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Clinical profile of organophosphate compound poisoning in a tertiary care centre

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Abstract
Background – We studies clinical profile of organophosphorous poisoning in our institute.

Material And Methods – This open prospective study was conducted In the Department of Medicine, Grmc, Gwalior, All patients admitted In J.A. Group of Hospitals from nov 2012 to oct. 2013 were studied. Out of total 218 patients, 91 patients who gave consent and fit in inclusion criteria were taken for the study and rest who refused to give consent and having character of exclusion criteria were excluded from the study.

Results- Males of age group 21-30 years, from rural population in our study were mainly affected by OP poisoning, and most patients belongs to low to middle class of society. Most patients were mildly affected. But most death were in severe toxicity group. Hypokalemia was most common electrolyte imbalance and QTc prolongation was mostly associated with mortality and significantly associated with the complications.

Conclusion- Organophosphorous poisoning is major poisoning in India and early hospitalization, correction of hypokalemia and QTc prolongation could be life saving in these patients.

Keywords: Poisoning, Organophorous Compounds, Organophosphorous Poisoning, Toxicity.

1. Introduction
The most common substance which produces the medical and medicolegal conditions are the organophosphorous compounds. This entity of chemicals was discovered by Schrader during 2nd world war since than it is used as agricultural pesticides, warfare agents in form of nerve gases and a tool for suicide attempts since five decades. The fact that in developing countries these products are easily available and could be leads to intentional, accidental and occupational exposure. As per literature about 3 million exposure occurs annually worldwide. [1,2,3] As per World health organization organophosphorous poisoning is a cause of more than 2.2 lakhs deaths. Countries like srilanka and india have higher rates of exposure and mortality. [3,4] Organophosphorous compounds are anticholinesterase in nature and exposure to human body manifests mainly by excessive accumulation of acetylcholine at synapses and neuromuscular junctions, and leads to various clinical manifestations and late treatment may cause death [5]. Our study is aims at to find out the clinical profile of organophosphorous poisoning.

Material and Methods
This open prospective study was conducted In the Department of Medicine, Grmc, Gwalior, All patients admitted in J.A. Group of Hospitals from NOV 2012 TO OCT. 2013 were studied. Out of total 218 patients, 91 patients who gave consent and fit in inclusion criteria were taken for the study and rest who refused to give consent and having character of exclusion criteria were excluded from the study.
The inclusion and exclusion criteria were following

Inclusion Criteria-
The Patient presenting with H/o organophosphate compound poisoning, sign & symptoms of acute organophosphate poisoning and not having been treated outside.

Exclusion Criteria-

1 Patients who have taken other poisons along with OP poisoning. 2-Patient who received partial treatment outside and referred later to our hospital were excluded.3 Patients with severe vomiting.4-Patient having chronic illness.5-Patient with renal failure.6-Patients with doubtful diagnosis.7-Patient with cardiac failure. All patients were subjected for detailed history in clinical examination, sign & symptom were analyzed. ECG was recorded on admission as before starting therapy (and from then on every 24 hrs during the period of stay in the hospital). ECG analysis included the rate, rhythm, QRS axis, ST-T changes, conduction defects, measurement of PR and QT intervals. The QT interval measured manually from the beginning of Q wave to the end of T wave. End of T wave was recognized by return of the T wave to T-P baseline. QT interval as per bezzets formula was measured in all the leads and the longest QT interval was used for calculation of QTc.The upper limit of the duration of QTc interval is approximately 0.46s (460ms). QTc interval more than 0.46s is taken as prolonged QTc interval in this study.Serum electrolytes (Na+, K+, Ca+2) were taken in all patients before administering any treatment and repeated as and when indicated and at the time of discharge. Data collected from the patients attenders included age, sex, occupation, mode of exposure and type of insecticidal agent, duration between exposure and hospitalization, patient's past history, family history, personal history. Socioeconomic status was assessed according to Kuppuswami classification of socioeconomic status. The duration of hospital stay and outcome in hospital was documented. Depending on the severity of manifestations, patients were classified into three grades as mild, moderate and severe groups on the basic of grading for organophosphorus poisoning suggested by Senanayak et al.[6] Statistical analysis was done and values were expressed for chance of occurrence. Whenever association of attributes was done statistical test $\chi 2$ test, fisher exact test and standard error of difference between two proportions were applied and inference was drawn.

