International Journal of Advances in Medicine
Tripathy SK et al. Int J Adv Med. 2018 Feb;5(1):50-56
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: http://dx.doi.org/10.18203/2349-3933.ijam20175520

Original Research Article

Study of clinical profile of organophosphorus poisoning with special reference to electrocardiographic changes and electrolyte derangement

S. K. Tripathy[1], P. K. Rout[1], N. Debta[2], S. Das[1], M. Panigrahi[3], S. K. Mishra[1], S. P. Suna[1], M. R. Behera[1]

[1]Department of Medicine, SCB Medical College, Cuttack, Odisha, India
[2]Department of Medicine, All India Institute of Medical Sciences (AIIMS), Bhubaneswar, Odisha, India
[3]Department of Ophthalmology, District Headquarter Hospital, Kendrapada, Odisha, India

Received: 28 November 2017
Accepted: 04 December 2017

***Correspondence:**
Dr. S. K. Tripathy,
E-mail: sarojtripathy1@hotmail.com

ABSTRACT

Background: Organophosphorus Compounds (OPC) are main cause of accidental and suicidal poisoning in agrarian countries like India. Aim was to study the clinical profile of OPC-Poisoning and correlate it with the electrocardiographic (ECG) changes and electrolyte abnormalities.
Methods: Hundred consecutive cases admitted to Medicine Department underwent clinical examination, ECG, electrolytes, Acetyl Choline Esterase (AChE) estimation from time to time and Paradeniya Organophosphorus Poisoning (POP) score at the time of admission. All these parameter with duration of hospital stay and outcome were statistically analysed using X2 test, Fisher exact test, and inference was drawn.
Results: In hundred OPC-Poisoning patients [Male (n=48), Female (n=52), M: F ratio 0.92:1] with mean age of 37.78±12.95 years, commonest poison was cholropyrifos+cypermethrin and was mostly suicidal (96%). Common symptoms were sweating (48%), salivation, blurring of vision, breathlessness and signs were smell of poison (90%), tachypnea, altered sensorium, miosis and fasciculation. POP scoring found 41% of patients in mild, 26% in moderate and 33% in sever grade of poisoning. Hospital stay ranged from 4-18 days. Complications were pulmonary Edema (PE) in 28%, Respiratory Failure (RF) 18%, Aspiration Pneumonia (AP) 15% and Intermediate Syndrome (IS) 4%. 10 died out of 42 patients who had complications and the cause of death was RF in 4, Ventricular Fibrillation (VF) 2 and IS in 2. ECG finding showed sinus tachycardia (31%), prolonged corrected QT (QTc) interval (28%), sinus bradycardia (25%), ST-T changes (17%) and Premature Ventricular Contraction (PVC) in 4% which degenerated to VF in 2%. 24 patients were Hypokalemic from which 16 developed complications.
Conclusions: Similar to earlier studies we observed poisoning which was suicidal. QTc prolongation and Hypokalemia are associated with high morbidity and mortality in OPC-Poisoning.

Keywords: Electrocardiography, Hypokalemia, OP compounds, Paradeniya OP score, QTc acetyl choline-estarage

INTRODUCTION

OPCs are used as insecticides and herbicides, in agriculture and solvents, plasticizer in industrial and domestic settings throughout the world. It has been used in warfare as nerve gas and most important of all as a tool for suicidal poisoning. The ease of availability of OPCs has resulted in increase in the occupational, accidental and suicidal poisoning.[1] Accidental poisoning can occur after exposure through skin or inhalation whereas suicidal poisoning is by ingestion of OPCs. Estimates show 80% of pesticide related hospital admissions are caused by

OPC-Poisoning and two thirds of self-harm deaths in developing countries is caused by OPC-Poisoning.[2,3] WHO estimates one million serious accidentals and two million suicidal poisoning by OPCs occurring worldwide and approximately 200,000 deaths in developing countries every year.[1] According to Davies et al 2008 OPC-Poisoning kills around two lakhs people every year due to self-poisoning in Asia-Pacific region.[4]

OPCs constitute a heterogeneous category of irreversible Anti Choline Esterase activity containing chemicals specifically designed for the control of pests, weeds and plant diseases. They irreversibly inhibit AChE and thereby protects Acetylcholine (Ach) from hydrolysis and consequent excessive ACh causes cholinergic stimulation via the muscarinic and nicotinic receptors found in the Central Nervous System (CNS), Peripheral Nervous System (PNS) and autonomic sympathetic preganglionic synapses parasympathetic post ganglionic synapses and neuro muscular junctions of skeletal muscle. Anti-choline esterase compounds are of two types i.e. Reversible type (Carbamates, Acridine) and irreversible type (OPCs). OPCs are chemically derived from Phosphoric, phosphonic, phosphonic or triphosphoric acids usually as esters, amides or thio derivatives.

