that subsequently increases the initial serum level of AntiChE substances, leading to irreversible organ damage **(Akdur et al., 2010).**

In the present study, a significant difference was noticed on comparing the mean GCS scores with the PSS grades of the three intoxicated-groups besides there was also a significant negative correlation between the degree of severity of poisoning and GCS. These findings are in agreement with the result of **Azab (2015)** who observed a significant distinction on matching GCS scores between the PSS grades (The lower the GCS score, the higher the PSS grade) as well as with the reports of **Sam et al. (2009)** and **Akdur et al. (2010)** who found a significant negative correlation between these two variables owing to low GCS scores and high PSS grades among AntiChE intoxicated patients. This may be due to high lipophilic properties of OPC with their ease of blood-brain barrier penetration. Although CMC do not easily cross into the nervous system, but in massive poisonings, neurological dysfunctions may occur from hypoxic effects secondary to pulmonary toxicity and paralysis **(Eddleston and Clark, 2011).**

In the current study, there was a significant inverse correlation between the PSS grades and BuChE enzyme levels. This finding is in line with previous other reports which have described a significant negative correlation between the derangement of serum BuChE levels and/or erythrocytes cholinesterase activities and the severity of cholinergic toxidrome following AntiChE poisoning **(Weissmann-Brenner et al., 2002; Rehiman et al., 2008; Soysal et al., 2011).** Conversely to this, several other authors did not find a relationship between the PSS and the decrease in cholinesterase activities of AntiChE intoxicated cases **(Sam et al., 2009; Akdur et al., 2010; Azab, 2015).** This negative correlation is due to that AntiChE chemicals mainly cause their toxicity through inhibition of the activities of different cholinesterase enzymes **(Lotti, 2002; Erdman, 2004).**

In the current study, the chief detected abnormal biochemical findings in some patients were low pH of acid-base balance (metabolic acidosis), hypernatremia, hypokalemia, and hyperglycaemia. There were significant differences between AntiChE-group at the time of admission and HC-group regarding the mean levels of these biochemical parameters; however, there were insignificant differences among both groups at discharge. These parameters showed different correlation coefficient significances with PSS grades, GCS scores, and BuChE levels.

Metabolic acidosis is a relatively

common complication in AntiChE poisoning and can occur due to either hypoventilation or hypotension **(Erdman, 2004**; **Bhattacharyya et al., 2011).** Also, several researchers have determined occurrence of electrolytes abnormalities including hypernatremia **(Laudari et al., 2014)** and hypokalemia **(Tsai et al., 2007; Laudari et al., 2014; Abd El Al et al., 2016)** in AntiChE poisoned cases. These changes in electrolytes levels may be explained by excess cholinergic stimulation and/or severe vomiting and diarrhea **(Eddleston and Clark, 2011).**

Additionally, several investigators proclaimed that hyperglycaemia is a common complication and a clinically significant finding accompanied with AntiChE poisoning **(Sungur and Güven, 2001; Sahin et al., 2002; Yurumez et al., 2007).** This increase in blood glucose levels may be due to secondary pancreatic injury **(Singh et al., 2007)** or release of catecholamines from the adrenal medulla that subsequently stimulate glycogenolysis and induce hyperglycemia **(Yurumez et al., 2007; Eddleston and Clark, 2011).**

Liu et al. (2008) and **Moon et al. (2016)**, respectively, have found that the low pH and hyperglycaemia positively correlated with the clinical severity of acute AntiChE poisoning on admission, which are in agreement with the present findings.

The present study demonstrated alternations in the assayed enzymatic biomarkers values of poisoned cases. Low BuChE levels were seen in all cases, whereas increased serum levels of CK-MB, cTnI, amylase, lipase, AST, ALT, BLU, and CRE were detected in some cases.

In general, BuChE levels typically decline more quickly after exposure to AntiChE chemicals; therefore, its activity is routinely assayed for the early diagnosis and continuous monitoring of poisonings by these compounds **(Lotti, 2002; Erdman, 2004; Rehiman et al., 2008).** In this work, poisonings by AntiChE chemicals were fundamentally associated with a significant reduction in BuChE activity, which is similar to numerous other studies conducted in Egypt **(Ibrahim et al., 2011; Abdel Haleem et al., 2012; Abd El Al et al., 2016)** and other countries **(Rehiman et al., 2008; Sam et al., 2009; Akdur et al., 2010; Soysal et al., 2011).**

Also, comparison between the mean serum BuChE level at the time of discharge and that at admission revealed significant increase, but still significantly lower than HC-group value. This might be attributed to the early arrival of patients to the hospital as well as early administration of the antidotes such as atropine and cholinesterase reactivator oxime (obidoxime chloride). In untreated patients, normalization of inhibited BuChE takes about 4-6 weeks to return to pre-exposure levels and its activity increases by about 25-30% in the first 7-10 days **(Eddleston and Clark, 2011).**

