European Journal of Molecular & Clinical Medicine (EJMCM)
ISSN: 2515-8260 Volume 07, Issue 11, 2020

Original research article

A study of ECG manifestations in patients of acute organophosphate poisoning

Dr. Kallem Venkata Reddy[1], Dr. Yelma Bhooma Reddy[2]

[1]Assistant Professor, Department of General Medicine, Rajiv Gandhi Institute of Medical Sciences [RIMS], Adilabad.

[2]Assistant Professor, Department of ENT, Rajiv Gandhi Institute of Medical Sciences [RIMS], Adilabad.

Corresponding Author: Dr. Yelma Bhooma Reddy

Abstract

Introduction: Poisoning with Organophosphate compounds is very common in rural and tribal areas of India. Cardiac toxicity is one of the important manifestations of acute organophosphate poisoning. We in the current study tried to evaluate the ECG manifestations in patients diagnosed with acute organophosphate poisoning reporting to our Institute. **Methods**: This cross-sectional study was conducted on n=80 adult patients with a history of organophosphate poisoning. All the investigations were performed before initiation treatment the included investigations were estimation of pseudocholinesterase levels, serum electrolytes, and Standard 12 lead ECG. The ECG was recorded before the administration of atropine for treatment. The analysis of ECG was done to determine the rate, rhythm, QRS axis, ST-T changes, measurement of PR intervals, conduction defects if any, measurement of QT interval in all leads, and the longest QT was used for calculation of rate corrected QT or QTc.

Results: A total of n=66 males and n=14 females were included in the study. Sinus tachycardia was in n=15(18.75%) cases and sinus bradycardia was in n=12(15%) of cases. The rhythm abnormalities (arrhythmia) were found in n=2 cases and normal sinus rhythm was in n=78 cases. Conduction defects in form of increased PR interval were found in n =3 males and ST-T changes were recorded among them QTc prolongation was observed in n=21(26.25%) cases. A total of n=10(12.5%) cases expired during the study out of which n=7 cases expired cases had prolonged QTc.

Conclusion: Cardiac complications are more frequently associated with the severity of organophosphate poisoning. Monitoring of ECG changes will be useful in the assessment of prognosis. Patients with prolongation of QTc should be intensively monitored chances of ventricular tachycardia, ventricular fibrillation and mortality are highest among these cases.

Keywords: *Acute Organophosphate poisoning, ECG changes, cardiac complications, QTc prolongation*

Introduction

Acute organophosphate poisoning is a common challenge in Hospitals especially in developing countries where organophosphate compounds are easily available. The organophosphate compounds which are used as insecticides in-home and agriculture inhibit enzymes cholinesterase and pseudo cholinesterase leading to cholinergic signs and symptoms.

[1] Poisoning may occur as a result of exposure or inhalation accidentally and serious poisoning often occurs following suicidal ingestion. [2] WHO analysis in developing countries reveals an estimated about 3 million cases per year of people being poisoned with pesticides and ending in mortality at the rate of 3 lakhs per year. [3] Organ phosphorus compounds (OPC) were first developed by Schrader during world war II. The compounds were first used as insecticides in agriculture and later used in weapons for chemical warfare as nerve gas. [2, 4] The organophosphorus compounds exert their actions by inhibiting acetylcholinesterase in the nervous system with an accumulation of high levels of unopposed actions of acetylcholine resulting in overstimulation of muscarinic and nicotinic receptors. [5, 6] It results in widespread clinical symptoms such as bradycardia, hypotension, increased salivation, blurred vision, and confusion. The cardiac complications of OP poisoning can be serious and often fatal. The development of these complications potentially preventable if they are recognized early and treated adequately.

The ECG manifestations range from normal ECG, bradycardia, sinus tachycardia to lifethreatening cardiogenic pulmonary edema. There may be abnormalities related to repolarization which include ST-segment elevation and T wave inversion and prolongation of QTc interval. Apart from these abnormalities, the OP compounds can activate the autonomic nervous system leading to an imbalance between sympathetic and parasympathetic activity, hypoxemia, acidosis, and electrolyte changes which may also contribute indirectly to myocardial damage. [7] Acute Myocardial infarction following OP poisoning has been reported however, it is a rare occurrence. Most of the causes of death due to cardiac issues in OP poisoning are related to arrhythmias or severe refractory hypotension.