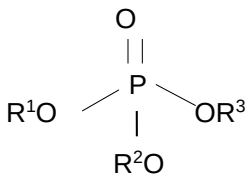

Figure 1: Organophosphorus compound general formula.[5]

OPCs are Dimethyl or Diethyl Phosphoryl compounds as presented in Table 1. Nerve gases like Tabun, Sarin and Soman are highly potent synthetic toxic OPC agents. OPCs can easily cross the respiratory epithelium, dermal and gastric mucosa because of their lipophilic structure. OPCs get distributed in the whole body with a special affinity for fatty tissues but their fast degradation prevents its long term accumulation.

Table 1: Types of organophosphorus.

Dimethyl OP	Diethyl OP
Parathion	Methyl parathion
Diazinon	Dichlorovos
Chloropyrifos	Dimethoate
Dichlorofenthion	Malathion
Coumaphos	Fenthion

The enzymes inhibited by OPCs provide specific biomarkers of exposure until the turnover of enzyme in favorable cases. Accessible AChE enzyme is found in red blood cells and butyl choline esterase (BChE) in plasma for estimation. As BChE is not specific, so AChE estimation in OPC poisoning is essential.

After initial inhibition of AChE and formation of AChE-OPC complex, two further reactions occur. First is Spontaneous Reactivation which is reactivation of the AChE usually occurring at a slower rate and the rate of regenerative process depends on the type of OPC like 0 to 7hours in Dimethyl group and takes several days (average of 31hours) in Diethyl group. This spontaneous reactivation of the enzyme can be hastened up by addition of nucleophillic agents like Oximes [ex-Pralidoxime (PAM). The second reaction is called Ageing which is nothing but with time the AChE-OPC complex losing an alkyl group makes it no longer responsive to the reactivating agents like Oximes. The rate of ageing depends on various factors like pH, temperature and the type of OPC. The ageing half-life of dimethyl OPC is 3-7hours and diethyl OPC is around 31hours. Since ageing is rapid in dimethyl OPCs, Oximes are useful when started before 12hour's into poisoning where as in diethyl OPC-Poisoning, oximes may be useful for several days

Clinical effects of OPC-Poisoning occur in 3 phases. The first is acute cholinergic crisis phase, second is delayed complications like polyneuropathy and a phase in between these two known as IS which is a distinct clinical entity.[6]

Muscarianic receptor stimulation results in dizziness, nausea, vomiting, abdominal Pain, diarrhoea, miosis, blurred Vision, salivation, lacrimantion, urinary, fecal incontinence and bronchial secrections leading to RF.[6] Nicotinic receptor stimulation result in easy fatigue, weakness, muscle cramps, fasciculations, skeletal muscle twitching, convulsions and flaccid paralysis.[7]

CNS effects include irritability, nervousness, ataxia, depression of respiratory centre and vasomotor centre resulting in dysnea, cyanosis, hypotension, lethargy, impairment of memory, confusion, vonvulsions, respiratory depression, hypoventilation and coma. In moderate to large doses of OPC-Poisoning the nicotinic and CNS effects predominate over the muscarianic effects.

Multifocal PVC's, Idioventricular arrhythmias, AF, VF, transient ST-T changes and different grades of heart blocks including complete heart block are also seen in OPC-Poisoning.[8-11] The mechanism of cardiac toxicity is caused by more than one mechanism and are sympathetic, parasympathetic overactivity, hypoxemia, acidosis, electrolyte derangements and direct toxicity on myocardium and conduction system of the heart.[12,13] The antidote Atropine may itself induce arrhythmia. Hypotension and prolong QTc interval have been described to be independent predictors of mortality in patients with OPC induced cardiotoxicity.[14-16] Muthu V et al described hypothermia and cardiotoxicity in a case of OPC-Poisoning (Phorate).[17] Patchy interstitial and myocardial inflammation on histopathological examinationhas been described.[18]

IS usually occurs 24 to 96hour's after initial cholinergic crisis phase but before delayed polyneuropathy in 10-40 percent of OPC-Poisoning and is characterised by prominent weakness of neck flexors, muscles of respiration and proximal limb muscles lasting for 5-14 days with RF as the dreaded complication in this phase needing mechanical ventilatory support for survival. Oximes have no role only early recognition and respiratory support usually tides over this crisis.[19]

The late phase delayed plyneuropathy is an uncommon consequence of OPC-Poisoning which begin after 2-5 weeks after exposure and may last for years. Wadia classified the neurological manifestations including RF into Type 1 paralysis (occurring within 24hour's) and Type 2 paralysis (occurring after 24hours).

