CORRECTED QT AS A SIMPLE TOOL FOR PREDICTION OF NEED FOR VENTILATION AND MORTALITY IN ACUTE ORGANOPHOSPHATE POISONING

BY

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ABSTRACT

Organophosphate (OP) toxicity represents a major public health concern, primarily in the developing world. The most common complication from OP exposure is respiratory failure. Nevertheless, cardiac complications have been reported with OP poisoning. This current prospective observational cross sectional study was conducted to evaluate electrocardiographic findings as predictor of major outcome (need for ventilation and mortality) in acute OP toxicity. Thirty eight adult male patients acutely intoxicated by OP were included in the study. The severity of symptoms and signs of acute OP poisoning (OPP) was graded into: mild, moderate and severe grade OPP. Electrocardiography (ECG) was recorded and analyzed for rate, rhythm, axis, voltage, ST and T wave abnormalities, conduction defects, PR interval. The ECG changes induced by acute OPP were graded into: minor, moderate and severe. Corrected QT interval (QTc) was estimated according to Bazett's formula;. Fifteen patients (39.5%) showed a prolonged QTc interval (0.518 \pm 0.055 seconds). Both need for ventilation and mortality were noticed in patients with prolonged QTc interval rather than in patients with a normal QTc interval. Significant statistical association was perceived between QTc interval and both manner and route of poisoning, OPP severity, serum cholinesterase level, number of toxogonine ampoules and need for intubation and mechanical ventilation. It is recommended to consider QTc interval as an applicable bedside parameter that helps to identify advanced grade OPP patients, especially those presented by suicidal attempts, severe organophosphate intoxication, oral route of exposure and prolonged QTc interval should be admitted in ICU as high risk patients.

Keywords: Organophosphate, electrocardiography, QTc, predictors, ventilation, mortality.

INTRODUCTION

Organophosphate compounds (OPCs) are esters of phosphoric acid. They were

designed as insecticides, herbicides and nerve agents (Carey et al., 2013). Their application is still the most effective and accepted mean for plant protection from pests (Vijayakumar et al., 2011).

Organophosphate (OP) toxicity represents a major public health concern, primarily in the developing world, where widespread availability of agricultural pesticides may account for 200,000 deaths every year (Connors et al., 2014). In Egypt, OP toxicity is a common cause of morbidity and mortality. It accounts for more than 50% of insecticide poisoned patients (Ibrahim et al., 2011).

Organophosphate toxicity is mediated through acetylcholinesterase enzyme (AChE) inhibition. Subsequently, initial overstimulation of cholinergic synapses, neuromuscular junctions and central nervous system will occur, followed by paralysis. Cholinergic overload leads to muscarinic, nicotinic and central nervous system signs and symptoms (Kose et al., 2010).

The most common complication from OP exposure is respiratory failure. Nevertheless, cardiac complications such as various arrhythmias, conduction disturbances, hypertension-hypotension and myocardial damage have been reported with OP poisoning. Such complications might be serious and are often fatal. However, they are potentially preventable if they are recognized early and treated adequately (Bar-Meir et al., 2007; Kose et al., 2009; Cha et al., 2014).

The existing knowledge of OP induced cardiac toxicity is limited publications and case reports. Furthermore, the extent, frequency, and pathogenesis of organophosphate induced cardiac toxicity have not been well defined. Moreover, studies correlating ECG findings with OP induced cardiac pathology are lacking (Anand et al., 2009; Yurumez et al., 2009).

Therefore, the current study was conducted to evaluate electrocardiographic findings as predictor of major outcome (need for ventilation and mortality) in acute OP toxicity.

PATIENTS and METHODS

This prospective observational cross sectional study was performed at Poison Control Unit, Emergency Hospital Tanta University in nine months period. It was approved by the Research Ethical Committee, Faculty of Medicine, Tanta University. All participants received detailed information concerning the aims of the research work, and informed consent was obtained from each participant or his relatives prior to the commencement of the study.

Adult male patients acutely intoxicated by OP have been included in the study. Patients with history of cardiac diseases (rheumatic heart disease, atrioventricular arrhythmias, congestive cardiac failure, 2nd or 3rd degree heart block, abnormalities on echocardiogram and ischemic heart disease) were excluded from the study. Furthermore, patients who received any medical treatment before admission together with patients of mixed exposure were similarly excluded. Patients with any pre-existing chronic diseases including; hypertension, diabetes, hepatic and renal diseases as well as cancer were excluded.