Material and Methods

This cross-sectional study was conducted on n=80 adult patients with a history of organophosphate poisoning admitted to the Medical wards of Rajiv Gandhi Institute of Medical Sciences, Adilabad. The protocol for ethical approval for human research was followed with permission obtained from the Institutional Ethical committee. Written consent was obtained from the patients/accompanying person. A complete detailed history was recorded which included type of insecticide agent (if available) duration between exposure and hospitalization, history of patients, family history, and a thorough clinical examination was done. The duration of hospitalization and outcome of the treatment were recorded. The clinical features of organophosphate poisoning included increased lacrimation, urination, diarrhea, GI upset, emesis, miosis, bradycardia or tachycardia, bronchospasm, muscle twitching, ataxia, tremors, cramps, weakness coma.

Inclusion criteria

1. Patients admitted with signs and symptoms of op poisoning
2. All cases of age > 20 years.
3. Males and females
4. Admitted within 24 hours of op poisoning

Exclusion criteria

1. History of cardiac diseases
2. On anticholinergic therapy
3. Patients are initially treated at other hospitals before being referred to our institute.

4. Cases with the doubtful diagnosis were not included.

All the investigations were performed before initiation treatment the included investigations were estimation of pseudocholinesterase levels, serum electrolytes, and Standard 12 lead ECG. The ECG was recorded before the administration of atropine for treatment. Repeat
ECG was recorded if deemed necessary during the stay at the hospital and ECG was recorded before the discharge of the patients from the Hospital. The analysis of ECG was done to determine the rate, rhythm, QRS axis, ST-T changes, measurement of PR intervals, conduction defects if any, measurement of QT interval in all leads, and the longest QT was used for calculation of rate corrected QT or QTc. The QTc was determined using the Bazette formula. All the patients were managed with gastric lavage.

Intravenous atropine 2-4mg bolus which was repeated every 5 – 15 minutes to complete atropinization. The process of atropinization was maintained for 24 – 48 hours with intermittent doses every 20 – 30 minutes depending on the patient's response. All patients were administered Pralidoxime chloride 2g IV bolus over 15 – 20 minutes after admission and following requirements the repeat doses were administered. Airway management with endotracheal intubation and mechanical ventilation was done in patients showing signs of respiratory failure. Counseling of the patients and relatives was done. All the data was recorded in an MS Excel spreadsheet and analyzed using SPSS version 19 for descriptive statics.

Results

During the study period, a total of n=130 cases were admitted in medical wards with organophosphate poisoning. After application of inclusion and exclusion criteria, n=80 cases were included in the study. The age range was from 20 – 70 years. The mean age was 39.5 ± 2.5 years. Out of n=80 cases n=66(82.5%) were males and n=14(17.5%) were females. The common age group of patients in males was 31 – 40 years with n=26(32.5%) cases, followed by N=15(18.75%) cases in 41 – 50 years of age. N=12(15%) patients were from 21 – 30 years age group n=11(13.75%) cases from 51 – 60 years and n=2(2.5%) cases from 61 – 70 years age group. The male and female distribution of cases in each age group is shown in Figure 1.

The mode of exposure of organophosphorus compounds was through the GI route none was exposed by the cutaneous route. Among the n= 66 males, n=58 (87.87%) were suicidal attempts while n=8 had stated that the intake was accidental. In females out of n=16 cases n=10(62.5%) were suicidal attempts and n=6(37.5%) cases were accidental exposure. The mean duration of poisoning was 9.05 ± 2.25 hours common clinical manifestations were vomiting, blurring of vision, increased salivation, and sweating (table 1). Similarly, the smell of poison, tachypnea, altered consciousness were common signs recorded in the cases depicted in table 2.

Among the ECG changes studied the rate was found to be normal in n=53(66.25%) and sinus tachycardia was in n=15(18.75%) cases and sinus bradycardia was in n=12(15%) of cases. The rhythm abnormalities (arrhythmia) were found in n=2 cases and normal sinus rhythm was in n=78 cases. Conduction defects in form of increased PR interval were found in n=3 males and ST-T changes were recorded among them QTc prolongation was observed in n=21(26.25%) cases (Table 3). A total of n=10(12.5%) cases expired during the study out of which n=7 cases expired cases had prolonged QTc. In the normal QTc cases, n=3 cases died the comparison between the mortality rates between normal QTc and prolonged QTc showed (p=0.05) statistically significant. The QTc prolonged cases who survived showed the ECG changes reverted to normal at the time of discharge.

