

DIFFERENT PATTERNS OF ECG IN ORGANOPHOSPHATE POISONING AND EFFECT ON MORTALITY

Aisha Javed¹, Firasat Waqar², Shaista Ahmed³, Attiya Sabeen Rahman⁴,
Qaiser Jamal⁵, Tahir Hussain⁶

¹⁻⁶ Karachi Medical and Dental College,
Karachi, Pakistan

Address for Correspondence:

Aisha Javed,

Karachi Medical and Dental College,
Karachi, Pakistan

E-Mail: draishaahmed@hotmail.com

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Contribution

AJ conceived the idea. FW, QJ helped in acquisition of data and did statistical analysis. SA, TH drafting the manuscript. ASR did critical review. All authors contributed significantly to the submitted manuscript.

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ABSTRACT

Objective: To determine different patterns of ECG changes in Organophosphate Poisoning and mortality related to it.

Methodology: We conducted an observational cross sectional study from 1st June 2015 to 31st Dec 2015 in the department of Medicine, Abbasi Shaheed Hospital, Karachi. Patients agreeing for informed consent following inclusion and exclusion criteria were consecutively included. All patients were above 13 years of age, of either gender with history of exposure to organophosphate compounds within previous 24 hours with characteristic manifestation of organophosphate poisoning. They were evaluated for cardiac status through electrocardiography (ECG) to observe the changes like heart rate, PR interval, ST-T changes, PVCs and QTc interval. The data was analyzed with SPSS 17.

Results: Out of 79 patients 44(55.6%) were females. The mean age of the sample was 24.41 ± 10.1 years. About 59 (74.68%) patients intended to commit suicide, whereas 20 (25.3%) patients were exposed to organophosphate due to accident. ECG was abnormal in 34 (43.03%) patients. Sinus bradycardia was present in 10 (29.41%) patients followed by sinus tachycardia which was present in 8 (23.52%) patients. Prolonged QTc was present in 6 (17.64%) and ST-T changes and PVCs were present in 4 (11.76%) patients. First degree block was least observed ECG finding which was present in 2 (5.88%) patients. Respiratory depression was found in 7 (8.86%) patients in which 6 were having prolonged QTc and only 1 had PVC. All these patients were put on ventilator but only 4 survived while the rest of the 3 (30.79%) died having prolonged QTc.

Conclusion: ECG changes are frequently observed in patients with organophosphate poisoning. Prolonged QTc interval is associated with respiratory depression and increased risk of mortality.

Key Words: Organophosphate Poisoning, ECG Changes, Prolonged QTc Interval, Premature Ventricular Contraction

INTRODUCTION

Worldwide, organophosphate compounds are the most widely used insecticides.¹ Recent analysis done by WHO reveals that in developing countries an estimated 3 million per year people are being poisoned with pesticides with eventual mortality of more than 3 lac per year.² In developing countries like Pakistan, agriculture is the main occupation and insecticides are easily available everywhere and often stored in an improper manner due to lack of awareness of their hazards.¹ Organophosphate compounds are a diverse group of chemicals used in both domestic and industrial settings.³ Organophosphate poisoning is one of the most common causes of poisoning in the third world with high morbidity and mortality.⁴ In Pakistan the prevalence of depression is high and suicidal tendencies are increasing.^{5,6} Suicidal attempts with pesticide intake is common in our society and in other developing countries, as it is readily available in every home.⁷⁻⁹

Organophosphorus pesticides exert their acute effects by inhibiting acetyl cholinesterase in the nervous system with subsequent accumulation of toxic levels of acetylcholine resulting in over stimulation of muscarinic and nicotinic receptors which results in wide spread clinical symptoms like bradycardia, hypotension, increased salivation, blurred vision and confusion etc.¹⁰ Overall, mortality studies report mortality rates between 3-25 %.¹¹ Worldwide an estimated 3 million people are exposed to organophosphate or carbamate agents each year, with up to 300,000 fatalities.^{12,13} Unintentional poisoning kills far fewer people but is a problem in places where highly toxic organophosphorus pesticides are available.¹⁴ The fatality rate following deliberate ingestion of Organophosphate poison in developing countries in Asia is approximately 20% and may reach 70% during certain seasons and at rural hospitals.² Mortality rates depend upon the type of compound used, amount ingested, general health of the patient, and delay in identification and medical assistance, insufficient respiratory management and inability to identify unusual complications.¹⁵