For management of OPC-Poisoning Senanayake N proposed POP Score for grading the severity of poisoning basing on five cardinal manifestations as presented in Table 2 and Table 3.[20]

Table 2: Paradeniya organophosphorus poisoning score.

Parameters	score
Miosis	
Pupil size>2mm	0
Pupil size<2mm	1
Pupil size pin point	2
Fasciculation	
None	0
Present but not generalized or conditions	1
Generalized and continuous with central cyanosis	2
Respiration	
Respiratory rate <20	0
Respiratory rate >20	1
Respiratory rate >20 with central cyanosis	2
Bradycardia	
Pulse rate >60/min	0
Pulse rate 40-60/min	1
Pulse rate <40/min	2
Level of consciousness	
Conscious and rational	0
Impaired, respond to verbal commands	1
Impaired no response to verbal commands (if convulsion present add1)	2
Total	11

POP Score, level of AChE and Potassium (K^+) at the time of presentation to the hospital are also useful in assessing the severity of poisoning and predicts the length of stay in the hospital.[2]

This study was carried out to determine the clinical profile of OPC compounds poisoning with special reference to ECG changes and electrolyte derangements and to establish correlation with the morbidity and mortality in OPC-Poisoning.

Table 3: Gradation of severity of OPC poisoning by POP score.

Score	Grade
<4	Mild
4-7	Moderate
>7	Severe

METHODS

Hundred (100) consecutively admitted cases of OPC-Poisoning to the Post Graduate Department of Medicine of SCB Medical College Hospital, Cuttack were included in the study. Inclusion criteria was adults with history of consumption and/or exposure to OPC admitted to the hospital within 12hours of exposure and not having been treated outside. We excluded poisoning other than OPCs, patients with prior history of OPC exposure, patients referred after partial treatment and patients having history of cardiac diseases. Informed consent was obtained from each patient. The study had Institute Ethical Committee (IEC) approval.

The diagnosis of OPC-Poisoning was based on the following Criteria

- History of exposure to or contact with insecticides,
- Characteristic clinical symptoms and signs of OPC-Poisoning,
- Improvement of clinical symptoms and signs after treatment with Atropine and Oximes,
- Decreased AChE activity (OPC-Poisoning was considered if serum cholinesterase activity was <50% of the laboratory minimum normal value of 4850U/L).

Evaluation of the patients started with grading of patients into mild, moderate and severe grade of poisoning by POP score at initial presentation.[21]

Immediately after admission to emergency care unit, the patients were atropinised according to the well-known protocol of 1.8-3mg IV bolus of atropine followed by doubling of the dose every 5minutes until the achievement of full atropinisation. Atropinisation was confirmed by dry secretions, dilated pupils and tachycardia (pulse rate ≈110-120/min).

Then maintenance dose of atropine (20-30% of total atropinisation dose) was calculated and patients were maintained for 2-3days until need for dose reduction came. High dose pralidoxime (PAM) was used as per WHO guidelines i.e. 30mg/kg body weight IV bolus followed by 8mg/kg/hr. IV infusion.[1,22] Subsequently atropine infusion was tapered at the rate of 1/3rd to 1/4th of daily dose and stopped when the patients recovered.

ECG was recorded on admission and every 24hour's after that throughout the hospital stay. ECG analysis included rate, rhythm, axis, ST-T changes, PR and QT interval calculation and conduction defects if any. QTc was measured as per Bezzet's fomula and QTc > 0.46secs was taken as prolonged.

The AChE measurement was done by Butyryl thiocoline potassium hexacyanoferrate (lll) method (lab value 4850-12000U/L). Serum electroytes (Na^+, K^+, Ca=2) were measured in all patients on admission and repeatedly every 24hour's. K^+ level of <3.5mmol/L was considered as hypokalemia.