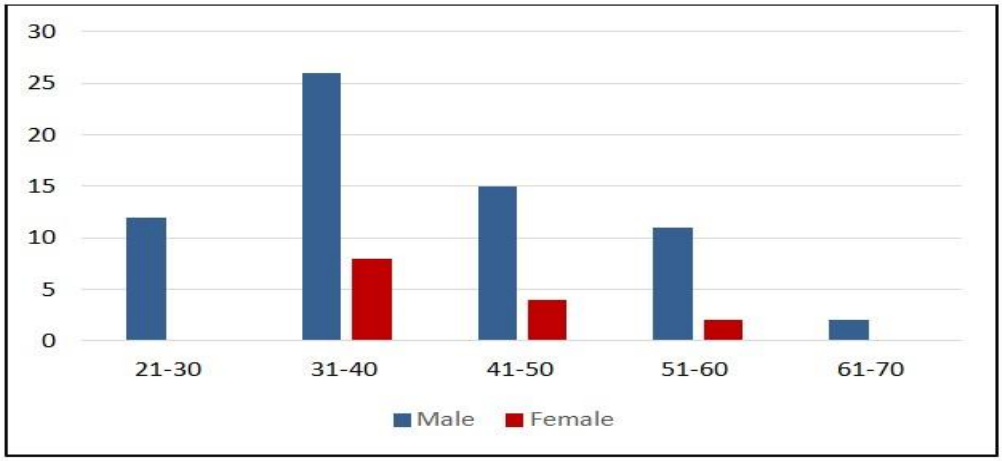

Figure 1: Age-wise and sex-wise distribution of cases

Table 1: Frequency of Symptoms recorded in the cases of the study

Symptom	*Male*	*Females*	*Total*	*Percentage*
Salivation	28	5	33	41.25
Lacrimation	26	9	35	43.75
Sweating	24	8	32	40.00
Blurring of Vision	27	11	38	47.50
Vomiting	40	8	48	60.00
Diarrhea	39	7	46	57.5
Breathlessness	29	6	25	31.25
Urinary Incontinence	12	3	15	18.75
Muscle Twitching	5	3	8	10.00
Weakness	3	3	6	3.75
Convulsions	3	1	4	5.00

Table 2: Frequency of signs recorded in the cases of the study

Signs	*Male*	*female*	*total*	*Percentage*
Smell of poison	60	12	72	90.00
Miosis	35	8	43	53.75
Altered consciousness	42	7	49	61.25
Tachypnoea	58	10	68	85.00
Tachycardia	18	3	21	26.25
Bradycardia	14	2	16	20.00
Fasciculations	24	5	29	36.25

Table 3: Frequency of ECG changes found in the cases of study

ECG changes	*Male*	*Female*	*Total*	*Percentage*
Elevated ST segment	2	0	2	2.50
Inverted T waves	6	1	7	8.75
T wave flattening	5	2	7	8.75
Prolonged QTc	18	3	21	26.25

Prolonged PR interval	3	0	3	3.75
Sinus tachycardia	13	2	15	18.75
Sinus bradycardia	11	1	12	15.00
Normal rhythm	42	11	53	66.25

Discussion

Cardiac complications due to organophosphate poisoning are generally found within the first few hours. The exact mechanisms of cardiac toxicity associated with these compounds are still unknown. The cardiac complications may range from innocuous ECG manifestation to life-threatening cardiogenic pulmonary edema. Ludomirsky A et al; [8] have described three phases of cardiac toxicity due to organophosphate poisoning. Phase 1 is a brief period of increased sympathetic tone. Phase 2 is a prolonged period of increased parasympathetic activity and phase 3 is the period where there is Q-T prolongation followed by the development of torsade de pointes type of ventricular tachycardia and ventricular fibrillation. [8] In our study out of n=80 cases with organophosphate poisoning N=68(85%) cases had consumed intending to commit suicide and 15% were accidental exposures no case of homicide attempt was recorded. Similar observations were found by Suleman MI et al; [9] have reported 77.62% of the consumption of organophosphate compounds were suicidal attempts and 22.37% were accidental exposures. Farooqui AN et al; [10] in their study of n=50 cases found 74.68% cases of organophosphate consumption for suicide intentions and 25.3% were accidental consumptions. The slightly higher percentage of suicidal consumption of organophosphate in our study may be because of the location of our institute in the remote predominantly tribal area of Adilabad. The socio-economic problems of these people are greater as compared to the other population as a result intention to commit suicide is greater in this type of population. In the current study, the age range was from 20 – 70 years. The mean age was 39.5 ± 2.5 years. Out of n=80 cases n=66(82.5%) were males and n=14(17.5%) were females. A study by Basvaraj GM et al; [11] found a predominance of male cases in their study. However, Yurumez, Y. et al; [12] and found a predominance of female cases in their study. Among the ECG abnormalities found in our study most common was QTc prolongation in 26.25% of cases this is in concordance with the findings of Yurumez Y et al; [12] and Saadeh AM et al; [13] in their studies found the most common ECG abnormality in organophosphate poisoning was a prolongation of QTc. Similarly, we found the ST-T changes in 16.25% of cases of our study. Karki P et al; [14] found ST-T changes in 29.7% of cases, sinus tachycardia in 40.5% of cases we in this study found 18.75% cases. Most of the cases of death were related to QTc prolongation with respiratory depression. Chuang et al; in their study showed similar findings where mortality was highest in cases of prolonged QTc and respiratory depression. This study found that the severity of poisoning and the stage at which treatment is started are important determinants of the outcome of treatment similar observations have been made by other studies in this field. [13, 15]

