

Electrocardiographic Changes in Acute Organophosphorus Poisoning

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Abstract: **Background:** Cardiac complications are the less common fatal effect of acute organophosphate poisoning. This study was undertaken to analyze Electrocardiographic (ECG) changes in acute organophosphate poisoning cases. **Materials and Methods:** 100 consecutive cases of acute organophosphate poisoning admitted in Silchar Medical College and hospital in one year. All adults more than 18 years of age with history exposure to OPC admitted in the hospital were included. Cases of organophosphate poisonings referred from other hospitals, poisoning with multiple agents, patients with history of previous cardiac diseases and coexisting medical conditions were excluded from the study. Poisoning Severity Score was calculated as per International Programme on Chemical Safety and patients were grouped into 3 grades. **Results:** Out of 100 cases, 58 were males with male: female ratio being 1.38:1. The maximum incidence were observed in the age group of 18–30 year (46%), unmarried (63%). Most of them resulted from suicidal purpose (90%). Muscarinic effects were noticed in 90%, nicotinic effects in 18% and central effects in 14% of cases. As per IPCS poison severity score, 24%, 8% and 68% of patients were in grade I, II and III severity. Majority of the patients with sinus tachycardia (76%), hypertension (92%) and hypotension (83%) were in Grade III severity. Among 28 patients with prolonged corrected QT interval, 86% were in grade III; and, among 26 patients who had inverted T wave, 54% were in grade III. **Conclusion:** Fatal cardiac complications do occur in acute organophosphate poisoning. Higher incidence of ECG changes in Grade III cases suggests that if the cardiac complications develop, the patient should be immediately transferred to an intensive cardiac care unit.

Keywords: Arrhythmias, Cholinergic Agents, Heart Conduction System, Hypotension, Organophosphate Poisoning

1. Introduction

The incidence of pesticide poisoning is increasing in the developing countries where agriculture is the main occupation. The widespread and negligent use, not so stringent legislation and the personal problems have made the pesticides, the most commonly encountered substance in human poisoning. The organophosphates are considered as the king of pesticides, because of their magnitude of use in agriculture. The organophosphate pesticide self-poisoning is estimated to kill around 2, 00,000 people each year, largely in the Asia-Pacific region and the mortality rate varies from 10-20% (1).

The organophosphates are powerful inhibitors of carboxylic esterase enzymes, including acetyl cholinesterase and pseudo cholinesterase. The symptoms of organophosphate poisoning appear in sympathetic and parasympathetic nervous system. The patients develop muscarinic, nicotinic and central nervous system manifestations (2). The literatures state that the most common, or at least the best recognized, cause of death in patients with acute poisoning is asphyxia, pulmonary edema complicating the clinical course in many cases. The macroscopic and histological changes were recognized in the respiratory tract at a time when the heart at autopsy was considered minimally affected. The electrocardiographic changes were noticed in such patients early, but only recently has the possible underlying histological myocardial picture been emphasized (3).

The present analysis was undertaken to study the electrocardiographic (ECG) changes in patients of acute organophosphate poisoning.

2. Materials and Methods

100 consecutive cases of acute organophosphate poisoning admitted in Silchar Medical College and hospital since 1st January 2016. This analysis was carried out to evaluate the pre-interventional cardiac and ECG changes in patients of acute organophosphate poisoning.

Inclusion criteria

All adults more than 18 years of age with history of consumption and/or exposure to OPC admitted in the hospital within 12 hours of the incident.

Exclusion criteria

All patients with poisoning due to compounds other than OPC or with prior history of prior or repeated incidences of consumption of OPC or those with cardiac diseases.

The cardiac changes were studied with respect to blood pressure and heart rate; and ECG changes with respect to rhythm, elevated ST segment, T wave depression and corrected QT interval. These parameters were recorded before administration of atropine, oxime and other therapeutic procedures. The QTc interval is prolonged if it is >450 millisecond in men and >460 millisecond in women (4). The diagnosis of organophosphate poisoning was done based on history and the clinical features. The patients were grouped into grade I, grade II and grade III as per the Poison Severity Score (IPCS PSS) developed by the International Program on Chemical Safety and the European Community, and the European Association of Poisons Centers and Clinical Toxicologists. The highest grade scored in any category is considered as the overall grade.

