Original Research Article

A study of the ECG changes associated with Organophosphorous compound poisoning

N. Jayaprakash¹, S. Geetha^{2*}

¹Assistant Professor, Department of Medicine, Kilpauk Medical College, Chennai, Tamil Nadu, India ²Assistant Professor, Department of Medicine, Government Royapettah Hospital, Chennai, Tamil Nadu, India

*Corresponding author email: drsgeetharamesh@gmail.com

	International Archives of Integrated Medicine, Vol. 6, Issue 2, February, 2019. Copy right © 2019, IAIM, All Rights Reserved.						
	Available online at http://iaimjournal.com/						
IAIM	ISSN: 2394-0026 (P)	ISSN: 2394-0034 (O)					
	Received on: 10-02-2019	Accepted on: 14-02-2019					
	Source of support: Nil	Conflict of interest: None declared.					
How to cite this article: N. Jayaprakash, S. Geetha. A study of the ECG changes associated with							
Organophosphorous compound poisoning. IAIM, 2019; 6(2): 69-73.							

Abstract

Introduction: Estimates from the World Health Organisation (WHO) indicate that 1 million accidental poisoning and 2 million suicide attempts involving pesticides occur the world over. Large proportion present to the ICU with an acute suicidal attempt with male predominance. The case mortality in the developing world is more than 20%. The cardiac manifestations are hypotension, bradycardia, and varied Electrocardiogram (ECG) changes manifested by prolonged QTc interval, inverted T waves, prolonged PR interval, and ST-segment elevation. There is also a high incidence of respiratory failure which has a high mortality.

Aim of the study: ECG changes and systemic manifestations with organophosphorous compounds and its relationship to the nature of the compound and its outcome.

Materials and methods: The study was conducted at Rajiv Gandhi Government General Hospital and Madras Medical College during a month period from June 2010 and October 2010. The study was conducted on a total of 102 patients admitted to the toxicology unit of Rajiv Gandhi Government General Hospital. A detailed history and physical examination and biochemical and ECG monitoring were done.

Results: Most of the patients were in the age group 21-30. The mortality rate was 17%. The percentage of death was noted to be increased in the extremes of age groups less than 20 and more than 60 years. Of the patients who expired mortality was highest in monocrotophos consumption and a majority of them had ECG changes and renal (16%)/respiratory failures (58%). The ECG abnormality was seen in 64% of individuals.

Conclusion: We concluded from this study that Electrocardiographic changes correlated independently with the prognosis of the OP poisoning cases and the identification of them, particularly QTc prolongation and timely shifting of cases to ICU and CCU where adequate

resuscitative measures, ventricular pacing facilities available can prevent such sudden cardiac deaths. Blood transfusion is said to have a role in severe poisoning in rapidly replenishing acetylcholinesterase enzyme.

Key words

Organophosphorus poisoning, ECG changes, Organ dysfunction.

Introduction

Organophosphorous compounds are chemical agents in widespread use throughout the world mainly in agriculture. They are readily available "over the counter" despite them being a major cause of morbidity and mortality [1]. Poisoning results in accumulation of acetylcholine causing overstimulation of both muscarinic and nicotinic receptors and subsequent disruption of nerve impulse transmission in the central and system peripheral nervous [2]. Organophosphorous compound poisoning is the most common medicotoxic emergency in India. These compounds, discovered more than 100 years ago, are at present the predominant group of insecticide and pesticide all over the world. Its widespread use and easy availability have increased the likelihood of poisoning with these compounds [3]. Cardiac manifestations often accompany poisoning with these compounds including, hypotension, hypertension, sinus bradycardia, sinus tachycardia and cardiac arrest [4]. Electrocardiographic changes reported in previous studies include QTc prolongation, ST-T along with various forms changes, of arrhythmias, which may be serious and fatal. These complications are potentially preventable if recognized early and treated adequately. The organophosphorus compounds are the organic derivatives of phosphorous-containing acids. The phosphonate, which are organic derivatives of phosphoric acid are, not used as insecticides but used as chemical warfare are agents. Organophosphorous compounds combine with esteratic sites of acetylcholinesterase, that is phosphorylated & phosphorylated esteratic sites undergo hydrolysis [5]. The phosphorylated enzyme is inactive and thus unable to hydrolyze acetylcholine. The biological effects of the organophosphorus compound are as a result of