Results-

In our study we found that out of total 91 patients maximum were males 54.95% (vs females 45.05%). Maximum number of patients were in age group 21-30 years, the incidence was 54.94%. Male to Female ratio in present study was 1.219:1(table 1). Majority of patient were from Rural areas (72.53%) in our study, And ratio of Rural to Urban population was 2.64:1 (table 2). 76.92% Patients belongs to low socioeconomic status, and 23.08% Patients belongs to Middle class. No patients in our study was from higher class of society. Mode of exposure in this study was suicidal ingestion (93%) and accidental inhalation (7%) (Table 3). Out of 91 patients, nature of compound was identified in 60.43% patients and unidentified in 39.56% patients. These patients had characteristic sign and symptoms of Organophosphate compound Poisoning or the patient gave history that he had taken insecticide. Mortality was more among Monocrotophos group (14.28%) followed by Dimethoate (11.11%) and unidentified group (11.11%) (Table 4). Majority of patients were hospitalized after 4 hours of exposure. Mortality rate (7.4%) was least among the patients who presented earliest as compared to those who presented later. (Table 5). Majority of patients (52.74%), were hospitalized for 6-10 days (table 6). Commonest symptom was vomiting (49.45%) followed by sweating (48.35%), salivation (48.35%), Blurring of vision (47.25%), lacrimation (39.56%) (Table -7). Commonest sign was Smell of poison (93.40%) followed by Tachypnoea (86.81%), Altered consciousness (67.03%), Miosis (64.83%), Fasciculation (48.35%), Tachycardia (29.67%) and Bradycardia (26.37%) (Table 8). In our study 40.66% patients were in mild class of toxicity, 25.27% were in moderate and 34.07% were in severe class of toxicity. Maximum death occurs in severe class (90%) (Table 09). In our study 35.16% patients develops atleast one complication and 100 % death occurs in this class (table 10). In our study most common complication was pulmonary edema 27.47% and respiratory failure 17.58%.(table 11).The above table shows ECG manifestation recorded before administration of atropine treatment. Sinus tachycardia was most common ECG abnormality (29.67%). QTc prolongation was seen in 28.57% patients, sinus bradycardia was seen in 26.37% patient. Total of 26 patients have QTc prolongation (table 12). Among QTc prolonged patients, 12 (46.15%) had severe poisoning and 8 patients (30.76%) expired. Among normal QTc interval patients 19(29.23%) had severe poisoning and only 2 (3.07%) expired. This difference in mortality is statistically significant ($\chi 2$= 10.67, P <0.001). In QTc prolonged group 18 (69.23%) patients developed at least 1 complication and 14 (21.53%) patient in normal QTc group also developed complication. This difference was statistically significant. ($\chi 2$=7.89, P<0.05). Association between severity of poisoning and QTc interval prolongation is not significant in our study ($\chi 2$=7.89, P>0.05) (table 13). In present study total 21 patients were hypokalemic .In hypokalamic group 12 (57.14%) patients developed at least 1 complication and 14 (20.58%) patient in normokalamic group also developed complication. This difference was statistically significant. ($\chi 2$=5.004, P<0.025). Association between severity of poisoning and QTc hypokalamia is not significant in our study ($\chi 2$=0.093, P>0.75). Among hypokalamic patients 3 patient (14.28%) expired. Among normokalamic patient 7 (10.29%) expired. This difference in mortality is statistically not significant (χ^2=0.093,P=0.75)(table 14) QTc prolongation was seen in 12 patients (75%) with severe group, 12 patients (33.33%) of moderate group and only 2(5.12%) patient of mild poisoning had QTc prolongation. This difference was statistically significant. Complications were not observed in mild cases. 11(30.55%) patient of moderate group had at least one complication. 14 (87.5%) patients of severe group had at least 1 complication. 90% deaths occurred in severe group.(table 15)

Table 1: showing age and sex wise distribution of study population

Age Group (Yrs)	Male	Female	Total
12-20	11(12.08%)	9(9.89%)	20(21.98%)
21-30	**28(30.76%)**	**22(24.17%)**	**50(54.94%)**
31-40	7(7.69%)	5(5.49%)	12(13.18%)
41-50	3(3.29%)	5(5.49%)	8(8.79%)
51-60	1(1.09%)	0(0%)	1(1.09%
Total	**50(54.95%)**	**41(45.05%)**	**91(100%)**

