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Original Research Article

Organophosphorus Poisoning Leading to Myocardial Injury – A Cross Sectional Study in Rural Area

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Abstract

Organophosphorus compounds are organic compounds containing phosphorus. They are used primarily in pest control as an alternative to chlorinated hydrocarbons that persist in the environment. Some organophosphorus compounds are highly effective insecticides, although some are extremely toxic to man, including sarin and VX nerve agents. To study the myocardial involvement in organophosphorus compound poisoning. Patients got admitted to the department of medicine, Adichunchanagiri institute of medical sciences, with history of organophosphorus compound poisoning during the period of January 2016 to December 2016. In the preset study, the incidence of OP compound poisoning common in males compared to females. Majority of patients were in the age group 31-49 years. The most common clinical finding in patients was tachycardia (36%) followed by bradycardia (32%). Hypertension was seen in 5 (20%) patients and 4 (16%) showed hypotension. Most common ECG finding was ST elevation (48%), Sinus tachycardia (24%) and QT prolongation (16%) and sinus bradycardia (12%). 15 (60%) of the patient had a significant levels of serum Acetylcholinesterase level >2500 IU/L. Cardiovascular effects are quite common following acute OP poisoning. These effects pertain to different muscarinic and nicotinic effects on the heart, electrolyte disturbances, ABG disorders, respiratory failure and over-atropinization.

Keywords: organophosphorus compounds, Cardiovascular effects, bradycardia, ECG finding, Acetylcholinesterase.

INTRODUCTION

Organophosphorus chemistry is the corresponding science of the properties and reactivity of organophosphorus compounds. Phosphorus, like nitrogen, is in group 15 of the periodic table, and thus phosphorus compounds and nitrogen compounds have many similar properties. The definition of organophosphorus compounds is variable, which can lead to confusion. In industrial and environmental chemistry, an organophosphorus compound need contain only an organic substituent, but need not have a direct phosphorus-carbon (P-C) bond. Thus a large proportion of pesticides (e.g., malathion), are often included in this class of compounds.

Phosphorus can adopt a variety of oxidation states, and it is general to classify organophosphorus compounds based on their being derivatives of phosphorus (V) vs phosphorus (III), which are the predominant classes of compounds. In a descriptive but only intermittently used nomenclature, phosphorus compounds are identified by their coordination number σand their valency λ. In this system, a phosphine is a $\sigma^3\lambda^3$ compound [3-5].

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 [3]. 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 [3]. 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 poisoning. The reported prevalence of various electrocardiaographical changes in organophosphorus compound is 89.1% [6].

Both sympathetic and parasympathetic overactivity have been shown to cause myocardial damage [7]. As early as 1974, Yasue *et al.,* [8] postulated that parasympathetic overactivity plays a major role in coronary artery spasm, and later Horio *et al.,* [9] induced coronary artery spasm in adult humans with healthy coronary arteries after intracoronary injection of acetylcholine. In a series of 168 cases of organophosphate poisoning reported by Kiss and Fazekas [10], five had a transient picture of myocardial infarction. Diffuse myocardial damage was found at necropsy in two cases of malathion poisoning (an old generation organophosphate) [11].

OBJECTIVE

To study the myocardial involvement in organophosphorus compound poisoning.

METHODOLOGY

Patients got admitted to the department of medicine, Adichunchanagiri institute of medical sciences, with history of organophosphorus compound poisoning during the period of January 2016 to December 2016.

Sample size: 25

Inclusion Criteria: All symptomatic patients having ingested organophosphorus compound.

Exclusion Criteria

- Patients who are known to have pre-existing heart disease
- Patients with unclear history of poison consumption

RESULTS

Table-1: Age and Sex wise distribution of cases

Age group (years)	Male		Female		Total	
	No.	%	No.	%	No.	%
31-40	**8**	32	**4**	16	**12**	**48**
41-50	**1**	4	**3**	12	**4**	**16**
>50	**5**	20	**4**	16	**9**	**36**
Total	**14**	56	**11**	44	**25**	**100**

The incidence of OP compound poisoning common in males compared to females. Majority of patients were in the age group 31-49 years.

Table-2: Distribution of patients according to the symptoms

Symptoms	Number of cases	Percentage
Tachycardia	**9**	**36**
Bradycardia	**7**	**32**
Hypertension	**5**	**20**
Hypotension	**4**	**16**

The most common clinical finding in patients was tachycardia (36%) followed by bradycardia (32%). Hypertension was seen in 5 (20%) patients and 4 (16%) showed hypotension.