Elevations of serum CK-MB and cTnI levels were noticed in some patients and there were significant increases in their mean serum values of AntiChE-group at the time of admission as compared to the control values. However, at the time of discharge, their levels were markedly decreased and became non-significantly different from the HC-GP. These findings are compatible with many other publications illustrating augmentation of CK-MB and/or cTnI cardiac enzymes levels in AntiChE

intoxicated patients **(Wanf et al., 2011; Cha et al., 2014)** with statistically significant differences between poisoned and control groups **(Wanf et al., 2011).**

The present study showed significant positive correlations between the changes in CK-MB and cTnI cardiac enzymes and the severity of poisoning, which have also been reported by others **(Kharoub and Elsharkawy, 2008; Wanf et al., 2011; Cha et al., 2014).**

These alterations in cardiac enzymes levels may be attributed to the direct myocardial cell damage or ischemic injury, resulting in leakage of these enzymes into blood **(Anand et al., 2009; Cha et al., 2014).**

In the current work, increases in serum amylase and lipase were noticed in several patients of AntiChE-group at the time of admission. These pancreatic enzymes showed significant differences when compared with HC-group. Analogously, several other literatures have shown transient development of hyperamylasemia and hyperlipasemia in patients with AntiChE poisoning **(Sahin et al., 2002; Singh et al., 2007; Yurumez et al., 2007; Abd El Al et al., 2016).**

Also, the present results illustrated significant positive association between the PSS grades and both lipase and amylase. These findings are in harmony with several other studies clarifying that elevation in serum amylase and lipase levels closely related to the clinical severity and the presence of different complications such as shock and respiratory failure **(Kozacı et al., 2012).** In contrast, the work of **Singh et al. (2007)** has not detected any correlation between hyperamylasemia and poisoning severity.

The possible pathogenetic mechanisms of AntiChE-induced pancreatic insult are multifactorial, including excessive release of acetylcholine from pancreatic tissue with subsequent prolonged hyperstimulation of pancreatic acinar cells **(Harputluoglu et al., 2007)** or development of pancreatitis secondary to increase pancreatic sensitivity and secretions in response to acetylcholine, acinar cell vacuolation, and edema **(Brahmi et al., 2005).**

The present study depicted elevations of serum AST and ALT levels in some intoxicated cases on admission and these changes were found to be statistically significant when compared with HC-group. These results are in concordant with many other literatures clarifying abnormal increases in liver enzymes among AntiChE intoxicated patients **(Sahin et al., 2002; Tsai et al., 2007; Yurumez et al., 2007; Awad et al., 2014).** On the contrary, other Egyptian study has shown insignificant differences between AntiChE poisoned patients and healthy control individuals regarding the serum enzyme levels of AST and ALT **(Abdel Haleem et al., 2012).**

In the present study, there was significant positive correlation between elevated liver enzymes and the severity of poisoning. Also, other results of acute AntiChE poisoning displaying higher liver enzymes levels as potential risk factors and could be used as a predictive value for poisoning severity **(Ram et al., 1991).**

Multiple mechanisms such as exposure time and dose **(Erdman, 2004),** formation and accumulation of more toxic metabolites than the parent AntiChE compounds, leading to P450 inactivation **(Ncibi et al. 2008)**, or oxidative stress in hepatocellular mitochondria, resulting in disturbance

of oxidants/antioxidants balance **(Yurumez et al., 2007)** may contribute in the development of AntiChE-induced hepatotoxicity with subsequent elevation of blood liver enzymes.

The current study showed augmentation of BLU and CRE levels in some intoxicated cases on admission as well as a significant positive correlation between the severity of poisoning and kidney function tests. These abnormalities are in harmonization with several other reports elucidating impairment of renal function in AntiChE poisoned patients, which is widely correlated with the severity of poisoning **(Agostini and Bianchin, 2003; Abd El Al et al., 2016).** In contrast, other authors have documented insignificant variations between AntiChE poisoned cases and healthy control persons concerning the serum enzyme levels of blood urea nitrogen and creatinine **(Abdel Haleem et al., 2012).** Acute nephrotoxicity after AntiChE poisoning appears to result from proximal renal tubular damage secondary to elevation of BuChE levels in renal tissue, high intratubular concentrations of AntiChE chemicals, rhabdomyolysis, and/or hypovolaemia from dehydration **(Agostini and Bianchin, 2003; Yurumez et al., 2007).**

The present work declared different significant correlation between BuChE levels and all investigated biochemical and enzymatic biomarkers of AntiChE-groups on admission. As noticed in the previous studies, severe perturbations of these laboratory parameters are strongly accompanied with markedly low BuChE levels in AntiChE poisoned patients.

CONCLUSION:

Acute AntiChE poisoning has induced various vital signs dysfunctions, metabolic derangements, and serious toxic effects on cardiac, pancreatic, hepatic, and renal organs as evidenced by elevations of their corresponding serum enzymatic biomarkers. Although the PSS grades, GCS scores, and BuChE levels at presentation appear similarly effective clinical indices for assessing and predicting the severity of AntiChE poisoning as manifested by their significant correlations with the investigated clinical and laboratory parameters as well as between each other, the efficacy of PSS outperform the GCS and BuChE effectiveness. The patients with evidence of moderate and severe degree of poisoning need to be monitored closely.

