International Journal of Multidisciplinary and Current Research ISSN: 2321-3124
Research Article Available at: http://ijmcr.com

Electrocardiographic Changes in Patients of Organophosphorus Compound Poisoning

Farhhan Uddin[#*], Dr Naushaba Rizwan[^], Dr Kiran Waheed[Ψ] and Dr Kashif Zakria

[#]Assistant professor, Department of Physiology, Mohammad Medical College Mirpur Khas, Pakistan
[^]Associate professor, department of Gynae & Obs, Liaquat University Hospital Hyderabad, Pakistan
[Ψ]Post graduate student Liaquat university hospital Hyderabad, Pakistan

Received 02 July 2020, Accepted 01 Sept 2020, Available online 02 Sept 2020, **Vol.8 (Sept/Oct 2020 issue)**

Abstract

Background: The increase demand of crops has increased the use of chemicals in cultivation especially organophosphorus compounds. These chemicals have increased the incidence of poisoning among the persons who are handling these compounds. These incidences are usually accidental, suicidal or homicidal.
Objective of study: To study the ECG changes in Organophosphorus poisoning
Material &Method: It was a retrospective observational type of study. It was conducted in the department of casualty Liaquat university hospital Hyderabad. The study was conducted from September 2019 to December 2019. One hundred patients participated in this study. They were divided in two groups. In both control group and test group 50 patients were included
Result: In the exposed group there were 14(28%) patients suffering from tachycardia, 7(14%) from bradycardia, 2(4%) from QT prolongation, 2(4%) from ST elevation, 2(4%) from extra systole, 2(4%) from T wave inversion, 4(4%) heart block, 3(6%) from MI, 14(28%) did not show any ECG changes. In the non-exposed group there were no ECG changes or cardiac effects were seen. The P value was 0.000.
Conclusion: It was concluded that Organophosphorus compounds increase the risk of myocardial damage which is reflected by ECG changes.

Keywords: *Organophosphorus compounds, ECG changes, tachycardia, bradycardia, ST segment elevation*

Introduction

As the population of world increased there was a call to enhance the production of crops to meet the challenges. This led to invention of organophosphorus compounds but that also opened the door for killing of peoples who were exposed to these deadly compounds [1]. Organophosphorus compounds are commonly used in agricultural lands as insecticide. As an agricultural compound it causes more death in Pakistan, India, Bangladesh and china than any other country in the world. In China approximately 175,000 deaths were recorded [2].

The rising incidence of poisoning is because this compound is easily available and is used by uneducated peoples who are not aware of its side effects [3].

Organophosphorus compound poisoning is one of the leading causes of suicidal deaths, accounting about 200,000 deaths per year [4]. Most of the morbidity and mortality occurs because of improper storage facilities and mishandling during spray on the crops [5].

*Corresponding author's ORCID ID: 0000-0002-8228-382X
DOI: https://doi.org/10.14741/ijmcr.v.8.5.2

Op poisoning is the leading cause of both accidental and suicidal death. An estimated one million accidental death and two million suicidal deaths recorded. Most of the countries involved are developing countries [6]. In Tehran and Middle East OP poisoning is one of the major causes of suicidal death. There are 34, 000 deaths in Iran annually [7]. About two million peoples in Asian pacific region suffer from death due to Organophosphorus poisoning [8]

It was widely assumed that most of deaths are because of respiratory failure but now latest research throughout the world has raised the suspicion of cardiac involvement in early deaths [9]. Cardiac involvement is a major complication of OP poisoning. The Organophosphorus compound either directly or indirectly damages the myocardium. Op compound may leads to MI, Arrhythmias and cardiac arrest. Most of cardiac effects are due to direct toxic effects to myocardium [4].

Op poisoning has cardiovascular effects including tachycardia, hypertension and rhythm irregularities [10]. The ECG changes found in patients were ST segment elevation and T wave inversion [11]. Aterial fibrillation, myocardial infarction and rhythm irregularities are the

main manifestation of cardiac involvement in op poisoning. Early diagnosis is mandatory in preventing cardiac complications [12].