Table 1: IPCS Poison Severity Score (5)

Grade 1		Grade 2	Grade 3
Respiratory			
Intubated	No	-	Yes
Neurological			
GCS	14-15	13-Sep	8-Mar
Seizures	No	-	Yes
Cardiovascular			
Bradycardia (Pulse)	> 50	41-50	≤ 40
Tachycardia (Pulse)	≤ 140	141-180	> 180
Hypotension (Systolic BP)	> 100	81-100	≤ 80

3. Results

Out of 100 cases, selected for study 58 were males and 42 were females with male: female ratio being 1.38:1. The mean age of the patients was 27.35±8.63 years. In this study, maximum incidence was observed in the age group of 18–30 years, 46 (46%) followed by 33(33%), 10(10%), 6(6%), 4(4%) and 1(1%) in age group 31-40, 41-50, 51-60, 61-70 and 71-80 years respectively. The marital status consisted of 63 (63%) being unmarried and the rest 37 (37%) married. Educational background of 67 (67%) being 10th level to 12th level and the rest 33 (33%) were below 10th level, primary or pre-primary level. History of suicidal attempts, homicidal and accidental exposure were noticed in 90(90%), 3 (3%) and 7 (7%) respectively (Table 1). The respective residential status of 68 (68%) and 32(32%) were from rural and urban background.

Table 2: Gender distribution in relation to mode of poisoning

Mode	Male	Female	Total
Accidental	5 (71.42%)	02 (28.57%)	7 (7%)
Suicidal	37 (41.11%)	53 (58.88%)	90 (90%)
Homicidal	00 (0%)	03 (100%)	03 (03%)
Total	58 (100%)	42 (100%)	100 (100%)

Muscarinic effects were noticed in 90(90%), nicotinic effects in 18 (18%) and central effects in 14 (14%) of cases. In the present study, vomiting was the most common symptom seen in 46 (46%) cases, followed by sweating and salivation in 44(44%) cases. Convulsions, seen in 10(10%) cases, were the rarest of the symptoms. Among 100 patients, 96(96%) had a characteristic odour of OPC. The other common clinical signs were disturbed consciousness in 72(72%), tachypnea in 86(86%), meiosis in 52(52%) and fasciculations in 44(44%) of cases. As per IPCS poison severity score, 24 (24%) patients were in grade I, 8(8%) in grade II and 68(68%) in grade III severity. Sinus tachycardia was present in 90 patients (90%), hypertension in 26 (26%) and hypotension in 24 (24%).

Majority of the patients with sinus tachycardia (76%), hypertension (92%) and hypotension (83%) were in Grade III severity (Table 3). Prolonged corrected QT interval was observed in 28 patients (28%), elevated ST segment in 4 (4%), inverted T wave in 26 (26%) and conduction defects in 2 (2%). Among 28 patients with prolonged corrected QT interval, 24 (86%) were in grade III and 4 (14%) in grade II severity; and, among 26 patients who had inverted T wave, 14 (54%), 8 (31%) and 4 (15%) were in grade III, grade II and grade I severity respectively (Table 4).

Table 3: Distribution of cases according to heart rate (HR) and blood pressure (B.P)

Grade	No. of case	Heart Rate		Blood Pressure		
		Normal	Tachycardia	Normal	Hypertension	Hypotension
I	24	10	14	24	0	0
II	8	0	8	2	2	4
III	68	0	68	24	24	20
Total	100	10	90	50	26	24

Table 4: Distribution of cases according to ECG changes

Grade	No. of case	QTc	ST	T	Ectopic
I	24	0	0	4	0
II	8	4	0	8	0
III	68	24	4	14	2
Total	100	28	4	26	2