Table-3: Distribution of patients according to their ECG changes

Characteristics	Number of cases	Percentage
ST Elevation	**12**	**48**
Sinus tachycardia	**6**	**24**
QT Prolongation	**4**	**16**
Sinus bradycardia	**3**	**12**

Most common ECG finding was ST elevation (48%), Sinus tachycardia (24%) and QT prolongation (16%) and sinus bradycardia (12%).

Table-4: Distribution of patients according to their acetylcholinesterase levels

Acetylcholinesterase levels (IU/L)	Number of cases	Percentage
< 2500	**10**	**40**
>2500	**15**	**60**

15 (60%) of the patient had a significant levels of serum Acetylcholinesterase level >2500 IU/L.

DISCUSSION

In the preset study, the incidence of OP compound poisoning common in males compared to females. Majority of patients were in the age group 31-49 years. The most common clinical finding in patients was tachycardia (36%) followed by bradycardia (32%). Hypertension was seen in 5 (20%) patients and 4 (16%) showed hypotension. Most common ECG finding was ST elevation (48%), Sinus tachycardia (24%) and QT prolongation (16%) and sinus bradycardia (12%). 15 (60%) of the patient had a significant levels of serum Acetylcholinesterase level >2500 IU/L.

The ECG reflects the widespread cardiac toxicity of organophosphate compounds. Ludromirsky *et al*., had described three phases of cardiotoxicity after organophosphate compound poisoning. Phase 1 – brief period of increased sympathetic tone; phase 2- prolonged period of parasympathetic activity; phase 3 – Q- T prolongation followed by torsade de pointes, ventricular tachycardia and the ventricular fibrillation. Both sympathetic and parasympathetic overactivity are known to cause cardiotoxicity.

Both sympathetic and parasympathetic over activity have been shown to cause myocardial damage. As early as 1974, Yasue *et al*., postulated that parasympathetic over activity plays a major role in coronary artery spasm, and later Horio et al. induced coronary artery spasm in adult humans with healthy coronary arteries after intracoronary injection of acetylcholine. In a series of 168 cases of organophosphate poisoning reported by Kiss and Fazekas, five had a transient picture of myocardial infarction. Diffuse myocardial damage was found at necropsy in two cases of malathion poisoning (an old generation organophosphate) [9, 10].

In the study done by Balouch *et al*., and Sadeesh *et al*., Q-T prolongation was the most common ECG abnormality, as compared ST elevation being the most common finding in this study. Q-T prolongation was seen only in 24% of the patients in this study as compared to 67% in study by Sadeesh *et al*., but it was comparable with the study done by Balouch *et al*., ST segment elevation was seen 32 % of the patients, which was similar to the study by Sadeesh *et al*., (24%). The ECG changes like Atrial fibrillation, prolonged P-R interval and ventricular tachycardia found in the study by Sadeesh *et al*., [12, 13].

CP Dalvi *et al*., studied the correlation of electrocardiographic changes in organophophorus poisoning with its prognosis. Abnormal ST-T changes and progressive fall in voltage and or low voltage were the commonest ECG changes encountered. These occurred significantly more often in patients with moderate or severe poisoning ($p< 0.001$). The 17 patients (5 moderate, 12 severe) with a combination of these ECG abnormalities required higher doses of atropine (mean 30 mg) and, in the 12 who survived, the ECG took longer (mean 5.5 days) to normalize (despite normal clinical recovery rate) as compared to other cases. All fatal cases in the study had both these ECG changes.

WANG Jian-dong *et al*., studied the dynamic changes of cardiac enzymes and the acute poisoning with organophophorus poisoning in Department of Emergency, Sichuan Provicial People's Hospital. Fasting serum level of troponin T and cardiac enzymes (CK-MB, CK, AST, and LDH) in 92 patients with acute organophophorus poisoning (AOPP) were measured after poisoning 1,2,3,5 and 7 days, and were measured one time in normal control group as well. There was an increase of different levels in troponin T and cardiac enzymes along with the degree of AOPP. They concluded that the level of cardiac troponin T and cardiac enzymes in patients with AOPP may be as useful markers of degree of poisoning and prognosis [14].

CONCLUSION

Cardiovascular effects are quite common following acute OP poisoning. These effects pertain to different muscarinic and nicotinic effects on the heart, electrolyte disturbances, ABG disorders, respiratory failure and over-atropinization. Prompt diagnosis, early supportive and definitive therapies with atropine and oximes along with vigilant monitoring of the patients for life-threatening cardiac effects such as QT prolongation, VT or VF during hospital stay can definitely save the lives of the victims.

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