World Applied Sciences Journal 16 (8): 1118-1122, 2012
ISSN 1818-4952

Electrocardiographical Manifestations of Acute Organophosphate Poisoning

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Abstract: This study evaluated the electrocardiographical manifestations of acute organophosphate poisoning & conducted at Liaquat University Hospital Hyderabad from September 2010 to February 2011. All patients above 12 years of age, of either gender with history of OP poisoning and evidence of exposure to OP compounds within previous 24 hours with characteristic manifestation of organophosphate poisoning were further evaluated for their cardiac status thru electrocardiography (ECG) to observe the electrocardiographical changes. Total 112 patients with OP poisoning presented to the emergency department, of which the cardiac manifestation was found in eighty seven (78%) patients. The ages of the patients ranged from 12 to 65 years, the overall mean age identified was 27.35±8.63 (SD) while the mean age in the male and female population was 25.67±7.21 (SD) and 24.85±6.53. The mean duration of poisoning identified was 10.09±6.28 (SD). Of 87 subjects with cardiac manifestations, sixty (69%) were males and twenty seven (31%) were females. Fifty five (63%) were unmarried while thirty two (37%) were married. Regarding mode of poisoning accidental, suicidal and homicidal was observed in 41(47.1%), 44(50.6%) and 02(2.3%) $p < 0.05$. The electrocardiographical manifestation observed in patients were bradycardia 13 (14.9%), tachycardia 11(12.6%), ST – elevation 09(10.3%), T–inversion 10(11.5%), prolonged P-R interval 07(8.0%), atrial fibrillation 05(5.7%), prolonged Q-T interval 15(17.2%), ventricular tachycardia 07(8.0%) and mix finding 10(11.5%). The organophosphate poisoning is associated with cardiac complications and most of them occur during the first few hours after exposure.

Key words: Organophosphate · Poisoning · Pesticide · Insecticide · Electrocradiography

INTRODUCTION

Acute organophosphate poisoning represents a major health problem in developing countries where organophosphate compounds are widely and easily available. Organophosphate insecticides inhibit both cholinesterase and pseudocholinesterase enzymatic activity and leads to cholinergic signs and symptoms [1]. It is estimated that there are 34,000 suicides annually in the Middle East region and 20% of suicides in the Middle East region are the result of pesticide ingestion [1]. In Iran, organophosphate poisoning (OPP) is a major health problem [2-4] and OPs are the main cause of pesticide poisoning and pesticide-related deaths in Tehran [5-6]. The mortality rate is between 2–30% despite appropriate treatment; therefore, the prediction of mortality is important to allow proper resource allocation and therapeutic aggressiveness [7]. Although the plasma or urine OP concentrations, cholinesterase (CE) activity and serum ß-glucuronidase level correlates with severity and mortality rates [8, 9]. These laboratory methods are not readily available and their usefulness remains controversial. The absence of specific laboratory methods indicates the need for a clinically based prognostic system [10].

The cardiac manifestations occur in a majority of affected patients and may range from innocuous electrocardiographic manifestations, such as sinus tachycardia, to life-threatening complications including cardiogenic pulmonary oedema [11]. Repolarisation abnormalities, including ST segment elevation and T wave inversion as well as prolongation of the QTc interval, are among the most frequent cardiac manifestations of acute organophosphate poisoning [11]. The mechanisms of organophosphate-induced cardiac toxicity are not fully understood. Aside from direct toxic effects of the organophosphate compounds, an increase in sympathetic and/or parasympathetic activity, hypoxaemia, acidosis and electrolyte abnormalities are thought to be involved in myocardial damage associated with organophosphate

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poisoning. Notably, experimental studies in isolated rat hearts suggested that obidoxime, a cholinesterase reactivator that is used as an antidote in cases of organophosphate poisoning, may even aggravate organophosphate-induced prolongation of the QT interval. The reported prevalence of various electrocardiographical changes in organophosphorous compound is 89.1% [11] Because of the limited former literature many health care professions may not be fully aware of the electrocardiographical changes of OP poisoning. Therefore the present study was conducted at Liaquat University Hospital Hyderabad and is specific to intoxication cases due to OP poisoning. The objective of the study was to determine the various electrocardiographical manifestations of acute organophosphate poisoning in patients presented at Liaquat University Hospital Hyderabad, Sindh, Pakistan. Early reorganization of abnormal rhythm will save the patients to acquire life threatening arrhythmias.