4. Discussion

The organophosphates are powerful inhibitors for acetyl cholinesterase enzyme. This enzyme hydrolyses acetylcholine [neurotransmitter at pre and postganglionic parasympathetic synapses, sympathetic preganglionic synapses and at neuromuscular junction] into choline and acetic acid after its release and completion of function. The inhibition of acetyl-cholinesterase results in the accumulation of acetylcholine with continued stimulation of local receptors and eventual paralysis of nerve or muscle. Apart from acetyl cholinesterase, organophosphates exert powerful inhibitory action over other carboxylic ester hydrolases such as chymotrypsin, butyrylcholinesterase,

plasma and hepatic carboxyl esterase, paraoxonases, and other non-specific proteases. The clinical features of acute organophosphate poisoning are due to cholinergic excess and CNS effects (6). The three phases of cardiac toxicity has been described due to organophosphate poisoning:
 Phase 1: Brief period of increased sympathetic tone
 Phase 2: Prolonged period of parasympathetic activity
 Phase 3: QT prolongation followed by torsade de pointes ventricular tachycardia and then ventricular fibrillation (7).

In the present study, mean age of the patients presented with organophosphate poisoning was 27.35±8.63 years. Similar findings were seen in the studies by **Karki P et al (7)** (26.85 years), **shankar laudari et al (8)** (29.8±13.9 years) and **Saadeh AM et al (9)** (23.9±59.2) years. In this study, maximum incidence of organophosphorus poisoning was seen in the age group of 18–30 years (46%). Similar observations found by **Karki P et al (7)** that majority (65%) of the patients were in the 15 to 30 years age group, **Shankar laudari et al (8)** that most of the patients belonged to the population of active productive age group

(86.9% were between 15 and 45 years of age). In a prospective study over a course of twelve months by **Pourzand H et al (10)** in Birjand, of 51 patients (21 males) 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.

In present study, male: female ratio was 1.38: 1 which correlates with studies by **Saadeh AM et al (9)** (1.1:1), **S Agarwal et al (11)** (1.8:1) whereas dissimilar observations were noticed by **G. Someswar et al (12)** (3.17:1), **Gouda H.S et al (13)** (male - 32; female - 18), **Ghulam Hussain Balouch et al (14)** (male - 60; female - 27) and **Rahbar Taromsari et al (15)** (male - 75; female - 25).

In present study, the marital status showed 63% were unmarried. These findings are in agreement with **Karki P et al (7)** and **Ghulam Hussain Balouch et al (14)** where 62% and 63% of cases were unmarried.

In present study, educational background of 67% being 10th level to 12th level and the rest 33% were below 10th level, primary or pre-primary level. Similar observations were seen by **S Agarwal et al (11)** where 70% cases were educated (matriculate and higher secondary level) and remaining illiterate.

In present study, history of suicidal attempts, homicidal and accidental exposure were noticed in 90%, 3% and 7% respectively. **Karki P et al (7)** found in their series of study that most common cause of OP poisoning was suicidal intentions (89% of patients) followed by accidental (11% of patients). Similar findings by **Saadeh A M et al (9)** that the major cause of poisoning was suicidal intention (67%), followed by accidental ingestion (24%); only 9% were due to occupational. **Shankar Laudari et al (8)** study found that the intention of poisoning was suicidal in most of the cases (93.9%), accidental 6(5.2%), Homicidal 1(0.9%). **Ghulam Hussain Balouch et al (14)** study revealed that regarding mode of poisoning accidental, suicidal and homicidal was observed in 41(47.1%), 44(50.6%) and 02(2.3%) respectively. **S Agarwal et al (11)** study noticed that suicidal and intentional poisoning being a major cause as recorded in 80.2% cases followed by occupational (9.1%), accidental (6.6%), homicidal (1.6%) and unknown (2.5%).

In present study, the respective residential status of 68% and 32% were from rural and urban background. Similar findings seen by **Ghulam Hussain Balouch et al (14)** that 80% patients were from rural area where as the 20% patients were from urban area. **G. Someswar et al (12)**, found that he majority of patients (54%) belong to rural areas.