Diagnosis of acute organophosphate poisoning (OPP) was based on history of OP exposure, characteristic OP symptoms and signs, clinical improvement after atropine and oximes (toxogonine) administration and decrease in serum cholinesterase enzyme levels (Karki et al., 2004; Liu et al., 2012). Sociodemographic data (age, occupation, education, residence and marital state) and toxicological data (route and circumstances of poisoning and pre-hospitalization period) were explored.

The severity of symptoms and signs of acute OPP was graded according to Minton and Murray (1988) into: mild OPP; fatigue, headache, blurred vision, dizziness, nausea, vomiting, excessive sweating, salivation, abdominal pain and tightness in chest. Moderate OPP include symptoms of mild poisoning plus muscular fasciculation, weakness, inability to walk, chest crepitation and miosis. Severe OPP include symptoms of moderate poisoning

plus unconsciousness, flaccid paralysis, respiratory distress, cyanosis and marked miosis with loss of pupil reflexes.

Blood samples were collected immediately after admission before administration of any medication under complete aseptic conditions. Two milliliters venous blood was kept into a clean dry centrifuge tube and left to stand for few hours before centrifugation to avoid hemolysis. Serum was separated and then used for estimation of serum cholinesterase level using butyrylthiocholine substrate, commercial kit supplied by Biodiagnostic, Egypt (normal value 5400-13200 U/L) (Blawen et al., 1983).

Electrocardiography (ECG) was recorded on admission, every six hours and when an abnormality was observed on cardiac monitor. The ECG was analyzed for rate, rhythm, axis, voltage, ST and T wave abnormalities, conduction defects, PR interval. Corrected QT interval (QTc) was estimated according to Bazett's formula; where QTc is the QT interval corrected for heart rate, and RR is the interval from the onset of one QRS complex to the onset of the next QRS complex, measured in seconds, often derived from the heart rate (HR) as 60/HR (here QT is measured in milliseconds). QT is considered normal up to 0.44 second. Echocardiography was done as soon as possible after admission (Bazett, 1920).

The ECG changes induced by acute OPP were graded according to Poisoning Severity Score into: minor; isolated extrasystoles. moderate; sinus tachycardia (HR ~140-180 in adults), frequent extrasystoles, atrial fibrillation/flutter, AV-block I-II, prolonged QRS and QTc-interval, repolarization abnormalities, myocardial ischemia. Severe; severe sinus bradycardia (HR ~40 in adults), severe sinus tachycardia (HR ~180 in adults), life-threatening ventricular dysrhythmias, AV block III, asystole, myocardial infarction (Persson et al., 1998).

The disposal of blood collection syringes, tubes and body fluids (blood samples) was safely done to avoid any risk of environmental pollution. Privacy and confidentiality of patients' records and data was ascertained through coding system.

Statistical analysis:

Data were collected and entered to the computer using SPSS (Statistical Package for Social Science) program for statistical analysis (version 20). Data were entered as numerical or categorical, as appropriate. (D) test of Kolmogorov-Smirnov (KS) was used to test normality of the distribution of variables, but non-parametric statistics (minimum and maximum, median and inter-quartile range) were adopted even when KS test was not significant, and comparison using Kruskal-Wallis test was used to test several independent samples,

and Mann-Whitney U test was used to test two independent samples. Chi-Square test (Monte-Carlo corrected) for n x m table or Yate's correction (for 2x2) tables as well as Phi and Cramer's V test were used to test association of categorical variables.

In the present study an alpha level was set to 5% with a significance level of 95%, and a beta error accepted up to 20% with a power of study of 80% (Field, 2006).

RESULTS

During the study period, forty three patients have fulfilled the inclusion and exclusion criteria. Five patients have refused to participate in the study and thirty eight patients have accepted study participation. The age of participant ranged from 18 to 65 years, mean 36.18 ± 13.34 . The rest of the sociodemographic characteristics of the participant patients are illustrated in table (1). Toxicological and clinical data of the participant patients are demonstrated in table 2.

Electrocardiographic examination revealed elevated ST segment in four patients (10.5%) and inverted T wave in nine patients (23.7%). In the same time, atrial fibrillation, extra systole, bundle branch block, hyperacute T wave, poor R wave progression and abnormal Q wave were found in two patients (5.3%) each. Table (3) revealed no significant statistical asso-

ciation between grading of electrocardiographical changes and any of the sociodemographic, poisoning, clinical data and major outcome (need for ventilation and mortality).