MATERIAL AND METHODS

This descriptive case series study was conducted in the department of Medicine at Liaquat University Hospital from September 2010 to February 2011. All patients above 12 years of age, of either gender with history of OP poisoning and evidence of exposure to OP compounds within the previous 24 hours with characteristic manifestation of organophosphate poisoning including salivation, lacrimation, urination, diarrhea, GI upset, emesis, diaphoresis, miosis, bradycardia, tachycardia, bronchospasm, bronchorrhea, emesis and muscle fasciculations, cramping, weakness, restlessness, confusion, ataxia, tremors, seizures and coma, came through outdoor patient department (OPD), casualty outdoor department (COD) or admitted in medical unit were evaluated and enrolled in the study. The detailed history of all such patients was taken and complete physical and relevant clinical examination was performed. All the routine and specific investigation including cardiac enzymes and troponin (when required) was performed. The informed consent was taken from every patient or from attendants of patients after full explanation of procedure regarding the study and all such maneuvers was performed under medical ethics and through the cooperation of whole research team. The diagnosis was based on the definite history of OP poisoning and evaluation of clinical features. All such patients were further evaluated for their cardiac status thru electrocardiography (ECG) to observe the electrocardiographical changes. For recruitment of the subjects the non - probability purposive technique was used while the exclusion criteria of the study were; (1). The patients with history of cardiac diseases, (2). The patients already on anticholinergic therapy. (3). Non cooperative patients who refused to participate in the study.

The data was collected on pre-designed proforma and analyzed in SPSS version 10.00. The frequency and percentage was calculated for gender distribution. The mean ±SD was computed for quantitative variables like age & duration of poisoning. The stratification was done on age, gender and electrocardiographical findings. The independent - samples t-test was applied between categorical variables, chi-square was applied to determine the statistical difference in gender and the p-value ≤ 0.005 was considered as statistically significant. The mentioned statistical tests were applied at 95% confidence interval (CI).

RESULTS

During six months 112 patients with OP poisoning presented to the emergency department, of which the cardiac manifestation was found in 87 (78%) patients. The ages of the patients ranged from 12 to 65 years, the overall mean age identified was 27.35±8.63 (SD) while the mean age in the male and female population was 25.67±7.21 (SD) and 24.85±6.53. The mean duration of poisoning identified was 10.09±6.28 (SD). Of 87 with cardiac manifestations sixty (69%) were males and twenty seven (31%) were females. Fifty five (63%) were unmarried while thirty two (37%) were married. Regarding clinical manifestation, salivation in eighty (92%), lacrimation in 75(86%), urination in forty eight (55%), diarrhea in forty five (52%), GI upset in sixty two (71%), emesis in eighty (92%), diaphoresis in sixty (69%), miosis in seventy eight (90%), bronchospasm in thirty two (37%), bronchorrhea in thirty five (40%), muscle fasciculations in sixty two (71%), cramping and weakness in sixty five (75%), restlessness in seventy (80%), confusion in seventy two (83%), ataxia in thirty (34%), tremors in forty (46%), seizures in thirty seven (43%) and coma in fifty seven (66%) patients. The mean amount of atropine used on day one was 20.5mg while the mean amount of atropine used in the total treatment of the patients was 80.8 mg. The duration of treatment with atropine was 3.5 days. The cardiac and electrocardiographical abnormalities all returned to normal before the patients were discharged although five patients died despite received the adequate doses of atropine and

Table 1: Gender distribution in relation to mode of poisoning

	Gender			
Mode	Male	Female	Total	P-value
Accidental	31(51.7%)	10(37%)	41(47.1%)	0.05*
Suicidal	29(48.3%)	15(55.6%)	44(50.6%)	
Homicidal	00(0%)	02 (7.4%)	02(2.3%)	
Total	60(100%)	27(100%)	87(100%)	

*P value is statistically significant

X^2 value = 5.48; df = 2

Table 2: Distribution of patients by gender and compound ingested

	Gender			
Compound	Male	Female	Total	P-value
Metacid (Methyl-parathion)	29(48.3%)	06 (22.2%)	35(40.2%)	0.23*
Baygon spray (Propoxur 1%, Sichlorvos 0.5%, Cytluthrine 0.04%)	10(16.7%)	04(14.8%)	14(16.1%)	
Monocrotophos	05(8.3%)	04 (14.8%)	09(10.3%)	
Malathion	05(8.3%)	03(11.1%)	08(9.2%)	
Unknown	06(10.0%)	06(22.2%)	12(13.8%)	
Total	60(100%)	27(100%)	87(100%)	

*P value is statistically non significant

X^2 value = 6.88; df = 5

Table 3: Electrocardiographical manifestations of acute organophosphate poisoning

	Gender			
E.C.G findings	Male	Female	Total	P-value
Bradycardia	10(16.7%)	03(11.1%)	13(14.9%)	0.04*
Tachycardia	08(13.3%)	03(11.1%)	11(12.6%)	
ST - elevation	07(11.7%)	02 (7.4%)	09(10.3%)	
T - inversion	07(11.7%)	03(11.1%)	10(11.5%)	
Prolonged P- R interval	05(8.3%)	02(7.4%)	07(8.0%)	
Atrial fibrillation	03(5.0%)	02(7.4%)	05(5.7%)	
Prolonged Q -T interval	10(16.7%)	05(18.5%)	15(17.2%)	
Ventricular tachycardia	05(8.3%)	02(7.4%)	07(8.0%)	
More than one finding	05(8.3%)	05(18.5%)	10(11.5%)	
Total	60(100%)	27(100%)	87(100%)	