Muscarinic effects were noticed in 90%, nicotinic effects in 18% and central effects in 14% of cases. These findings were in agreement with findings of **Shankar Laudari et al (8)** that the poisoned patients presented with muscarinic effects in 95.6%, nicotinic effects in 23.48% and central effects in 91.3%. **S Agarwal et al (11)** found that the main signs and symptoms reported by the poisoning cases were muscarinic manifestations.

In the present study, vomiting was the most common symptom seen in 46% cases, followed by sweating and salivation in each of 44% cases. Convulsions, seen in 10% cases, were the rarest of the symptoms. Among 100 patients, 96% had a characteristic odour of OPC. The other common clinical signs were disturbed consciousness in 72%, tachypnea in 86%, and meiosis in 52% and fasciculations in 44% of cases. **Ghulam Hussain Balouch et al (14)** found the clinical manifestation as follows as salivation in 92%, lacrimation in 86%, urination in 55%, diarrhoea in 52%, GI upset in 71%, emesis in 92%, diaphoresis in 69%, meiosis in 90%, bronchospasm in 37%, bronchorrhea in 40%, muscle fasciculations in 71%, cramping and weakness in 75%, restlessness in 80%, confusion in 83%, ataxia in 34%, tremors in 46%, seizures in 43% and coma in 66% patients.

Bardin P. G et al (16) revealed that clinical features in the following decreasing order: meiosis (82%), salivation and disturbed level of consciousness (61%), fasciculations (54%), tachycardia (49%), rhonchi or crepitations (48%), tachypnoea (39%), vomiting (38%), sweating (23%), diarrhoea and hypoventilation (21%) and hypotension (20%).

The present study showed cardiovascular abnormalities including sinus tachycardia in 90%, normal heart rate in 10%, normal blood pressure in 50%, hypertension in 26% and hypotension in 24% of cases. In a relatively small series over a longer period of study **Karki P et al (7)** found sinus tachycardia in 40.5 % and sinus bradycardia in 18.9 %, hypertension in 13.5% and hypotension in 10.8%. **Saadeh AM et al (9)** observed that sinus tachycardia in 35% and sinus bradycardia in 28%, hypertension in 22% and hypotension in 17%. **Shankar Laudari et al (8)** revealed that 49.6% of cases developed cardiac effects, the most common abnormality was sinus tachycardia (49.6%). Other abnormalities were sinus bradycardia in 2.6%, and hypertension in 20%, **Ghulam Hussain Balouch et al (14)** observed bradycardia in 14.9%, tachycardia in 12.6% and **Morteza Rahbar Taromsari et al (15)** observed sinus tachycardia as the most common ECG abnormality that was seen in 49.2% and sinus bradycardia 10 % which are comparable to the present series.

In present series, common ECG changes were found as follows as QTc prolongation (28%), T wave changes (26%), ST segment changes (4%) and ectopics in 2% of patients. Similar observations seen by **Karki P et al (7)** as QTc prolongation (37.8%), T wave changes (13.5%), ST segment changes (16.2%) and ectopics in 5.4% of patients. **Vijayakumar S et al (17)** study revealed that the most common ECG finding was QTc prolongation (60%) followed by T wave changes and ST segment changes (40%).

5. Conclusion

The fatal cardiac complications do occur in acute organophosphate poisoning, which are overlooked at times as most common complications expected are respiratory complications. In our region, OP poisoning especially affected to young unmarried females, and most of them resulted from suicidal purpose. The 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. Once the condition is recognised, the patient should immediately be transferred to an intensive or coronary care unit where appropriate monitoring and resuscitative facilities are available. 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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Author Contribution

Study conception and design: Ravikumar. P
Supervision: Agrawal Piyush
Drafting of Manuscript and critical revision: Ravikumar. P
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References