The duration of hospital stays and outcome in hospital was documented. Statistical analysis was done, and values were expressed for chance of occurrence. Whenever association of attributes was done statistical X2 test, fisher exact test and standard error of difference between two proportions were applied and inference was drawn.

RESULTS

Hundred OPC-Poisoning patients admitted to Medicine Department of SCB Medical College Hospital, Cuttack consisting of 48(48%) male and 52 (52%) female patients in a M: F ratio of 0.92:1 formed the study group. The age ranged from 16-75years with mean age of 33.78-12.95 years. Maximum number of patients i.e. 49% were in 16 to 30years age group. Poisoning was suicidal in 96% and accidental in 4%of patients. The most common OPC was Cholropyrifos+Cypemethrin in 58% followed by Dichlorovos in 15%, Methylparathiion in 9%, and unknown in 7% in our study as presented in Figure 2 and Table 4.

Table 4: Showing distribution of patients as per type of poison consumed.

Types of compound	%	Mortality
Chlorpyrifos +cypermethrin	58	6 (10.34%)
Dichlorvos	15	1 (6.66%)
Methylparathion	9	0
Unknown	7	2 (28.57%)
Profenfos+cypermethrin	5	1 (20%)
Chlorpyrifos	4	0
Triazos	2	0

Vomiting (50%), followed by sweating (48%), salivation, blurring of vision, lacrimation, breathlessness and smell of poison (90%) tachypnea (82%), altered sensorium (60%), miosis (55%), fasciculation (43%), tachycardia (28%) and bradycardia (22%) were the common symptoms and signs. POP score revealed 41(41%) patients in mild, 26 (26%) in Moderate and 33 (33%) in severe grade of poisoning with mortality of 0%, 3.84% and 27.7% respectively as presented in Table 5.Maximum motality of 40% was seen in patients who got admitted >6hours after exposure. Highest mortality of 28.57% occurred in unknown poisoning followed by 20% in profenfos+cypermethrin and 10.34% in Chloropyrifos+cypermethrin.

Table 5: Showing distribution of patients as per severity of toxicity.

Patients having	No. of patients	Death
Mild	41	0
Moderate	26	1 (3.84%)
Severe	33	9 (27.27%)
Total	100	10 (10%)

Hospital stay ranged from 4-18days with majority (54%) staying for 6-10days and only 6% of the patients stayed for >15days. ECG interpretation revealed sinus tachycardia in 31%, QTc prolongation in 28%, sinus bradychardia in 25%, T wave invertion in 9%, ST depression in 8% and prolonged P-R interval in 5%.[23] 4% had arrhythmia was found in the form of PVCs and 2 patients developed VF and succumbed as presented in Table 6.

Table 6: ECG findings.

ECG changes	No. of patients
Rate	
Normal	40
Sinus tachycardia	31
Sinus bradycardia	25
Rhytm	
Sinus rhytm	96
Arrhytmia	4
Conduction defect	
Prolonged PR interval	5
St-t changes	
St elevation	0
St depression	8
T wave inversion	9
T wave flatenning	5
QTC interval prolongation	28

In patients with QTc Prolongation Group 8 (28.57%) and in normal QTc group only 2 (2.77%) expired and this difference in mortality was statistically significant X_2=10.67. (P<0.001) In QTc prolonged group 21 patients (75%) developed at least one complication where as in normal QTc group 21 (29.16%) developed complications and this difference was statistically significant x_2= 7.89, p<0.05) as presented in Table 7.

Table 7: Showing comparing severity and mortality among normal QTc and QTc prolonged patients.

No. of patients	Prolonged QTC		Normal QTC		P value
	N=28	%	N=72	%	
Complication	21	75	21	29.16	P=0.005
Severe poisoning	13	46.42	20	27.77	P=0.29
Death	8	28.57	2	2.77	P=0.001

Commonest complication was PE in 28% followed by RF in 18%, AP in 15% and IS in 4%. 42 patients had complications in our study from which 10 died and all deaths were in patients with complications.[19,24,25] In present study 24 patients were hypokalemic. In hypokalemic patients 16 (66.66%) developed at least one complication whereas in normomokalemic group 26 (34.21%) had complications and this difference was statistically significant (X2=5.004, p<0.025) as presented in Table 8.[26]

Table 8: Showing comparing severity and mortality among normokalamic and hypokalamic patients.