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الأهمية التشخيصية والتنبؤية لدرجة شدة التسمم، مقياس غلاسكو للغيبوبة، العلامات الحيوية، والدلائل الحيوية الأنزيمية في حالات التسمم الحاد بالمبيدات الحشرية المضادة لإنزيم الكولينستريز

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الملخص العربى

المقدمة : مركبات الفوسفات العضوية والكربامات هي مبيدات الآفات المضادة لعمل إنزيم الكولينستريز ذات سمية شديدة ، تستخدم على نطاق واسع في جميع أنحاء العالم، ولا تزال مسؤولة عن التسمم الوبائي. وقد أفادت التقاريرعن حدوث اختلال وظيفي بالعديد من أعضاء الجسم عقب التسمم بمضادات إنزيم الكولينستريز. **الهدف من البحث:** هدفت هذه الدراسة إلى تقييم العلاقة بين درجة شدة التسمم، مقياس غلاسكو للغيبوبة، ومستويات البوتيريلكولينستريز مع التغيرات السريرية والمخبرية فى المرضى البالغين الذين يعانون من التسمم الحاد بمضادات الكولينستريز. **طرق البحث:** اشملت هذه الدراسة على 25 فردا في المجموعة الضابطة و75 مريضا في المجموعة المضادة للكولينستريز. وفقا لخصائص شدة التسمم، تم تقسيم المرضى إلى ثلاث مجموعات تسمم طفيفة، معتدلة، أو شديدة. تم تسجيل البيانات الخاصة بالجنس، والعمر، والمادة المضادة للكولينستريز المسببة للتسمم. تم قياس المتغيرات السريرية مثل درجة شدة التسمم، التأخير في وقت الوصول للمستشفى، وظائف العلامات الحيوية، مجموع نقاط مقياس غلاسكو للغيبوبة ، مدة الإقامة في المستشفى. مستويات الدم من المتغيرات البيوكيميائية (الرقم الهيدروجيني والصوديوم والبوتاسيوم، والسكر العشوائي) والدلائل الحيوية الأنزيمية (بوتيريلكولينستريز، إنزيمات القلب مثل الكرياتين كيناز وتروبونين-ي، إنزيمات البنكرياس مثل الأميليز والليباز، إنزيمات الكبد مثل ناقلات أمين الألانين والأسبارتات، إنزيمات الكلى مثل اليوريا والكرياتينين. **النتائج:** كانت معظم الحالات من الإناث، تتراوح أعمارهم بين 23,82 ± 0,82 سنة، تسممت بمركبات الفوسفات العضوية، وخاصة الملاثيون. النتائج السريرية الأكثر شيوعا كانت أعراض تسمم طفيفة ، تأخير في الوصول للمستشفى لمدة 2 ساعة ، ومستوى النعاس على مقياس غلاسكو للغيبوبة، ومدة اقامة بالمستشفى قصيرة نسبيا ≤24 ساعة ، في حين أن الخلل بالعلامات الحيوية اشتمل على زيادة بدقات القلب، ارتفاع ضغط الدم ، زيادة بسرعة التنفس، وارتفاع بالحرارة. اشتملت التغيرات الكيميائية الحيوية الرئيسية على استقلاب حمضي، زيادة مستوى الصوديوم ، نقص مستوى البوتاسيوم ، وارتفاع السكر. كانت مستويات بوتيريلكولينستريز منخفضة في جميع الحالات في حين زيادة مستويات الدلائل الحيوية الأنزيمية التى تم دراستها لوحظت في بعض الحالات. عند الدخول للمستشفى، أظهرت النسب والقيم المتوسطة لجميع المتغيرات السريرية والمخبرية فروق ذات دلالة إحصائية بين مجموعات التسمم الثلاث وبين المجموعة المضادة للكولينستريز والمجموعة الضابطة. بالإضافة إلى ذلك، أظهرت مستويات درجة شدة التسمم، مجموع نقاط مقياس غلاسكو للغيبوبة، ودرجة تثبيط البوتيريلكولينستريز ارتباطا كبيرا مع التأخير في وقت الوصول للمستشفى، جميع العلامات الحيوية، مدة اقامة بالمستشفى، وجميع المتغيرات المختبرية وكذلك بين بعضها البعض. عند الخروج من المستشفى، أظهرت التغيرات الأولية السريرية والمختبرية تحسنا ملحوظا والفروق ليست ذات دلالة إحصائية مقارنة بالمجموعة الضابطة باستثناء مستويات البوتيريلكولينستريز التى مازالت أقل من قيم المجموعة الضابطة. **الخلاصة:** ينبغي أن تؤخذ هذه المضاعفات الخطيرة المحتملة في الاعتبار من قبل الأطباء الذين يتعاملون مع حالات التسمم بالمركبات المضادة للكولينستريز. أيضا، درجة شدة التسمم، مقياس غلاسكو للغيبوبة، ومستويات البوتيريلكولينستريز تعد أدوات مفيدة للتنبؤ بشدة التسمم بالمركبات المضادة للكولينستريز.