Cardiac complications often accompany poisoning with these compounds, particularly during the first few hours.¹⁶ Cardiac manifestations may range from innocuous ECG manifestation such as sinus tachycardia to life threatening complications including cardiogenic pulmonary edema. Repolarization abnormalities including ST segment elevation and T wave inversion as well as prolongation of QTc interval are amongst the most frequent cardiac manifestation of acute organophosphate poisoning. Aside from direct toxic effects of the OP compounds an increase in the sympathetic and/or parasympathetic activity, hypoxemia, acidosis, and electrolyte derangements are major predisposing factors to myocardial damage.^{17,18} Acute myocardial infarction as a result of acute OPP

(Organophosphorus Poisoning) though have been reported but are exceptionally rare.¹⁹ Death due to cardiac causes in OP poisoning occurs either due to arrhythmias or severe and refractory hypotension.^{13,20,21}

Although we come across a large number of patients with this poisoning now and then in our clinical practice, there is paucity of local data regarding the electrocardiographic changes in organophosphate poisoning. This study is therefore designed to determine the frequency of ECG changes in organophosphate poisoning in order to create better understanding and to reduce mortality and hospital stay by its early detection and management.

METHODOLOGY

This observational cross sectional study was conducted from 1st Jun 2015 to 31st Dec 2015 in the Department of Medicine, Abbasi Shaheed Hospital, and Karachi. All patients above 13 years of age, of either gender with history of organophosphate poisoning and evidence of exposure to Organophosphate Poisoning compounds within 24 hours with characteristic manifestation of organophosphate poisoning were included in the study. While patients with history of cardiac disease, anti-cholinergic therapy were excluded. They were further evaluated for cardiac status through electrocardiography (ECG) to observe the ECG changes like heart rate, PR interval, ST-T changes, PVCs and QTc interval. The data was analyzed with SPSS 17.

RESULT

Out of 79 patients with Organophosphate poisoning cardiac manifestations were found in 34 (43.03%) patients. Among them, 59(74.68%) intended to commit suicide, 20(25.32%) were exposed to organophosphate due to accidental event and none came with homicidal attempt. All patients were exposed through gastrointestinal route and none were exposed through inhalation or by cutaneous route. Mean age of presentation was 24.41 ± 10 years. The youngest was 15 years old whereas the oldest was 50 years old as shown in

Figure1:Age Distribution of Patients with Organophosphate Poisoning

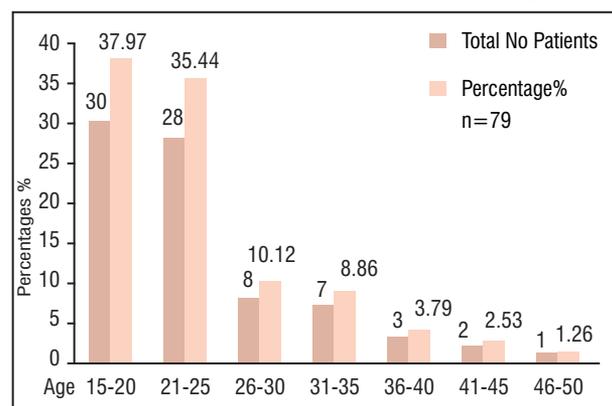


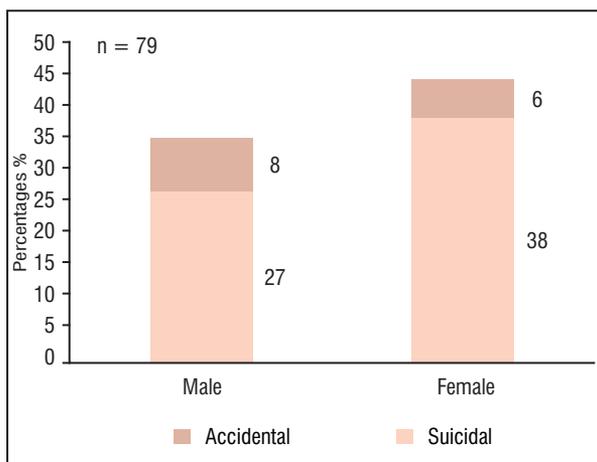
Figure 2: Gender Distribution Along with Mode of Exposure of Organophosphate Poisoning in Each Gender

figure 1. The gender distribution showed that 44 (55.6%) were females. Female to male ratio was 1.25:1. Among 44 females, 31 (70.45%) had intended suicidal attempt while 13(29.54%) had given statement of accidental intake of organophosphate poisoning. Among males 27 (77.14%) had intended suicidal attempt while 8(22.85%) had given history of accidental intake of OP poisoning as shown in Figure 2.