The studied group revealed a mean QTc interval of 0.45 ± 0.067 seconds (range 0.35–0.61 seconds). Fifteen patients (39.5%) showed a prolonged QTc interval (0.518 \pm 0.055 seconds). Both need for ventilation and mortality were noticed in patients with prolonged QTc interval rather than in patients with a normal QTc interval. Significant statistical association was

perceived between QTc interval and both manner & route of poisoning, OPP severity, serum cholinesterase level, number of toxogonine ampoules and intubation & mechanical ventilation (Table 4).

It was observed that eight patients (21.05%) required intubation and mechanical ventilation assistance. Among those intubated patients, two patients died. Seven patients out of the intubated patients registered prolonged QTc interval. All of the intubated patients were suicidal attempts. Moreover, oral route was the main route of exposure (seven patients) (Table 5).

Table (1) : Sociodemographic data of the organophosphorus poisoning patients (number= 38).

Chara	acteristic variable	n	0/0
Sex	Males	38	100
	Females	0	0
Marital status	Single	13	34.2
	Married	25	65.8
Residence	Urban	13	34.2
	Rural	25	65.8
Education	Illiterate	19	50
	Read & write	5	13.2
	Secondary school	14	36.8
	High education	0	0
Work	Unemployed	5	13.2
	Farmer	18	47.4
	Skilled worker	6	15.79
	Student	9	23.68

n: number, %: percentage.

Table (2): Toxicological and clinical data of the organophosphorus poisoning patients (number=38).

Characteristic variable		n	%	
Manner of poisoning	Accidental		22	57.9
	Suicidal		16	42.1
Route of poisoning	Ingestion		15	39.5
	Combined		23	60.5
Organophosphorus poisoning	Normal			
severity	Mild		9	23.7
	Moderate		20	52.6
	Severe		9	23.7
ECG severity grading	Normal		7	18.4
	Mild		4	10.5
	Moderate		11	28.9
	Severe		16	42.1
Outcome	Improved & discharged		23	60.5
	Escaped		13	34.2
	Intubation & Mechanical ventilation		8	21.05
	Mortality		2	5.3
	Minimum	Maximum	Mean	SD
Pre-hospitalization period (hr)	1	24	6.5	5.2
Cholinesterase level	10.83	11169	2413.77	2057.19
Number of Atropine ampoules	0	88	16.43	19.85
Number of Toxogonine ampoules	0	28	4.75	5.99
QTc	0.35	0.61	0.45	0.067

n : number, %: percentage, QTc: corrected QT interval, SD.: standard deviation.

Table (3) : ECG grading against sociodemographic, toxicological and clinical data (number=38).

Characteristic variable		ECG severity grading	
		X ^{2 a}	P value
Age ^b		1.477	0.478
Marital Status	c	0.695	0.882
Residence ^c		2.412	0.539
Education ^c		12.613	0.044
Work ^c		14.042	0.558
Manner of poisoning ^c		7.633	0.052
Route of poison	ing ^c	8.994	0.203
Pre-hospitalizati	ion period (hr) ^b	0.971 0.615	
Cholinesterase l	linesterase level ^b 0.917 0.66		0.632
No. of Atropine ampoules ^b		5.780	0.056
		0.308	
Organophosphorus poisoning severity		10.261	0.118
Major	Intubation & Mechanical ventilation ^c	4.190	0.237
Outcome	Mortality ^c	0.966	1.000

a: Pearson Chi-Squared test, b: Kruskal-Wallis Test, c: Monte Carlo significance (PMC)

Table (4) : QTc against sociodemographic, toxicological and clinical data (number=38).

	Characteristic variable	QTc	
	Characteristic variable	X ^{2 a}	P value
Age ^b		0.524	0.616
Marital Status ^c		0.369	0.544
Residence ^c		1.708	0.191
Education ^c		1.372	0.549
Work ^c		7.067	0.224
Manner of poisoning	ng ^c	9.914	0.002*
Route of poisoning	e	10.35	0.003*
Pre-hospitalization	period (hr) ^b	0.833	0.411
Cholinesterase leve	l ^b	2.344	0.018*
No. of Atropine an	poules ^b	0.882	0.391
No. of Toxogonine ampoules ^b		2.765	0.007*
Organophosphorus poisoning severity 11.399		0.004*	
Major Outcome d	Intubation & Mechanical ventilation ^e	7.402	0.007*
	Mortality	0.000	1.000

^{*} Significant, QTc: corrected QT interval a: Pearson Chi-Squared test , b: Mann-Whitney U test, c: Monte Carlo. (PMC), d: Yate's correction for Chi-squared test, e: Yate's continuity correction.