the accumulation of endogenous acetylcholine at sites of cholinergic transmission. This causes disruption of transmission of nerve impulses in both peripheral and central nervous system [6]. Most organophosphorus compounds are readily absorbed through the respiratory, oral and gastrointestinal mucous membrane, and through intact skin, as they are lipid soluble. This binding is irreversible, except with early pharmacological intervention [7]. The diagnosis is based on the history of exposure and features of cholinergic over activity. The treatment includes atropine or glycopyrrolate, which acts as a physiological antidote and oximes which help in reactivating the enzyme [8]. Complications like respiratory failure, CNS depression, and ventricular arrhythmias should be anticipated and treated. manifestations Cardiac often accompany poisoning with these compounds including, hypotension, hypertension, sinus bradycardia, sinus tachycardia and cardiac arrest [9]. Electrocardiographic changes reported in previous studies include Sinus tachycardia, Sinus bradycardia, QTc prolongation, ST-T changes, along with various forms of arrhythmias, which may be serious and fatal. These complications are potentially preventable if recognized early treated adequately. Organophosphate and poisoning has been postulated both in animal and human studies to cause myocardiotoxic damage (myocardial necrosis). Electrocardiographic changes organophosphate compound in poisoning have been reported along with the myocardial associated structural damage. Organophosphate compound poisoning itself causes diarrhea and vomiting which can lead to electrolyte derangements which by themselves may impart electrocardiographic changes [10].

Materials and methods

This study was conducted in the department of toxicology Government General Hospital Chennai between June 2010 and October 2010. It included 102 of the total 155 cases admitted. A detailed history and physical examination and biochemical and ECG monitoring were done.

Inclusion criteria: All adults with a history of consumption and/or exposure to OPC of either sex, admitted in the hospital within 12 hours of ingestion.

Exclusion criteria: All patients with poisoning due to compounds other than OPC, Patients with prior H/o consumption of OPC, Patients having H/o cardiac diseases.

ECG was recorded in all cases before administering atropine, pralidoxime or any other medications in casualty and serial ECG were taken in all cases until discharge following recovery from poisoning/death to study the possible abnormalities which could have been from organophosphate compound poisoning.

Statistical analysis

this software frequencies, Using range, percentages, means, standard deviations, chisquare, and 'p' values were calculated. Kruskal Wallis chi-square test was used to test the significance of the difference between quantitative variables and Yate's chi-square test for qualitative variables. A 'p' value less than 0.05 was taken to denote the significant relationship

Results

In our study of the total 102 organophosphorus compound admissions, 82 patients were referrals which accounted for 80%. 59% were in the age group of 21-30. Death percentage was maximum in age >50 years at 31.12%. There was a male predominance of 70.37% and of that expired 58.35 % were males. 35 persons-25.32% were on mechanical ventilation. The mortality in ventilated patients was 14 that was 37.14%. 43.7%-No changes, 31%- QT prolongation, 20%- ST-T changes, 25%- bradycardia, 5.4%extra systoles, 16.4% - arrhythmias (Table – 1).

	MILD		MODER	ATE	SEVER	E	Р	Significance
	Mean	SD	Mean	SD	Mean	SD	Value	
	(days)		(days)		(days)			
QTc	2.39	0.92	1.88	0.77	1.28	0.95	0.001	Highly significant
ST elevation	1.22	0.26	0.82	0.32	0.50	0.00	0.008	Highly significant
T inversion	2.22	0.67	1.81	0.53	0.75	0.35	0.019	Significant
Low voltage	-	-	1.75	0.35	1.41	0.99	0.651	Not significant
PR prolongation	2.00	0.00	2.50	0.71	-	-	0.424	Not significant
AF	-	-	2.25	1.06	-	-	-	Not significant
Extra systole	0.87	0.25	1.60	0.55	0.81	0.27	0.016	Significant

<u>**Table** – 1</u>: Association of ECG changes with op poisoning regarding their number of days of occurrence in serial 12 hour recordings.