- [1] Thunga G, Sam KG, Khera K, Pandey P, Sagar SV. Evaluation of incidence, clinical characteristics and management in organophosphorus poisoning patients in a tertiary care hospital. *J. Toxicol. Environ. Health Sci* 2010; 2 (5): 73 – 6.
- [2] Reddy KSN. *The Essentials of Forensic Medicine and Toxicology*. 29th ed. Hyderabad: K Sugunadevi, 2010.
- [3] Dalvi CP, Abraham PP, Iyer SS. Correlation of electrocardiographic changes with prognosis in organophosphorus poisoning. *J Postgrad Med*. 1986;32(3):115-9.
- [4] Al-Khatib SM, LaPointe NMA, Kramer JM, Califf RM. What Clinicians Should Know About the QT Interval. *JAMA*. 2003;289(16):2120-7.
- [5] Persson HE, Sjoberg GK, Haines JA, Pronczuk dG. Poisoning severity score. Grading of acute poisoning. *J Toxicol Clin Toxicol*. 1998;36:205–13.
- [6] Pillay VV. *Comprehensive Medical Toxicology*. 2nd ed. Hyderabad: Paras Publications, 2008.
- [7] Karki P, Ansari JA, Bhandary S, Koirala S. Cardiac and electrocardiographical manifestations of acute organophosphate poisoning. *Singapore Med J*. 2004;45(8):385-9.
- [8] Laudari S, Patowary BS, Sharma SK, Dhungel S, Subedi K, Bhattacharya R, Guru-Prasad S, Gangapatnam S. Cardiovascular Effects of Acute Organophosphate Poisoning. *Asia Pacific Journal of Medical Toxicology*. 2014 Jun 20;3(2):64-7.
- [9] Saadeh AM, Farsak NA, A-Ali MK. Cardiac manifestations of acute carbamate and organophosphate poisoning. *Heart*. 1997;77:461-4.
- [10] Pourzand H, Rafigh Doost A. Epidemiology of organophosphate poisoning, and its cardiac and pulmonary effects. *Journal of Birjand University of Medical Sciences*. 2007;13(29):49-55.
- [11] Agarwal SB, Bhatnagar VK, Agarwal A, Agarwal U, Venkaiah K, Nigam SK, Kashyap SK. Impairment in clinical indices in acute organophosphate insecticide poisoning patients in India. *The Internet Journal of Toxicology*. 2007;4(1).
- [12] Someswar G, Reddy Y, Kumari VS, Gupta A, Prabhakar Rao R. Study of Clinical Profile of Organophosphate Compound Poisoning with Special Reference to Electrocardiographic Changes and Electrolyte Derangements. *Indian Journal of Mednobot and Allied Sciences*. 2015;3(1):12-7.
- [13] Gouda HS, Kodali R, Sasanka P, KH MB. Pre-Interventional Cardiac and ECG Changes in Acute Organophosphate Poisoning Cases Admitted to a Tertiary Hospital in India. *International Journal of Medical Toxicology and Forensic Medicine*. 2014 Sep 5;4(4 (Autumn)):130-5.
- [14] Balouch GH, Yousfani AH, Jaffery MH, Devrajani BR, Shah SZ, Baloch ZA. Electrocardiographical manifestations of acute organophosphate poisoning. *World Applied Sciences Journal*. 2012;16(8):1118-22.
- [15] Rahbar Taromsari M, Badsar A, Aghajankhah M, Akbar Poor M, Farhamand Porkar N, Fallah Karkan M. The Study of Electrocardiographic Findings in Patients with Organophosphate Poisoning. *Iranian Journal of Toxicology*. 2013 Jan 15;6(19):751-6.
- [16] Bardin, P. G., S. F. Van Eeden, and J. R. Joubert. "Intensive care management of acute organophosphate poisoning. A 7-year experience in the western Cape." (1987).
- [17] Vijayakumar S, Fareedullah M, Kumar AE, Rao MK. A prospective study on electrocardiographic findings of patients with organophosphorus poisoning. *Cardiovascular Toxicol*. 2011;11(2):113-7.