No. of patients	Hypokalemia		Normokalemia		P value
	N=24	%	N=76	%	
Complication	16	66.66	26	34.21	P=0.025
Severe poisoning	4	16.66	13	17.10	P=0.75
Death	4	16.66	6	7.89	P=0.654

QTc Prolongation was seen in 13 patients of severe POP grade, 12 in moderate and 3 in mild grade and the difference was statistically significant as presented in Table 9.

Table 9: Showing comparison of mild, moderate ad severe groups of poisoning.

	Mild	Moderate	Severe	P-value
Patients with	41	26	33	
Prolonged QTC	3	12	13	P < 0.001 (fisher exact test)
Complication	2	18	22	P < 0.0001 (fisher exact test
Death	0	1	9	P < 0.0001 (fisher exact test

In this study 22 (66.66%) had complications in severe grade, 18 in moderate and no complications in mild grade. The hospital stay was 4 to 18 days with mean of 7.2days Ninety (90%) patients were discharged after full recovery whereas 10 (10%) patients died in the hospital. Analysis of death in these 10 patients showed RF in 4, VF in 2 and IS in 2 as the cause.

DISCUSSION

In the developed countries 80% of suicidal poisoning result from intake of sedatives, antidepressants and related agents where as in developing countries like India OPCs remain the main cause of suicidal attempts.

In this study of 100 patients of OPC-Poisoning the mean age was 33.78±12.95years. Most of the patients i.e. 49% were in 16-30yrs of age and 81% belonged to active productive age group. Common symptoms were vomiting (50%), followed by sweating (48%), salivation (47%), blurring of vision and lacrimation. These results are comparable to the studies of Agarwal SB et al.[7] In most of the cases suicidal poisoning was severe and accidental poisoning was mild in nature in our study.

Furthermore, the main reason for OP exposure was suicidal (96%) in our study. Similar to Rafighdoost et al study in Birjand where 78.43%, Taiwan study by Lin TJ et al 64.72% and Yurumez et al study in Turkey where 85.9% of poisoning was suicidal.[2,3,10]

We observed significant higher mortality in patients hospitalized more than 4hours after exposure than hospitalization within 2hrs. Majority of the patients with a lag time <6hrs recovered and survived, whereas the recovery and survival of patients decreased with the increase in lag time for hospitalization.

Chlorpyrifos + Cypermethrin was the most common OPC agent for poisoning in our study. Wide availability and being the most commonly sold insecticide could have accounted for this.

In our study we found very high incidence of ECG changes and most significant finding was QTc prolongation and was associated with severe exposure and increased mortality. In our study 60 (60%) of patients developed cardiac manifestations. The ECG findings detected in our series were prolonged QTc, bradycardia, tachycardia, T wave inversion, AF, prolonged P-R interval, ST-elevation, PVCs, ventricular tachycardia and combined arrhythmias. These ECG findings are concordant with Karki et al study where similar arrhythmias in different proportions were observed.[13]

In a series of 168 cases of OPC-Poisoning reported by Kiss and Fazekas, five had transient picture of myocardial infarction which was not found in our study.[27-29]

The bradycardia in the early cholinergic phase of the poisoning was less frequently observed whereas the hypertension and sinus tachycardia seen in organophosphate and carbamate poisoning due to nicotinic effects was found in our study. Administration of atropine in high doses has been implicated in the development of ventricular arrhythmias. However, Ludomirsky et al and Lyzhnikov et al found no correlation between atropine therapy and ventricular arrhythmias in OPC-Poisoning.[9,18]

Overall, the frequency of QTc prolongation in several series of severe OPC-Poisoning was shown to be 20 to 80% depending on the severity of the poisoning and the type of the toxic agent. This complication usually starts during the second to third day and may last up to two weeks post-intoxication. Predisposing factors for QT prolongation and development of torsade's de pointes are older age, female gender, low left ventricular ejection fractions, left ventricular hypertrophy, ischemia and electrolyte abnormalities (commonly hypokalemia).