Discussion

ECG changes were noted mostly in highly toxic compounds like monocrotophos, Quinalphos, Chlorpyrifos, Baytex. We have observed in our study that the majority of the patients were farmers coming from nearby villages. Majority of them were in the lower socioeconomic group. Of the cases, only 9 were spray poisoning. The rest were accidental in nature [11]. All the patients except 2 received stomach and body wash. Atropine was administered to all of them. The average days of atropine requirement were 2 days [12]. Pralidoxime was administered to 112 patients. Ventilatory support was given to 35 patients [13]. Among the ECG changes observed overall, QTc prolongation tops the list with 68

cases (60.7%) followed by ST elevation (21.4%), T inversion (16.9%), Extrasystole (12.5%), Low voltage complexes (11.6%), PR prolongation (3.5%) and AF (1.7%) with 24, 19, 14, 13, 4 and 2 cases respectively. Among the ECG changes of survivors, OTc prolongation tops the list with 48 cases followed by ST elevation, T inversion, Extrasystole, Low voltage complexes, PR prolongation and AF with 20,14,11,5,4 and 2 cases respectively [14]. Thus the association of QTc prolongation, T inversion and Extrasystole with OP poisoning with regard to the number of days of their hospital stay appears to be highly significant; for Low voltage, complexes appears ST significant; and for elevation, PR prolongation and AF appears not significant [15].

Conclusion

The organophosphorus compound is a widely used suicidal agent in our population. A few are accidental poisoning due to spray. The mortality rate in our hospital which is a referral center, where 80% were referral cases was 17% with the highest mortality in 20-30 years which is a very productive age group. Mortality is associated with a wide variety of ECG changes. Mortality is related to the nature of the compound. Monitoring of the sales of these compounds may help to limit its availability as a suicidal agent and a huge burden on the productive population.

References

- Karalliedde L, Senanayake N. Acute organophosphorus insecticide poisoning in Sri Lanka. Forensic Science International, 1988; 36: 97-100.
- Taylor P. Anticholinesterase agents In Gilman AG, Goodman LS, Rall TW, Murad F, eds. The pharmacological basis of therapeutics. New York: Mac Millan, 1985; p. 110-129.
- Mutalik GS, Wadia RS, Pai VR. Poisoning by diazinon an organophosphorus insecticide. Journal of Indian medical association, 1962; 38: 67-71.

- Maroni M. Review of toxicological properties and biotransformation of organophosphorus esters. In: WHO Manual of Analytical methods. Cremona: WHO collaboration center for occupational health, 1985; 3-39.
- Hayes WJ. Organophosphorous insecticide. In: Hayes WJ, ed. Pesticides studied in man. Baltimore, Williams, and Wilkins, 1982; p. 284-413.
- Karalliedde L. Organophosphorous poisoning and anesthesia. Anaesthesia, 1999; 54: 1073-1088.
- Johnson MK, Lauwerys R. Protection by some carbamates against the delayed neurotoxic effects of diesopropylphosphorofluoriddate. Nature, 1969; 222: 1066-1067.
- Moss DW, Henderson DR, Kachmar JF, Enzymes. In: Tietz NW, ed. Textbook of Clinical Chemistry. Philadelphia, WB Saunders Co; 1986; p. 619- 774.
- Grob D, John RJ. Use of oximes in the treatment of intoxication by anticholinesterase compounds in normal subjects. American Journal of medicine, 1953; 24: 497-511.
- Garcia-Repetto R, Soria ML, Gemini MP, Menendez M, Repetto M. Deaths from pesticide poisoning in Spain from 1991 to 1996. Veterinary and Human Toxicology, 1998; 40: 166-168.
- Senanayake N, Karalliedde L. Neurotoxic effects of organophosphorus insecticide. NEJM, 1987; 316: 716-763.
- 12. Leon-S-Fidas E, Padilla G, et al. Neurological effects of the organophosphorus pesticide. BMJ, 1996; 313: 690-691.
- Surjit Singh, Sharma. Neurological syndromes following Organophosphorus poisoning. Neurology India, 2000; 48: 308-313.
- 14. Johnson MK, Lauwerys R. Protection by some carbamates against the delayed neurotoxic effects of diisoproylphosphoroflouride. Nature, 1969; 222: 1066-1067.

15. Bidstrup PL, Bonnell JA, Beckett AG. Paralysis following poisoning by a new Organophosphorous insecticide (mipafox). BMJ, 1953; 1: 1068-1072.