Table 2: showing place distribution

Place	Male	Female	Total
RURAL	37(40.65%)	29(31.86%)	66(72.53%)
URBAN	13(14.28%)	12(13.19%)	25(27.47%)
total	50(54.95%)	41(45.05%)	91(100%)

Table 3: showing patients distribution as per socio economic status

Group	Number of cases
Low	70(76.92%)
Middle	21(23.08%)
High	0(0%)

Table 4: showing distribution of patients as per type of poison consumed

Type of compound	Number of Cases	Death
Monocrotophos	28(30.76%)	4(14.28%)
Dimethoate	18(19.78%)	2(11.11%)
Diazinon	5(5.49%)	0(0%)
Melathion	2(2.19%)	0(0%)
Parathion	2(2.19%)	0(0%)
Unidentified	36(39.56%)	4(11.11%)
Total	**91(100%)**	**10(10.99%)**

Table 5: duration from exposure to hospitalization

Time in hours	Number of cases	Mortality
< 2 hours	27(29.67%)	2(7.40%)
2-4 hours	22(24.17%)	2(9.09%)
>4 hours	42(46.15%)	6(14.28%)

Table 6: total stay in hospital

Number of days	Number of cases
0-5	28(30.76%)
6-10	48(52.74%)
11-15	14(15.38%)
>15	1(1.09%)
Total	91(100%)

Table 7: presenting symptoms

Symptoms	Number Of Cases
Vomiting	45(49.45%)
Sweating	44(48.35%)
Salivation	44(48.35%)
Blurring Of Vision	43(47.25%)
Lacrimation	36(39.56%)
Breathlessness	36(39.56%)
Urinary Incontinence	13(14.28%)
Convulsions	12(13.18%)
Fecal Incontinence	6(6.59%)

Table 8: clinical signs

SIGNS	NUMBER OF CASES
SMELL OF POISON	85(93.40%)
Tachypnoea	79(86.81%)
Altered Consciousness	61(67.03%)
Miosis	59(64.83%)
Fasciculation	44(48.35%)
Tachycardia	27(29.67%)
Bradycardia	24(26.37%)

Table 9: showing distribution of patients as per severity of toxicity-

Patients having	Number of patients	deaths
Mild toxicity	37(40.66%)	oo
Moderate toxicity	23(25.27%)	01(10%)
Severe toxicity	31(34.07%)	09(90%)
Total number of patients	**91(100%)**	**10(100%)**

Table 10: showing distribution of patients as per presence of complications –

Patients having	Number of patients	Death
Complications	32(35.16%)	10
Not having complications	59(64.84%)	00
Total	**91(100%)**	**10**

Table 11: Showing Various Complications in Patients of Acute Organophosphorous Poisoning

Complication	Number of patients	Percentage
Pulmonary edema	25	27.47
Respiratory failure	16	17.58
Aspiration pneumonia	14	15.38
Intermediate syndrome	4	4.39

Table 12: showing observed ecg changes

Ecg Changes	Number Of Patients
Rate	
Normal	37(40.65%)
Sinus Tachycardia	27(29.67%)
Sinus Bradycardia	24(26.37%)
Rhythm	
Sinus Rhythm	88(96.70%)
Arrhythmia	3(3.29%)
Conduction Defect	
Prolonged Pr Interval	5(5.49%)
St Segment And T Wave Changes	
St Elevation	0(0%)
St Depression	8(8.79%)
T Wave Inversion	9(9.89%)
T Wave Flattening	5(5.49%)
Qtc Interval Prolongation	26(28.57%)

Table 13: showing comparing severity and mortality among normal qtc and qtc prolonged patients

Number of patients	Prolonged QTc		Normal QTc		P value
	n =26	%	n =65	%	
Complications	18	69.23	14	21.53	χ^2=7.89,P=0.005
Severe poisoning	12	46.15	19	29.23	χ^2=1.108,P=0.29
Death	8	30.76	2	3.07	χ^2=10.67,P=0.001

Table 14: showing comparing severity and mortality among normokalamic and hypokalamic patients

Number of patients	Hypokalamia		Normokalamia		P value
	n =21	%	n =70	%	
Complications	12	57.14	20	20.58	χ^2=5.004,P=0.025
Severe poisoning	3	14.28	28	17.68	χ^2=0.093,P=0.75
Death	3	14.28	7	10.29	χ^2=0.2008,P=0.654