Table (5): Distribution of intubated and mechanically ventilated patients (number=8) as regard manner, route of exposure, OPP poisoning severity, QTc interval and fate of exposure (number=38).

Characteristic variable		n	%
Manner	Suicidal	8	100
Route	Ingestion	7	87.5
	Combined	1	12.5
Organophosphorus poisoning severity	Severe	8	100
QTc	Normal	1	12.5
	Prolonged	7	87.5
Fate	Died	2	25
	Improved & Discharged	6	75

n.: number, %: percentage QTc: corrected QT interval.

DISCUSION

Organophosphate poisoning (OP) is a major global health problem and high mortality is seen in developing countries. The present study was designed to assess the effectiveness of ECG examination to predict the major outcome of OP poisoning (need for ventilation and mortality). In order to achieve this target, sociodemographic, poisoning, clinical data and major outcome were analyzed against ECG findings in OP poisoned patients.

In the current study electrocardiographic examination revealed elevated ST segment, abnormal Q, R, and T wave which indicate ischemia, in addition to different types of arrhythmias. Worldwide and over time, several researches have described more or less similar ECG changes in OP toxicity. The particular mechanism of such ECG changes is not well known. However, it might include sympathetic and parasympathetic over-activity, hypoxemia, acidosis, electrolyte derangements, and direct cardiotoxic effect of the compounds (Kiss and Fazekas, 1979; Saadeh et al., 1997; Karki et al., 2004; Taira et al., 2006; Bar-Meir et al., 2007; Lakhair et al., 2012; Cha et al., 2014).

Grading of electrocardiographical changes was not significantly associated with any of the sociodemographic poisoning and clinical data. Consequently, it was not significantly associated with the major outcome (need for ventilation and mortality). A result supported by Akdur et al. (2010) who believe that ECG findings of OP poisoned patients cannot solely suffice to determine poisoning severity or short-term prognosis and show no relationship with poisoning severity score.

Nevertheless, Ludomirsky et al. (1982) described three phases of cardiac toxicity after OP poisoning. The first phase is a brief period of increased sympathetic tone. Phase 2 is a prolonged period of parasympathetic activity and phase 3 in which QT prolongation is followed by torsade de pointes, ventricular tachycardia, and then ventricular fibrillation.

In the current study fifteen patients (39.5%) showed a prolonged QTc interval. Comparable studies by Jang et al. (1995); Grmec et al. (2004); Karki et al. (2004); Shadnia et al. (2009) and Lakhair et al. (2012) signified prolonged QTc interval in 39%, 37.8%, 65%, 59.5% and 67% of OP poisoned cases respectively. The exact cellular mechanism of OP induced QT prolongation are not fully understood. It may involve inhibition of potassium outward channels. The ensuing intracellular surplus of positive ions delays ventricular repolarization (thus prolonging the QT interval) and may trigger early afterdepolarizations (EADs). These EADs, may reach threshold amplitude and trigger

ventricular arrhythmias (Viskin, 2000; Khan and Gowda, 2004).

The results obtained in this study pointed to association between QTc interval and both manner and route of poisoning, OPP severity, serum cholinesterase level and number of toxogonine ampoules. Data reported in previous comparable study designs displayed conflicting results. For example, significant association was noticed between QTc interval and both period of hospital stay, atropine requirements and respiratory failure with severe poisoning (Grmec et al., 2004; Shadnia et al., 2009). Contradictory to these results, no statistically significant association could be found between QTc interval and either poison severity score or blood cholinesterase level (Baydin et al., 2007; Akdur et al., 2010).

In the current study, it was anticipated to find significant statistical association between QTc interval and need for intubation & mechanical ventilation. Where, it was observed that eight patients required intubation and mechanical ventilation assistance, out of them seven patients registered prolonged QTc interval. Previous literatures supported this finding by identifying respiratory failure in patients with QTc interval prolongation rather than in those with normal QTc interval in OPP (Grmec et al., 2004).

In the current study, two patients died, both of them registered prolonged QTc interval. Despite this, no association was detected between prolonged QTc interval and mortality. One of them grieved from ventricular tachyarrythmia not responding to DC shock then cardiac arrest. The other underwent severe chest infection leading to hypoxia and brain death.