The Organophosphorus compounds block the activity of Acetyl cholinesterase enzyme leading to the blocking the parasympathetic and sympathetic transmission throughout the body including striated muscles and diaphragm leading to respiratory failure. The patients usually come with history of shortness of breath, dizziness, frothing from mouth and excessive micturation. On clinical examination fasciculation, pinpoint pupil and ECG changes are noted [13]. Inhibition of cholinesterase leads to an increased level of acetylcholine leading to clinical manifeststations. These include, vomiting, meiosis, bronchospasm, increased salivation and lacrimation. Other features include fasiculations, increased urination and diarrhea [14].

It is a hypothesis that endothelium derived relaxing factor cause damaging effects on the endothelium and causes cardiac and respiratory failure [15]. The mortality in op poisoning mostly depend upon first aid treatment, availability of antidote and respiratory support [16].

The protocol fallowed throughout the world includes early stomach washout, administration of atropine and Injection Pralidoxime [17].

Hypothesis

To conduct the study a null hypothesis was created which state that there is no relationship between Organophosphorus compound poisoning and myocardial changes and there is no ECG manifestation in OP poisoning

Objective of Study

To study the relationship between Organophosphorus compound poisoning and ECG changes.

Rationale of Study

To aware the doctors and paramedics about the effects of Organophosphorus compound on cardiac activities.

Conflict of Study

There is no conflict of interest between the authors

Funding

There is no funding from any private and government organization

Methodology

Research area

The research area was the causality department of Liaquat university hospital Hyderabad.

Study Design

This was retrospective and observational type of study. Those patients who were in the causality department with history of pesticide and other Organophosphorus compounds were included in the study.

Sampling

The random sampling method was used.

Duration of study

Four months from September 2019 to December 2019.
Inclusion criteria

1. Those patients who were below 50 years of age.
2. Those patients who were brought to emergency department with the history of Organophosphorus compounds poisoning

Excluding criteria

1. All those patients who were above 50 years of age group.
2. Those who are suffering from poisoning other then Organophosphorus compounds.
3. Those that have the history of cardiac diseases
4. Those with history of hypertension.

Statistical analysis

The software SPSS 16 was used analyses of data. Descriptive analysis was done to detect the frequency of participants. To detect P value cross tab was used. Significant P value was less than 0.005.

Test Procedure

Patients who suffered from Organophosphorus poisoning were first admitted in emergency department. They were clinically examined and graded according to paradeniya Organophosphorus poisoning (POP) scale. The patients were examined for their pupil size, Fasiculations, respiratory, cardiac rate, blood pressure, fits and state of consciousness. The initial dose of atropine was 1.8-3 mg and it was doubled after every 5 minutes. It was continued until full atropinization occurred which was characterized by dilated pupil, stoppage of excessive secretion and increase in heart rate i.e. 110-120 beats per minute. After the achievement of atropinization 20% of dose was continued as maintenance dose for 2 days. A high bolus dose of injection Pralidoxime 30mg/kg per body weight fallowed by 8mg/per body weight. All the parameters regarding the patient were monitored including their age, gender, intentions of poisoning, time passed after the poisoning, hypotension, hypertension, aterial fibrillation and heart block. The ECG changes were evaluated carefully.

Results

1. Total 100 patients participated in the study. There were76 (76%) patients who were less than 30 years while 24(24%) were between 30-50 years.

Table 1 Frequency of participants

Age	Frequency	percent
Less than 30	76.0	76.0
Between 30-50	24.0	24.0
Total	100.0	100.0

2. There were 46(46%) female and 54(54%) male included in the study

Table 2 Frequency of gender

Gender	Frequency	Percent
Female	46	46.0
Male	54	54.0
Total	100	100.0

3. There were 50 participants from the control or non-exposed group while 50 were from exposed group

Table 3 Status of participants

Status of patients	Frequency	Percentage
non exposed	50	50.0
exposed	50	50.0
Total	100	100.0

4. There were 10 out of 100 participants who suffered from accidental type of poisoning, 35 suffered from suicidal poisoning and 5 suffered from homicidal type of poisoning.

