The Study of Electrocardiographic Findings in Patients with Organophosphate Poisoning

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ABSTRACT

Background: Cardiac manifestations that occur in a majority of patients with organophosphate (OP) poisoning may range from innocuous electrocardiographic manifestations, such as sinus tachycardia, to life-threatening complications, including cardiogenic pulmonary edema and myocardial necrosis. In this study, we evaluated the various electrocardiographic manifestations in patients with OP poisoning.

Methods: This retrospective-descriptive study was performed by reviewing the medical records from all patients poisoned with organophosphate admitted to Razi Educational Hospital, Rasht, Iran, from April 2008 to March 2011. Patients with incomplete records were excluded from the study. Histories of all patients were collected and ECG analysis was conducted including the rate, rhythm, ST-T abnormalities, conduction defects, and measurement of PR and QT intervals by a cardiologist. Descriptive statistical analysis was conducted by SPSS software version18.

Results: Of the total 100 patients (75 were male) with OP poisoning that referred to the Emergency Ward of Razi Hospital, 63 patients presented ECG abnormalities. The mean age of the patients was 35.78 ± 12.91 years. The causes of poisoning were occupational in 71 patients, suicidal in 26 patients, and accidental in 3 patients. Sinus tachycardia (31%) was the most common ECG abnormality, followed by non-specific ST-T changes (24%). Overall, mortality rate was 5% and all of the deceased patients presented changes in ECG.

Conclusion: OP poisoning is associated with significant ECG abnormalities, especially tachycardia and non-specific ST-T changes.

Keywords: ECG Abnormalities, Organophosphate Poisoning, Rasht.

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INTRODUCTION

Organophosphates (OP) compounds are used as insecticides in agricultural and domestic settings throughout the world (1). This ease of availability of the organophosphate insecticides has resulted in a gradual increase in occupational, accidental, and suicidal poisoning mainly in developing countries (2). Accidental poisoning can occur after exposure through skin or inhalation and serious poisoning often follows suicidal ingestion (3).

According to the World Health Organization (WHO), one million serious accidental and two million suicidal poisonings with organophosphate occur worldwide every year, and of these, approximately 200,000 died, mostly in developing countries (4). In Iran, OP poisoning is a major health problem and is the main cause of insecticide poisoning

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and insecticides-related deaths. In Tehran, mortality rate was between 2–30% despite appropriate treatment (5).

OP compounds are phosphoric or phosphonic acid derivatives that act as irreversible cholinesterase inhibitors, causing a syndrome of cholinergic excess involving muscarinic, nicotinic, and central nervous system receptors (6,7). The most common presenting signs of OP poisoning include constricted pupils, hypersalivation, abdominal pain, depressed level of consciousness, muscle fasciculation, etc. The most fatality results from respiratory failure (8).

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, such as cardiogenic pulmonary edema (3,8).

The mechanism by which OP compounds induce cardiotoxicity has not been elucidated thus far, and it is difficult to pinpoint one mechanism as being the cause of cardiac toxicity related to organophosphate (9,10). Sympathetic and parasympathetic over-activity, hypoxemia, acidosis, electrolyte derangements, and a direct toxic effect of the compounds on the myocardium and vascular system are thought to be involved in myocardial damage associated with OP poisoning (3,11).

The electrocardiogram (ECG) in patients with OP poisoning may display a variety of abnormalities, such as sinus tachycardia or bradycardia, atrioventricular block, and ST segment and T-wave abnormalities, but extreme QT interval prolongation and ventricular tachydysrhythmia of the torsades de pointes type are not common (12,13).

Since early reorganization of abnormal rhythm in organophosphate poisoning protects the patients against acquiring life-threatening arrhythmias (5), this study was done to determine the electrocardiographic findings in patients with organophosphate poisoning at Razi Educational Hospital, Rasht, Iran.

MATERIALS AND METHODS

This retrospective-descriptive study was performed by reviewing the medical records of all patients with organophosphate poisoning admitted to the Emergency Ward of Razi Hospital, Rasht, from April 2008 to March 2011.