The previous finding contrasts Chuang et al. (1996) who reported that OP exposed patients with QTc interval prolongation had a higher mortality ratio, compared with those without QTc interval prolongation. Moreover, Shadnia et al. (2009) described significantly higher mortality rate in the long QTc OP exposed patients than in normal QTc OP exposed patients. Such controversy could be attributed to the difference in the sample size, exclusion and inclusion criteria.

It was striking to find that all of the intubated, ventilated and died patients were suicidal attempts and classified as severe grade OP toxicity. Furthermore, the main route of exposure was the oral route together with prolonged QTc interval (seven patients out of eight intubated, ventilated and died patients).

From this study, it could be concluded that, in OP exposed patients, ECG changes is not a predictor for major outcome (need for ventilation and mortality). In the same time, QTc interval could be considered as good predictor for need for ventilation. Suicidal attempts, severe organophosphate intoxication, oral route of exposure together with prolonged QTc interval are alarming signs of need for ventilation and mortality.

It is recommended to consider QTc interval as an easy and applicable bedside parameter that helps young toxicologists to identify advanced grade OP poisoning patients in the initial assessment in the ED. OP poisoned patients who are presented by suicidal attempts, severe organophosphate intoxication, oral route of exposure and prolonged QTc interval should be admitted in ICU as high risk patients. Further researches to investigate the association between QTc interval and mortality are required.

The principal limitation of the current study is its small sample size precipitated by the wide range of inclusion and exclusion criteria. Moreover, name of the used organophosphates could not be clearly determined.

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طول الفاصلة QT المعدلة كوسيلة سملة للتنبؤ بالحاجة إلى التنفس الصناعي وبالوفاة في حالات التسمم الحاد بمركبات الفوسفات العضوى

المشتركون في البحث

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التسمم بمركبات الفوسفات العضوي يمثل مصدر قلق رئيسي للصحة العامة، ولا سيما في العالم النامي. ويعد أكثر المضاعفات شيوعاً نتيجة التسمم بمركبات الفوسفات العضوي فشل الجهاز التنفسي. ومع ذلك فإن مضاعفات القلب قد تم تسجيلها في حالات التسمم بمركبات الفوسفات العضوي.

أجريت هذه الدراسة المقطعية المراقبة الحالية لتقييم نتائج التغيرات في تخطيط القلب الكهربائي كمتنبئات لنتائج رئيسية في حالات التسمم الحاد بمركبات الفوسفات العضو يمثل الحاجة للتنفس الصناعي والوفاة.

شارك ثمانية وثلاثون من المرضى الذكور البالغين الذين تم تشخيصهم بالتسمم الحاد بمركبات الفوسفات العضوي في الدراسة. وتم تدريج حالة التسمم حسب شدة الأعراض إلى: خفيف، معتدل، وشديد. تم تسجيل تخطيط القلب الكهربائي (ECG) وتحليله حسب المعدل، الإيقاع، المحور، والجهد، و شذوذ موجة ST ، وعبوب التوصيل، الفاصلة PR. وطول الفاصلة (QTc) المعدكة وفقا لمعادلة Bazett . وتم تصنيف التغيرات في تخطيط القلب الكهربائي الناجمة عن التسمم الحاد بمركبات الفوسفات العضوي إلى: صغرى، متوسطة، شديدة.

أظهر خمسة عشر مريضا (\$39.5) زيادة في طول الفاصلة QT المعدلة (\$0.050 + 0.518 ثانية). وقد لوحظ كلا من الحاجة للتنفس الصناعي والوفاة في المرضى الذين أظهروا زيادة في طول الفاصلة QT المعدلة وليس في المرضى الذين أظهروا طولاً طبيعيا للفاصلة QTc) المعدلة. تبين وجود علاقة ذات دلالة إحصائية بين طول الفاصلة QTc المعدلة بطريقة التسمم وطريقة تناول السم، شدة التسمم ومستوى إنزيم الكولين استيراز، وعدد أمبولات toxogonine التي احتاجها المرضى في العلاج والحاجة إلى تركيب أنبوب تنفسي والتنفس الصناعى.

طول الفاصلة QTc المعدلة يعد وسيلة سريرية عملية سهلة التطبيق لتقييم شدة أعراض التسمم بمركبات الفوسفات العضوية، خاصة عند المرضى المتسممين بالتناول عن طريق الفم، وحالات الانتحار، والزيادة في طول الفاصلة QT المعدلة، حيث يعد هذا مؤشراً لخطورة حالة المريض واحتياجه للتنفس الصناعي.