High frequency mortality in hypokalemia i.e. 12 out of 21 patients with complications (57.14%) in our patients had prolonged QTc which could be the major contributing risk factors. In our study, 5 patients developed polymorphic VT and 2 patients developed VF which could not be reverted back despite defibrillation, advanced cardiopulmonary resuscitation and correction of hypokalemia, and consequently led to death. In the study by Karki et al two patients died from non-cardiogenic PE and one from VF, giving a hospital mortality rate of 8.1%, similar to our study (6.9%).[13]

24% of our patients developed non-cardiogenic PE which is concordant with Karki et.al study (21.6%) contrasting Saadeh et al study showing 43% non-cardiogenic PE in Jordan.[12] In our study 5% had AV block similar to Russian series by Lyzhnikov et al 5.4% and Paul et al 8.4% of patients.[18]

Hypokalemia and paralysis are potentially reversible medical emergencies, however, when it was coupled with AChE reduction, high morbidity and mortality was observed. Balali-Mood revealed that hypokalemia may aggravate muscular weakness due to inhibition of AChE by OPC.[22]

In our study hypokalemia was most common electrolyte imbalance (24%) like past studies was seen to be associated with significant morbidity and mortality secondary to cardiac arrhythmias and RF.[26] We also found hypokalemia is related to OPC induced muscle weakness as well suggesting alteration in serum K^+ hamper neuromuscular junction activity and contribute to overall morbidity and mortality in OPC-Poisoning.[26]

Hence, the level of AChE and serum K^+ can be proposed as predictive markers for outcome in OPC-Poisoning. POP grading was used in several clinical studies and has been found useful. In our study 41 (41%) had mild, 26 (26%) had moderate and 33 (33%) had severe manifestations by POP score.

7% in mild grade and 39.39% patients in severe grade had QTc prolongation. This was statistically highly significant ($\chi^2 = 16.72$, $P < 0.001$). 66.66% of the patients in severe grade had at least one complication during hospital stay where as in mild grade no complication and no mortality was observed.

In ECG study QTc prolongation was common with mortality rate of 28.57%. Complications were significantly higher in QTc prolonged patients than in patients with normal QTc and was statistically significant ($\chi^2 = 21.18$, $P < 0.001$). Association between severity of poisoning and QTc prolongation was not significant in our study ((χ^2=7.89, P>0.05). The complications in our study are comparable to studies of Goel et al and Saadeh et al.[12,25]

CONCLUSION

The ease of access to OPCs in developing countries like India has made this the main tool for suicidal poisoning. Present study was conducted keeping in mind the paucity of studies for cardiotoxicity in OPC-Poisoning and the relation of electrolyte derangements with it.

We conclude that OPC-Poisoning is a major cause of morbidity and mortality and is suicidal in most of the cases. ECG abnormalities are more in POP severe grade of poisoning. Morbidity and mortality increases in patients with prolonged QTc and hypokalemia found in severe POP grade of OPC-Poisoning secondary to complication cardiac arrhythmias and RF. Early hospitalization and correction of hypokalemia and follow up with ECG can be life saving in OPC-Poisoning.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the institutional ethics committee

REFERENCES

1. World Health Organization. Organophosphorus insecticides: a general introduction,1986. Available at http://apps.who.int/iris/handle/10665/40198.
2. Rafighdoost A, Mirhoseini E, Pourzand H, Rafighdoust H. Epidemiology of organophosphate poisoning, and its cardiac and pulmonary effects. J Bir Uni Medic Sci. 2007;13(4(29)):49-54.
3. Lin TJ, Walter FG, Hung DZ, Tsai JL, Hu SC, Chang JS, Epidemiology of organophosphate pesticide