All patients of both genders with history of OP poisoning or evidence of exposure to OP compounds within the previous 24 hours with characteristic manifestations and improvement of the signs and symptoms of OP poisoning after administration of atropine were enrolled in this study. Patients with history of atrioventricular arrhythmias, congestive cardiac failure, two or three heart blocks, ischemic heart diseases, or preexisting motor/sensory neuropathy were excluded from the study. Overall, 121 patients with OP poisoning were admitted over a period of three years. Eventually, 21 cases were excluded due to a past history significant for cardiac disease and lack of the registration of medical records.

Age, sex, cause of poisoning, time elapsed between exposure and admission to the hospital, treatment duration, need for assisted ventilation, and outcome in terms of morbidity and mortality were recorded from the registration medical records. Manifestations of poisoning in patients was categorized to mild poisoning (including anorexia, weakness, restlessness, headache, obtundation, miosis, tremors), moderate poisoning (including emesis, increased salivation, increased respiratory secretions, diarrhea, GI upset, diaphoresis, muscle fasciculation, cramping), and sever poisoning (pinpoint miosis, pulmonary edema, cyanosis, defecation, urination, confusion, ataxia, seizures, cardiac arrest and coma). During their hospital stay, electrocardiography (ECG) was carried out once daily on all patients in the general medical ward. ECG analysis was conducted for rate rhythm, ST/T

abnormalities, conduction defects and measurement of P-R interval, R-R interval, Q-T interval, and QTc interval. QT interval was corrected according to the formula applied by Bazett (14). QTc was considered prolonged when it was longer than 0.41 second in men and longer than 0.42 second in women and QRS duration was measured by a cardiologist. All this information was collected for each patient and then descriptive statistical analysis was conducted by SPSS version 18. Values were presented as frequency and mean ± standard deviation.

RESULTS

In this study, 100 patients with OP poisoning were studied and 75 of the patients were men. The mean age of the patients was 35.78 ± 12.91 years. The majority (39%) of the patients were in the 31 to 40 age group (Figure 1). The causes of poisoning were occupational in 71 patients, suicidal intentions in 26 patients, and accidental in 3 patients. The mean time interval of OP exposure and admission to the hospital were 6.54 ± 5.3 hours; 69 patients within 6 hours, 18 patients between 6 and 12 hours, and 13 patients more than 12 hours presented to hospital after poisoning. Overall, 51 patients with mild poisoning, 38 patients with moderate poisoning, and 17 patients with severe poisoning were hospitalized. Of these, 7 patients needed assisted ventilation in treatment duration. The mean duration of treatment was 2.42 ± 5.45 days.

Changes in ECG records were observed in 63 patients. Electrocardiographic manifestations of OP poisoning are summarized in Table 1. Sinus tachycardia (HR > 100 bpm) was the most common ECG abnormality that was seen in 31 patients (49.2%), followed by non-specific changes in ST segment and T wave was found in 24 patients (38.9%). Three patients had prolonged QTc interval (0.41 s in men and 0.42 s in women).

Overall, mortality rate was 5%, due to respiratory failure, and all of the dead patients had changes in ECG recording.