ECG was done in all patients who were admitted with history of organophosphate poisoning before starting medication. ECG was abnormal in 34(43.03%) patients. Sinus bradycardia was most frequently observed ECG finding which was present in 10 (29.41%) patients. Sinus tachycardia followed by sinus bradycardia was present in 8 (23.52%) patients. Prolonged QTc was present in 6 (17.64%) patients among which 4 were females. ST-T changes and PVCs were present in 4 (11.76%) patients. First degree AV block was least observed ECG finding which was present in 2 (5.88%) patients as shown in Table 1.

Patients with ECG changes were admitted in Intensive Care Unit and were closely monitored.

All 6 patients with prolonged QTc suffered from respiratory depression. This makes percentage of 100. One patient with PVC also suffered from respiratory depression. Of 7 patients with respiratory depression who were put on ventilator, 3 expired (30.79%) and all of them had prolonged QTc interval.

DISCUSSION

Worldwide, cardiac toxicity associated with organophosphate and carbamate poisoning is caused by more than one mechanism and is still unknown. Possible mechanisms include sympathetic and parasympathetic over activity, hypoxemia, acidosis, electrolyte derangements, and a direct toxic effect of the compounds on the myocardium. Ludomirsky et al described three phases of cardiac toxicity

Table 1: ECG Abnormalities in Patients with Organophosphate Poisoning

	Male n	Female n	Total n	Percentage (%)
Sinus Bradycardia	4	6	10	29.41%
Sinus Tachycardia	5	3	8	23.52%
Prolonged QTc	2	4	6	17.64%
ST-T changes	3	1	4	11.76%
1st Degree AV block	-	2	2	5.88%
PVCs	1	3	4	11.76%

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.²²

In our study, 79 patients presented with history of organophosphate poisoning. Among them 59(74.68%) intended to commit suicide whereas 20 (23.32%) were exposed to Organophosphate compounds due to accidental event and none came with homicidal attempt. This predominantly suicidal intake of Organophosphate compounds observed in our study was consistent with a study conducted by Suleman MI et al on the analysis of organophosphate cases who reported suicidal attempts in 77.62% while accidental attempts were only 22.37%.²³ Similar statistics were revealed in a study conducted by Farooqui AN et al who enrolled 50 patients of Organophosphate compounds poisoning and got 74.68% cases with suicidal intent while remaining 25.3% were with accidental exposure.⁶

As far as the gender distribution is concerned, our study highlighted that suicidal intake was greater among males (77.14%) in comparison to females (70.45%). This is in contradiction with study of Suleman MI et al. which showed female preponderance of suicidal organophosphate intake with a percentage of 56.75%.²³

A number of electrocardiographic abnormalities were detected in our conducted study among which sinus bradycardia was the most frequently observed with percentage of 29.41% while the study conducted by Saadeh AM and Yurumez Y depicted that most common Ecg abnormality was prolonged QTc interval with a percentage of 67% and 55.5% respectively.^{24,25} On the contrary, sinus bradycardia was observed in lesser frequency in the study conducted by Saadeh AM and Agarwal S et al.^{24,26} Karki P et al

found that electrocardiographic abnormalities including sinus tachycardia occurred in 40.5 %, prolonged QTc interval in 37.8 %, ST-T changes in 29.7percent%, sinus bradycardia in (18.9 %), and conduction defects were least observed ECG abnormality, a similar finding reported by Saadeh AM et al , found in 5.4 percent%.^{16,24}

In a study conducted by Chuang FR et al. at Taiwan they found that there were higher rate of respiratory depression and mortality in patients of Organophosphate poisoning whose ECG were showing prolonged QTc interval. This observation is consistent with our study.

CONCLUSION

Our study showed significant ECG abnormalities in patients with organophosphate poisoning and nearly 100% patients with respiratory depression showed prolonged QTc interval, serves to emphasize the importance of close monitoring by doing serial ECG and observing for respiratory depression . It also highlights the need for timely shifting of patients in ICU with prolonged QTc so that necessary management and intervention can be done in order to reduce the mortality and morbidity.