poisoning in Taiwan. Clinic Toxicol. 2008;46(9):794-801.
4. Davies JO, Eddleston M, Buckley NA. Predicting outcome in acute organophosphorus poisoning with a poison severity score or the Glasgow coma scale. QJM: Inter J Medic. 2008;101(5):371-9.
5. Marrazza G. Piezoelectric biosensors for organophosphate and carbamate pesticides: a review. Biosensors. 2014;4(3):301-17.
6. Wadia RS, Sadangopal C, Amin RB, Sardesai HV. Neurological manifestations of organophosphorus insecticide poisoning. J Neurol Neurosurg psychiatry. 1974;37;841-7.
7. 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. Inter J Toxicol. 2007;4(1).
8. Mathur A, Swaroop A, Agarwal A. ECG changes in acute organophosphorus compound and almunium phosphide poisoning. Ind Practitioner. 1999;52;248-252.
9. Ludomirsky A, Klein HO, Sarelli P, Becker B, Hoffman S, Taitelman U, et al. QT prolongation and polymorphous ("torsade de pointes") ventricular arrhythmias associated with organophosphorus insecticide poisoning. Am J Cardiol. 1982;49(7):1654-8.
10. Yurumez Y, Yavuz Y, Saglam H, Durukan P, Ozkan S, Akdur O, et al. Electrocardiographic findings of Acute Organophosphate Poisoning. J Emergency Medic. 2009;36(1):39-42
11. Dalvi CP, Abraham PP, Iyer SS. Correlation of electrocardiographic changes with prognosis in organophosphorus poisoning. J Postgraduate Medic. 1986;32(3):115
12. Saadeh AM, Farsakh NA, Al-Ali MK. Cardiac manifestations of acute carbamate and organophosphate poisoning. Heart. 1997;77(5):461-4.
13. Karki P, Ansari JA, Bhandary S, Koirala S. Cardiac and electrocardiographical manifestations of acute organophosphate poisoning. Singapore Medic J. 2004;45:385-9.
14. Balouch GH, Yousfani AH, Jaffery MH, Devrajani BR, Shah SZ, Baloch ZA. Electrocardiographical manifestations of acute organophosphate poisoning. W Applied Sci J. 2012;16(8):1118-22.
15. Taromsari MR, Badsar A, Aghajankhah M, Poor MA, Porkar NF. The study of electrocardiographic findings in patient with OPC-Poisoning. Iran J Toxicol. 2013;6(19):751-756.
16. Paul UK, Bhattacharyya AK. ECG manifestation in OPC-Poisoning. J Ind Medic Asso. 2012;110(2):107-108.
17. Muthu V, Dhooria S, Sehgeal IS. A rare Manifestation of organophosphorus poisoning: Hypothermia with Cardiotoxicity. Inter J Clinic Cardiol. 2014;1(1):1-5.
18. Lyzhnikov EA, Savina AS, Shepelev VM. Pathogenesis of disorders of cardiac rhythm and conductivity in acute organophasphate insecticide poisoning. Kardiologiia. 1975;15(9):126-9.
19. Palimar V, Saralaya KM, Arun M. Review of intermediate syndrome in organophosphorous poisoning. J Punjab Academy Forensic Medic Toxicol. 2006;6(6):34-5.
20. Senanayake N, de Silva HJ, Karalliedde L. A scale to assess severity in organophosphorus intoxication: POP scale. Human Ex Toxicol. 1993;12:297-9.
21. Soysal D, Karakus V, Soysal A, Tatar E, Yildiz B, Simsek H. Evaluation of Cases with Acute Organophosphate Pesticide Poisoning Presenting at a Tertiary Training Hospital Emergency Department: Intoxication or Suicide?/Bir Üçüncü Basamak Hastanesi Acil Servisine Basvuran Akut Organofosfat Zehirlenmesi Vakalarinin Degerlendirilmesi. J Aca Emerg Medic. 2011;10(4):156.
22. Balali-Mood M, Shariat M. Treatment of organophosphate poisoning. Experience of nerve agents and acute pesticide poisoning on the effects of oximes. J Physiol. 1998;92:375-8.
23. Laudari S, Patowary BS. Analysis of Organophosphorus compound poisoning patients attending CMS-TH, Bharatpur, Nepal. J Coll Medic Sci-Nepal. 2012;7(4):9-19.
24. Goswamy R, Chaudhuri A, Mahasur AA. Study of respiratory failure in organophosphorus and carbamate poisoning. Heart Lung. 1994;23(6):466-72
25. Goel A, Joseph S, Dutta TK. Organophosphate poisoning: predicting the need for ventilator support. J Asso Physic Ind. 1998;46(9)786-90.
26. Stedwell RE, Allen KM, Binder LS. Hypokalemic paralyses: a review of the etiologies, pathophysiology, presentation, and therapy. Ame J Emerg Medic. 1992;10:143-8.
27. Kiss Z, Fazekas T. Arrhythmias in organophosphate poisonings. Acta cardiologica. 1979;34(5):323-30.
28. Joshi P, Manoria P, Joseph D, Gandhi Z. Acute myocardial infarction: Can it be a complication of acute organophosphorus compound poisoning? J Postgraduate Medic. 2013;59:142-4.
29. Kumar S, Diwan SK, Dubey S. Myocardial infarction in organophosphorus poisoning: Association or just chance? J Emerg Trauma Shock. 2014;7:131-2.

Cite this article as: Tripathy SK, Rout PK, Debta N, Das S, Panigrahi M, Mishra SK, et al. Study of clinical profile of organophosphorus poisoning with special reference to electrocardiographic changes and electrolyte derangement. Int J Adv Med 2018;5:50-6.