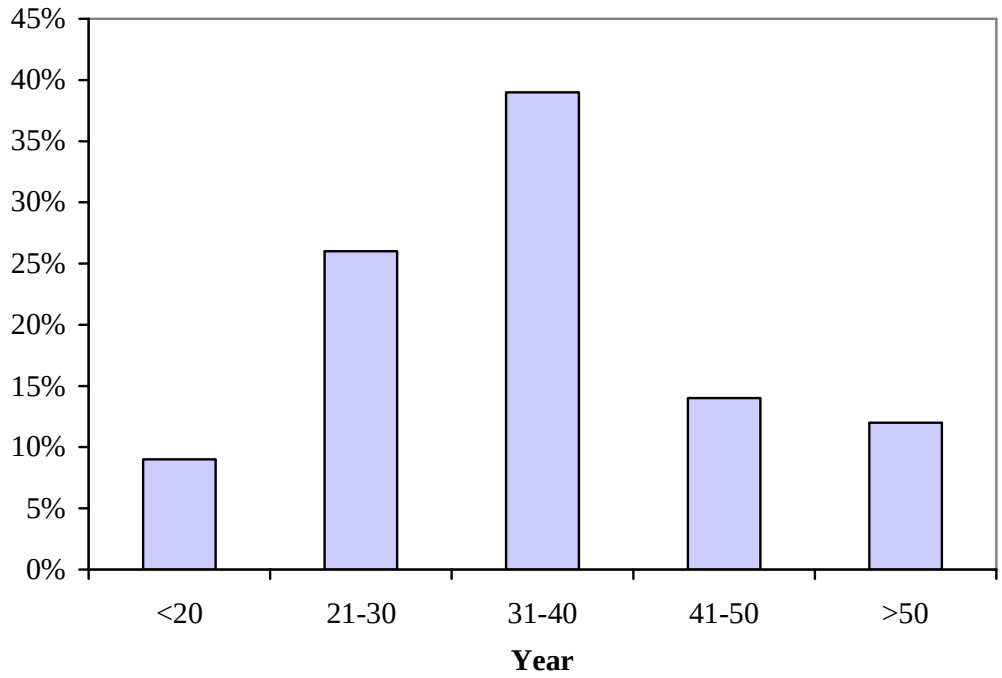

Figure 1. Age group distribution in patients with organophosphates poisoning

Table1. Electrocardiographic findings of organophosphates poisoning

ECG Abnormalities	Male n (%)	Female n (%)	Total n (%)
Sinus tachycardia	25 (33.3 %)	6 (24 %)	31(31%)
Sinus bradycardia	6 (8 %)	4 (16 %)	10 (10 %)
Atrioventricular arrhythmia	5 (6.6 %)	1(4 %)	6 (6 %)
Conduction disturbances	15 (20 %)	2 (8 %)	17 (17 %)
Non-specific ST-T changes	18 (24 %)	6 (24%)	24 (24 %)
Prolonged QTc interval	2 (2.6 %)	1(4 %)	3 (3 %)

DISCUSSION

Organophosphate poisoning is always considered a life-threatening condition. The mechanism by which organophosphates induce cardiotoxicity is still uncertain (11,12). Cardiac toxicity after OP compounds poisoning may occur in three phases. Initially, there is a brief period of increased sympathetic activity characterized by tachycardia and hypertension. This is followed by a more prolonged period of extreme cholinergic activity during which bradycardia and hypotension along with ST-T changes and life-threatening rhythm disturbances can occur. A third longer phase is usually associated with a prolonged QT interval and polymorphic ventricular tachycardia that can result in sudden death (9).

In this study, 75 of the patients with OP poisoning were men and the mean age of the patients was 35.78 ± 12.91 years. Furthermore, the main reason for OP exposure was occupational (71%). In a prospective study over a course of twelve months by Rafigh Doost *et al*. in Birjand, of 51 patients (21males) with OP poisoning, 19 patients were in the 15 to 24 age group, and followed by 17 patients were in the 25 to 45 age group. Also, 78.43% of patients had suicidal exposure (15). In another study in Taiwan, of the 4799 patients with OP poisoning, 3117 were male (64.95%), 1657 were female (34.53%), and 25 (0.52%) had no gender reported. The mean age of the patients was 46.28 ±18.32 years and there were 101 patients less than 6 years old (2.10%), 181 patients 6 through 19 years old (3.77%), 4475 people over 19 years old (93.25%), and 42 patients had no age recorded (0.88%). In Jihlin *et al*'s study on 4799 cases of OP exposure, 3106 cases (64.72%) were suicidal, and only 759 cases (15.82%) were occupational (16). In Yurumez *et al*'s study in Turkey, 85 patients (51 female) with OP poisoning with mean age of 32.2 ± 14.9 years were studied that in 85.9% of them, exposure reasons were also suicidal (12). The differences between the findings of these studies and the present study in terms of the causes of exposure , the cause of exposure was mostly occupational in the present study, can be attributed to the fact that the main occupation of people is farming in the region under study, Rasht. Men, especially young age group, are at a greater risk for OP poisoning due to use of these compounds in agricultural settings.