*P value is statistically significant

X^2 value = 5.21; df = 2

pralidoxime as well. The cause of death was cardiac arrhythmia (ventricular fibrillation) in four patient and non-cardiogenic pulmonary oedema in one patient. Regarding rate, the bradyarrhythmia and tachyarrhythmia was identified in 32 (37%) and 23 (26%) patients [p=1.27] respectively. Of eighty seven (80%) patients were from rural area where as the 17(20%) patients were from urban area of the province. The gender distribution in relation to mode of poisoning and compound ingested is shown in Table: 01-02 while the electrographical manifestation of patients with acute organophosphate poisoning is shown in Table 03.

DISCUSSION

The mechanism by which organophosphates and carbamates induce cardiotoxicity is still uncertain. Ludomirsky [12] described three phases of cardiac toxicity after organophosphate poisoning: phase 1, a brief period of increased sympathetic tone; phase 2, a prolonged period of parasympathetic activity; and phase 3, in which Q-T prolongation followed by torsade de pointes ventricular tachycardia and then ventricular fibrillation occur. In our study 112 patients presented with the history of OPP poisoning, of which 87 developed cardiac manifestations. The mean age of our population is a 27.35±8.63 (SD) year where as the mean age in the study published in 2009 was 32.2±14.9 (SD) years [13]. In present study the male population is predominant while a study on electrocardiographic findings of acute organophosphate poisoning published in Journal of emergency medicine, the female population was predominant [13]. The electrocardiographic presentation detected in our series was prolonged Q-T interval, bradycardia, tachycardia, T – inversion, atrial fibrillation, prolonged P- R interval, ST – elevation, ventricular tachycardia and combined arrhythmia. The electrographic picture of our study is consistent with the study by Karki that also identified similar arrhythmia with different proportions [11]. In a series of 168 cases of organophosphate poisoning reported by Kiss and Fazekas, [14] five had a transient picture of myocardial infarction. Diffuse myocardial damage was found during necropsy in two cases of malathion poisoning (an old generation organophosphate) and diffuse myocarditis has been reported after carbamate poisoning. Some investigators have reported a relatively high incidence (43%) of polymorphous ventricular tachycardias of the torsades de pointes type in patients with organophosphate-related prolongation of the QT interval [12]. However, in a recent case series of 37 patients with organophosphate poisoning, of which 14 patients presented with prolongation of the QT interval, only one (7%) developed ventricular tachycardia [11]. Thus, given these conflicting findings, the occurrence of malignant ventricular arrhythmias cannot be predicted in the individual case.

In our study the patients with arrhythmia (two with atrial fibrillation and one with ventricular tachycardia) had severe anoxia and pulmonary edema on admission during the early cholinergic phase of the poisoning. Three of the 5 patients with atrial fibrillation had hypokalaemia and two had transient elevation of the ST segment and raised cardiac enzymes. These findings suggest that the cardiac toxicity associated with organophosphate and carbamate poisoning is caused by more than one mechanism. Possible mechanisms include sympathetic and parasympathetic overactivity, hypoxaemia, acidosis, electrolyte derangements and a direct toxic effect of the compounds on the myocardium. The bradycardia is thought to dominate in the early cholonergic phase of the poisoning and it is more frequently observed in our study whereas the hypertension and sinus tachycardia may be seen in organophosphate and carbamate poisoning are due to nicotinic effects.

The administration of atropine in high doses has been implicated in the development of ventricular arrhythmias [14]. In our, study there was no such correlation. Lyzhnikov [15] also found no correlation between atropine therapy and ventricular arrhythmias in organophosphate poisoning. In present study no chronic sequelae was noted while regarding the clinical presentation the findings are consistent with the former literature published in the Singapore Medical Journal [11]. We believe that the type of poisonous agent (organophosphate versus carbamate), the severity of the poisoning, the stage at which treatment is started and the presence or absence of intensive care facilities are the main determinant factors for the hospital mortality and similar observations have also been made by Saadeh. [16] Therefore, appropriate and prolonged rhythm monitoring must be provided during the "vulnerable" phase of cardiac arrhythmias.

CONCLUSION

The organophosphate poisoning is associated with cardiac complications and most of them occur during the first few hours after exposure. Hypoxemia and electrolyte derangements are major predisposing factors for the development of these complications. Intensive supportive treatment, meticulous respiratory care and administration of atropine in adequate doses very early in the course of the illness are the keys to successful management of these cases.

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