Conclusion

Cardiac complications are more frequently associated with the severity of organophosphate poisoning. Monitoring of ECG changes will be useful in the assessment of prognosis. Patients with prolongation of QTc should be intensively monitored chances of ventricular tachycardia, ventricular fibrillation and mortality are highest among these cases. Other factors affecting treatment outcomes are the duration between intake of poison and presentation to hospital and intensive supportive treatment and respiratory care and administration of atropine in adequate doses will prevent poor outcomes.

References

1. Gunnell D, M. Eddleston, M.R. Phillips, F. Konradsen. The global distribution of fatal
2. pesticide self-poisoning: a systematic review. BMC Public Health 2007; 7:357.
3. Dutoit PW, Maller FO, Ventonder WM, Ungerer MJ. Experience with intensive care management of organophosphate insecticide poisoning. S Afr Med J 1981; 60:227-29.
4. Lakhair MA, Shaikh MA, Kumar S, Zafarullah, Bano R, Maheshwari BK. Frequency of various clinical and electro cardiac manifestations in patients with acute organophosphorus compound (OPC) poisoning. J Liaquat Uni Med Health Sci 2012;11(1):34-8.
5. Wyckoff DW, Davies JE, Barquet A, Davis JH. Diagnostic and therapeutic problems of parathion poisoning. Ann Intern Med 1968; 68:875-82.
6. Sikary AK. Homicidal poisoning in India: A short review. J Forensic Leg Med. 2019; 61:13-16.
7. Jokanović M. Neurotoxic effects of organophosphorus pesticides and possible association with neurodegenerative diseases in man: A review. Toxicology. 2018; 410:125-131.
8. Peter G Blain. Organophosphate poisoning (acute). BMJ Clin Evid 2011; 05:2102.
9. Ludomirsky A, H Klein, P Sarelli, B Becker, S Hoffman, U Taitelman. Q-T prolongation and polymorphous (torsade de pointes) ventricular arrhythmias associated with organophosphorus insecticide poisoning. Am. J. Cardiol. 1982; 49:1654-58.
10. 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; 22:244-49.
11. 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):502-05.
12. Basavaraj G. Mangshetty, Niraj Saraf, Nagender Sharma. Study of Clinical Profile of Organophosphate Poisoning with Special Reference to Electrocardiographic Changes and Electrolyte Derangements. Journal of Evolution of Medical and Dental Sciences 2014; 3(67): 14514-23.
13. Yurumez Y, Y Yavuz, H Saglam, P Durukan, S Ozkan, O Akdur, M Yucel. Electrocardiographic findings of acute organophosphate poisoning. J Emerg Med. 2009; 36(1): 39-42.
14. Saadeh AM, NA Farsakh, MK Al-Ali. Cardiac manifestations of acute carbamate and organophosphate poisoning. Heart 1997; 77:461-64.
15. Karki P, Hansdak SG, Bhandari S, Shukla A, Koirala S. A clinicoepidemiological study of organophosphorus poisoning at a rural-based teaching hospital of eastern Nepal. Trop Doct 2001; 31:32-34.
16. Balouch GH, Yousfani AH, Jaffery MH, Devrajani BR, Shah SZA, Baloch ZAQ. Electrocardiographic manifestations of acute organophosphate poisoning. World Appl Sci J 2012;16(8):1118-22.