In our study, 63 patients (49 males) had electrocardiographic changes. This finding is similar to finding of Rafigh Doost *et al*'s study which indicated that of the 51 patients with OP poisoning, 33 presented electrocardiographic changes. In another study in Pakistan, of 115 patients with OPP, 85 patients (60 males) presented changes in ECG recording (5). These findings showed that poisoning with organophosphate is associated with significant ECG abnormalities.

Overall, in the present study, the electrocardiographic presentations observed in the cases were bradycardia, tachycardia, atrioventricular arrhythmia, conduction disturbances, prolonged QTc interval, and non-specific changes in ST segment and T wave.

Hypertension and sinus tachycardia, which may be seen in OP poisoning, are nicotinic effects, whereas hypotension and sinus bradycardia are cholinergic manifestations (17). In this study, sinus tachycardia was seen in 31 % of the patients with OP poisoning. Although bradycardia is thought to dominate in the early cholinergic phase of poisoning, sinus tachycardia was a more frequent finding in our study. Others have also made the same observation (18,19). Karki *et al*. reported that sinus tachycardia occurred in 40.5% of patients (3). Yurumez *et al*. reported that sinus tachycardia was a more frequent finding (in 31.8% cases) in their study (13). In another study, Saadeh *et al*. reported that sinus tachycardia was seen in 35.0% of cases (10).

Although non-specific ST-T changes have generally been recognized as not being directly related to any cardiac diseases, they also have been observed before ST elevation associated with coronary spasm (20). Thus, the non-specific ST-T change for the patient with OP poisoning has a considerable clinical meaning (3). In addition, large QT dispersion (longest-shortest QT interval on any of the 12 leads of the ECG) is a result of ischemic change which may conceal the QT prolongation in the affecting vascular area (10). In our study, non-specific ST-T change was seen in 24 patients, prolonged QTc interval was reported in three patients, and conduction disturbances were observed in 13 patients. However, in Yurumez *et al*'s study, 47 patients (55.5%) had a prolonged QTc interval, and 15 patients (17.6%) non-specific ST-T change (13). Similarly, in Rafigh Doost *et al*'s study in Iran, 64.71% of the patients had a prolonged QTc interval (15). Karki *et al*. also reported prolonged QTc interval in 14 cases (37.8%), ST-T changes in 11 cases (29.7%), and conduction defects in two cases (5.4%). Chuang *et al*. determined that 97 (43.5%) patients had QTc prolongation, and these patients had poor prognosis. Moreover, Jang *et al*. determined that 67 of 170 patients had QTc prolongation and in this group, mortality rate, respiratory failure rate, and frequency of ventricular premature contractions were significantly higher than those of patients without QTc prolongation (21). It seems that in our study, QTc prolongation occurred in fewer patients with OP poisoning compared with the other studies.

CONCLUSION

Poisoning with OP compounds can produce significant ECG abnormalities, especially tachycardia and non-specific ST-T changes. Since these abnormalities can cause lethal arrhythmia and cardiac damage, careful observation of the electrocardiogram of the patients exposed to OP compounds is necessary, parallel to the appropriate medical treatment.

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REFERENCES

1. Aardema H, Meertens J, Ligtenberg J, Peters-Polman O, Tulleken J, Zijlstra J. Organophosphorus pesticide poisoning: cases and developments. Neth J Med. 2008;66(4):149-53.
2. Bardin PG, van Eeden SF, Moolman JA, Foden AP, Joubert JR. Organophosphate and carbamate poisoning. Archives of internal medicine. 1994;154(13):1433-4.
3. Karki P, Ansari J, Bhandary S, Koirala S. Cardiac and electrocardiographical manifestations of acute organophosphate poisoning. Singapore medical journal. 2004;45:385-9.