Table 4 Type of poisoning

Type of poisoning	Frequency	Cumulative Percent
accidental	10	10.0
suicidal	35	35.0
homicidal	5	5.0
control group	50	50.0
Total	100	100.0

5. Table no 5 showing that 72% of patients showed ECG changes.

Table 5 ECG changes in different status of patients

Status of patients		P value
Non exposed	0 (0%)	0.000
Exposed	36 (72%)	
total	36 (72%)	

6. There were 47 literate and 53 illiterate patients showing more incidence among the illiterate peoples

Table 6 Frequency of literacy rate in patients of OP poisoning

Education	Frequency
Literate	47
illiterate	53
Total	100

7. In patients less than 30 there were 12(8.3%) patients suffering from tachycardia, 2 (8.3 %) from bradycardia, 2(2.6%) showed ST elevation,2(2.6%) showed extrasystole,2(2.6%), 2(2.6%) showed t wave inversion, 2(2.6%) showed heart block,),0 (0%) showed MI while 49(64%) did not showed any ECG changes.
In patients between 30-50 less there were 12(15.8%) patients suffering from tachycardia, 0 (0%) from bradycardia, 0 (0%) showed QT prongation, 0(0%) showed ST elevation, 2(2.6%) showed extrasystole, 0(0%) showed t wave inversion, 2(8.3%) showed heart block,), 3 (12.5%) showed MI while 15(62.5%) did not showed any ECG changes.

Table 7 Age wise ECG and Cardiac changes in patients of OP poisoning

Age	Tachycardia	bradycardia	QT prolong	ST elevation	Extra systole	T inversion	Heart block	MI	No ECG changes	total	P value
Less than 30	12(15.8%)	5(6.6%)	2(2.6%)	2(2.6%)	2(2.6%)	2(2.6%)	2(2.6%)	0(0%)	49(64%)	76(100%)	.057
Between 30-50	2(8.3%)	2(8.3%)	0(0%)	0(0%)	0(0%)	0(0%)	2(8.3%)	3(12.5%)	15(62.5%)	24(100%)	
Total	14(14%)	7(7%)	2(2%)	2(2%)	2(2%)	2(2%)	4(4%)	3%)	64%)	100(100%)	

8. In the exposed group there were 14(28%) patients suffering from tachycardia, 7(14%) from bradycardia, 2(4%) from QT prolongation, 2(4%) from ST elevation, 2(4%) from extra systole, 2(4%) from T wave inversion, 4(4%) heart block, 3(6%) from MI, 14(28%) did not show any ECG changes. In the non-exposed group there were no ECG changes or cardiac effects were seen. The P value was 0.000.

Table 8 ECG changes in different status of patients

Status of patient	tachycardia	bradycardia	QTc Prolongation	ST elevation	Extra systole	T inversion	Heart block	MI	No ECG changes	Total	P value
Non exposed	0(.0%)	0(.0%)	0(.0%)	0(.0%)	0(.0%)	0(.0%)	0(.0%)	0(.0%)	50(100%)	50(100%)	.000
exposed	14(28%)	7(14%)	2(4%)	2(4%)	2(4%)	2(4%)	4(4%)	3(6%)	14(28%)	50(100%)	
total	79(7%)	7(7%)	2(2%)	2(2%)	2(2%)	2(2%)	4(%)	393%)	64(64%)	100(100%)	

Discussion

The involvement of organophosphorus compounds in cultivation has increased the risks of harming persons who are involved in its handling. The pulmonary complications are renowned worldwide but awareness regarding the cardiac complications is less. This study was designed to access the cardiac complication through changes in ECG findings. The present study shows 28% tachycardia, 14% bradycardia and 2% prolongation of QT interval. 4% patients showed extra systole, T inversion and heart block.6% suffered from MI while 28% did not show any ECG changes. Most of tachycardia changes were noted after the administration of atropine while the bradycardia effects were before the administration of atropine. In the study conducted by Tripathy S in 2017 a quite similar result was observed i.e. 31% tachycardia,28% increase QT interval and 4% extrasystole.in the study conducted by Launderi S in 2014 49% cardiac changes were observed. An increase QT interval in 14% was found while 12% showed extrasystole.in the study conducted by Kumar S a similar ECG manifestations was found with increase ST elevation wave inversion, prolonged QT and interval. The study

conducted by Karki Pin 2004 62% cardiac complications were detected with prolonged QT interval in 37% of cases, ST elevation in 29% of cases while tachycardia in 40% of cases. These results also showed that OP compounds disturb cardiac rhythm but the results were in higher proportion. In the study conducted by Bika Ram et al in 2010 also observed a similar type of results. 78% cardiac changes were noted with Bradycardia 14%, tachycardia 12%, St Elevation in 10% and T inversion in 10% of patients.