REFERENCES

- Eddleston M. Patterns and problems of deliberate self-poisoning in the developing world. *QJM* 2000;93(11):715-31.
- Lakhair MA, Shaikh MA, Kumar S, Zafarullah, Bano R, Maheshwari BK. Frequency of various clinical and electrocardiac manifestation in patients with acute organophosphorous compound (OPC) poisoning. *J Liaquat Uni Med Health Sci* 2012;11(1):34-8.
- Jones AL, Karalliedde L. Poisoning. In: Boon NA, College NR, Walker BR, editors. *Davidson's principles and practice of medicine*. 20th ed. New Delhi: Churchill Livingstone; 2006. p. 216.
- Rahimi R, Nifkar S, Abdollahi M. Increased morbidity and mortality in acute human organophosphate poisoned patients treated by oximes. *Human Expo Toxicol* 2006;25(3):157-62.
- Abbas S, Riaz MN, Akram S. Organophosphate poisoning. Emergency management in intensive care unit. *Prof Med J* 2003;10:308-14.
- Farooqui AN, Tariq S, Asad F, Abid F, Tariq O. Epidemiological profile of suicidal poisoning at Abbasi Shaheed Hospital. *Ann Abbasi Shaheed Hosp Karachi Med Dent Coll* 2004;9(1):502-5.
- Chishti P, Stone DH, Corcoran P, Williamson E, Petridou E. Suicide mortality in European Union. *Eur J Public Health* 2003;13(2):108-14.
- Nadesan K. Pattern of suicide: a review of autopsies conducted at the University Hospital, Kuala Lumpur. *Malays J Pathol* 1999;21(2):95-9.
- Ahmed Z, Ahmed A, Mubeen SM. An Audit of suicide in Karachi from 1995-2001. *Ann Abbasi Shaheed Hosp Karachi Med Dent Coll* 2003;8:424-8.
- Lee WC, Yang CC, Deng JF, Wu ML, Ger J, Lin HC, et al. The clinical significance of hyperamylasemia in organophosphate poisoning. *J Toxicol Clin Toxicol* 1998;36(7):673-81.
- Boota M, Shehzad S. Insecticide poisoning, management strategy in a hospital. *Prof Med J* 2004;11:95-7.
- Eddleston M, Phillips MR. Self-poisoning with pesticides. *BMJ* 2004;328:42-4.
- Eyer P. The role of oximes in the management of organophosphorus pesticide poisoning. *Toxicol Rev* 2003;22:165.
- Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. *Lancet* 2008;371(9612):597-607.
- Sahin I, Onbasi K, Sahin H, Karakaya C, Ustun Y, Noyan T. The prevalence of pancreatitis in organophosphate poisoning. *Hum Exp Toxicol* 2002;21:175-7.
- Karki P, Ansari JA, Bhandary S, Koirala S. Cardiac and electrocardiographic manifestation of acute organophosphate poisoning. *Singapore Med J* 2004;45(8):385-9.
- Paul UK, Bhattacharyya AK. ECG manifestations in acute organophosphorus poisoning. *J Indian Med Assoc* 2012;110(2):98.
- Balouch GH, Yousfani AH, Jaffery MH, Devrajani BR, Shah SZA, Baloch ZAQ. Electrocardiographical manifestations of acute organophosphate poisoning. *World Appl Sci J* 2012;16(8):1118-22.
- Joshi P, Manoria P, Joseph D, Gandhi Z. Acute myocardial infarction: can it be a complication of acute organophosphorus compound poisoning? *J Postgrad Med* 2013;59:142-4.
- Davies J, Roberts D, Eyer P, Buckley N, Eddleston M. Hypotension in severe dimethoate self-poisoning. *Clin Toxicol (Phila)* 2008;46(9):880-4.
- Peter JV, Sudarsan TI, Moran JL. Clinical features of organophosphate poisoning: a review of different classification systems and approaches. *Indian J Crit Care Med* 2014;18(11):735-45.
- Ludomirsky A, Klein HO, Sarelli P, Becker B, Hoffman S, Taitelman U, et al. Q-T prolongation and polymorphous

- (torsade de pointes) ventricular arrhythmias associated with organophosphorus insecticide poisoning. *Am J Cardiol* 1982;49(7):1654-8.
23. Suleman MI, Jibran R, Rai M. The analysis of organophosphate poisoning case treated at Bhawal Victoria Hospital, Bahawalpur in 2000-2003. *Pak J Med Sci* 2006;22:244-9.
 24. Saadeh AM, Farsakh NA, al-Ali MK. Cardiac manifestation of acute carbamate and organophosphate poisoning. *Heart* 1997;77(5):461-4.
 25. Yurumez Y, Yavuz Y, Saglam H, Durukan P, Ozkan S, Akdur O, et al. Electrocardiographic findings of acute organophosphate poisoning. *J Emerg Med* 2009;36(1):39-42.
 26. Agarwal SB, Bhatnagar VK, Agarwal A, Agarwal U, Venkaiah K, Nigam SK, et al. Impairment in clinical indices in acute organophosphate insecticide poisoning patients in India. *Internet J Toxicol* 2007;4:1.