4. Sivagnanam S. Potential therapeutic agents in the management of organophosphorus poisoning. Critical Care-London. 2002;6(3):260-1.
5. Balouch GH, Yousfani AH, Jaffery MH, Devrajani BR, Shah SZA, Baloch ZAQ. Electrocardiographical Manifestations of Acute Organophosphate Poisoning. World Applied Sciences Journal. 2012;16(8):1118-22.
6. Kose A, Gunay N, Kose B, Ocak AR, Erel O, Demiryurek AT. Effects of atropine and pralidoxime pretreatment on serum and cardiac oxidative stress parameters in acute dichlorvos toxicity in rats. Pesticide Biochemistry and Physiology. 2010;97(3):249-55.
7. Emerson GM, Gray NML, Jelinek GA, Mountain D, Mead HJ. Organophosphate poisoning in perth, western australia, 1987–1996. The Journal of emergency medicine. 1999;17(2):273-7.
8. Tafuri J, Roberts J. Organophosphate poisoning. Annals of emergency medicine. 1987;16(2):193-202.
9. Ludomirsky A, Klein HO, Sarelli P, Becker B, Hoffman S, Taitelman U, et al. QT prolongation and polymorphous. The American journal of cardiology. 1982;49(7):1654-8.
10. Saadeh A, Farsakh N, Al-Ali M. Cardiac manifestations of acute carbamate and organophosphate poisoning. Heart. 1997;77(5):461-4.
11. Taira K, Aoyama Y, Kawamata M. Long QT and ST-T change associated with organophosphate exposure by aerial spray. Environmental Toxicology and Pharmacology. 2006;22(1):40-5.
12. Yurumez Y, Yavuz Y, Saglam H, Durukan P, Ozkan S, Akdur O, et al. Electrocardiographic findings of acute organophosphate poisoning. The Journal of emergency medicine. 2009;36(1):39-42.
13. Namba T, Greenfield M, Grob D. Malathion poisoning. A fatal case with cardiac manifestations. Archives of environmental health. 1970;21(4):533-41.
14. Bazett HC. An analysis of the time-relationship of electrocardiograms. Heart. 1920; 7: 353–7.
15. Rafighdoost A, Mirhoseini E, Pourzand H, Rafighdoust H. Epidemiology of organophosphate poisoning, and its cardiac and pulmonary effects. Journal of birjand university of medical science. 2007; 13 (4 (29)):49-54.
16. Lin TJ, Walter FG, Hung DZ, Tsai JL, Hu SC, Chang JS, et al. Epidemiology of organophosphate pesticide poisoning in Taiwan. Clinical Toxicology. 2008;46(9):794-801.
17. Lovejoy FH, Linden C. Acute poison and drug over dosage. In: Harrison's principles of internal medicine. New York: McGraw-Hill; 1991.p.2178-82.
18. Hayes M, Van der Westhuizen N, Gelfand M. Organophosphate poisoning in Rhodesia. S Afr Med J. 1978;54(230):230-4.
19. Bardin P, Van Eeden S, Joubert J. Intensive care management organophosphate. SAMJ. 1987;72:593-7.
20. Brill DM, Maisel AS, Prabhu R. Polymorphic ventricular tachycardia and other complex arrhythmias in organophosphate insecticide poisoning*. Journal of electrocardiology. 1984;17(1):97-102.
21. Jang S, Lin J, Chuang F. Electrocardiographic findings of organophosphate intoxication in emergency department as predictors of prognosis: a retrospective analysis. Changgeng yi xue za zhi/Changgeng ji nian yi yuan= Chang Gung medical journal/Chang Gung Memorial Hospital. 1995;18(2):120-5.