Conclusion

It was concluded that Organophosphorus compounds has a direct injury to myocardium which is reflected by ECG changes.

References

[1]. Elspeth J, Hulse, James O and Eddleston M, Respiratory Complications of Organophosphorus Nerve Agent and Insecticide Poisoning. Implications for Respiratory and Critical Care, Am J Res Crit Care Med, 2019, 1,200(7):946.

[2]. Bilal M, Yaseen K, Saad A et al, The pattern of Organophosphorus poisoning and it's short term outcomes in various socioeconomic groups, KJMS, 2014,7, (1)11-16.

[3]. Joshi P, Manoria P and Joseph D, Gandhi Z. Acute myocardial infarction: Can it be a complication of acute Organophosphorus compound poisoning? J Postgrad Med, 2013; 59:142-4.

[4]. Laudari S, Patowary BS et al, Cardiovascular Effects of Acute Organophosphate Poisoning, Asia Pac J Med Toxicol, 2014, 3, 64-67.

[5]. Karki P, J A Ansari, S Bhandary, S Koirala, Cardiac and Electrocardiographical manifestations of acute organophosphate poisoning, Singapore Med J 2004, 45(8) : 385.

[6]. Mahrous A, Ibrahim A, Mohy K et al Comparison of the accuracy of two scoring systemsin predicting the outcome of organophosphate intoxicated patients admitted to intensive care unit, a Egyptian journal of forensic sciences,2011,1,41-47.

[7]. Balouch GH,Yousfani AH Jaffery MH et al, Electrocardiographical manifestation of acute Organophosphorus poisoning, World Applied Sciences Journal 16 (8): 1118-1122, 2012

[8]. Tripathy SK, Rout PK, Debta N, Study of clinical profile of Organophosphorus poisoning with special reference to electrocardiographic changes and electrolyte derangement, International Journal of . Int J Adv Med. 2018 Feb; 5(1): xxx-xxx http://www.ijmedicine.com

[9]. Venkatesan VNA, Anandhi PG, Shridharan P et al, A study on cardiac effects of acute organo-phosphorus compound poisoning and its postmortem toxicological findings, wjpmr, 2019, 5(2), 170-174.

[10]. Nurulain SM, Different approaches to acute Organophosphorus poison treatment, JPMA, 2012, 62 (7), 712-717.

[11]. Kumar S, Diwan SK and Dubby S, S Myocardial infarction in Organophosphorus poisoning: Association or just chance, , J Emerg Trauma Shock. 2014 Apr-Jun; 7(2): 131–132.

[12]. Makwana S, Saiyad M N, Makwana V. Electrocardiographic changes in organophosphate poisoning - A prospective study of 50 cases at a tertiary care center in Gujarat. J Integr Health Sci 2017; 5:18-24.

[13]. Aftab A, Ali l, Shehbaz L et al, Prevalence and characteristics of organophosphate poisoning at a tertiary care centre in Karachi, Pakistan, Pak J Surg, 2016; 32(4):269-273.

[14]. Ho Chul Kwon, Yong Sung Cha, Gyo Jin An, Yoonsuk Lee, Hyun Kim, Usefulness of serum lactate as a predictor of successful discontinuation of continuous atropine infusion in patients with severe acute organophosphate poisoning Clin Exp Emerg Med 2018;5(3):177-184.

[15]. Menezes MT, Celotte AC, Sumerelli AA et al, In vitro Effects of the Organophosphorus Pesticide Malathion on the Reactivity of Rat Aorta, Pharmacology, 2014;94:157–162,DOI: 10.1159/000367897.

[16]. Gunduz E, Dursun R, Mustafa I et al, Factors affecting mortality in patients with organophosphate poisoning, JPMA, 2015, 65: 967-972.

[17]. Banerjee I, Tripathi S K, Roy A S. Efficacy of pralidoxime in Organophosphorus poisoning: Revisiting the controversy in Indian setting. J Postgrad Med, 2014, 60:27-30.