Table 15: showing comparison of mild, moderate and severe groups of poisoning

	Mild	Moderate	Severe	
Patients with	37	23	31	
Prolonged QTc	2	12	12	P < 0.001 (fisher exact test)
Complications	0	11	14	P < 0.0001 (fisher exact test)
Death	0	1	9	P < 0.0001 (fisher exact test)

Discussion

Of various agents used for suicidal attempts in India, Organophosphorus Compounds form a significant group. This is peculiar to developing countries like India. In the developed countries 80% of suicidal poisoning result from intake of sedatives, antidepressants and related agents. In our hospital statistics, more than 50% of the cases were due to Organophosphorus compounds. Most affected age group as per group previous studies [7,8] is less than 30 years so is in our study. Males are more affected by O P poisoning/toxicities as per past [9] and present study. Our institute is attended mainly by Rural population so O P poisoning was present mainly in rural population in our study, This is discordant finding as per previous study [10].

Most patients in our study belongs to low socioecomic class, and past studies also have maximum patients from this group. [9,11] .Past studies [8,9] indicates that most common route of O P poising is ingestion and then inhalation so is in this study. In this study we observed that mortality was significantly higher in those patients who were hospitalized more than 4 hours after exposure vs within 2 hrs .most common encountered sign and symptoms (as enlisted in tables) are due to Acetylcholine excess in Neuromuscular and Synaptic junctions. In our study we found very high incidence of electrocardiographic changes and most significant finding was QTc prolongation and was associated with severe exposure and also death, This finding is also concordant with previous study done by Ludomirsky et al [12] in our study hypokalemia was most common electrolyte imbalance (23.01%) and this was also present in past studies [13,14,15] and hypokalemia was also associated with significant morality.

Conclusion

Organophosphorous Poisoning is major poisoning in india and early hospitalization and correction of Hypokalemia and QTc prolongation could be life saving in these patients.

References

1. V Palaniappen, Current concept in management of Organophosporus compound poisoning -API-medicine update vol. 23, 2013, chapter-95, and p427-433
2. Taylor P. Anticholinesterase agents. In: Goodman and Gilman's the Pharmacological Basis of Therapeutics. Ed. Hardman J G ,Limbard L E, Molinoff P B, Ruddon R W. 9th ed., p161-76.
3. 3 Jayaratnam J. Acute pesticide poisoning: A major global health problem. World Health Stat Q 1990; 43:139-144
4. Bardin P G, Van Eeden S F, Moolman J A, Foden A P, Joubert J R. Organophosphate and Carbamate poisoning. Arch Intern Med 1994; 154:1433-41.
5. Basavaraj G. Mangshetty, Niraj Saraf, Nagender Sharma. Study Of Clinical Profile Of Organophosphate Poisoning With Special Reference To Electrocardiographic Changes And Electrolyte Derangements.Jemds; Volume :3issue :67page :14514-14523: December2014:
6. Senanayake N,De Silva H J, Karalliedde L. A scale to assess the severity of organophosphorus intoxication: POP scale. Hum Exp Toxicol 1993; 12;297-299.
7. Mathur A., Swaroop A., Agarwal A. ECG changes in acute organophosphorus compound and almunium phosphide poisoning. The Indian Practitioner 1999; 52 ; 248-252.
8. Karki P, Ansari J A, Bhandary S, Koirala S. Cardiac and Singapore Med J Electrocardiographical manifestations of acute organophosphate poisoning. Singapore Med J 2004; 45:385-89.
9. Goel A., Joseph S. Dutta T.K Organophosphate poisoning: predicting the need for ventilator support. JAPI 1998; 46:786-790.
10. Adlakha A., Philip P.J., Dhar K.L. Organophosphate and carbamate poisoning in Punjab. JAPI 1988; 36:210-212.
11. Chaterjee D.C. Poisoning due to organophosphate insecticide. JIMA 1967; 48:163.
12. Ludomirsky A, Klein H, Sarelli P, Becker B, Hoffman S, Taitelman U et al. Q-T prolongation and polymorphous (torsades de pointes) ventricular arrhythmias associated with organophosphorus insecticide poisoning. Am J Cardiol 1982; 49: 1654-8.
13. Dilek soysal, Volkan karakus, Ahmet Soysal, Erhan Tator,Bayram Yildiz, Hatice Simsek, Evaluation of caseswith acute OP poisoning, JAEM 2011;10:156 60.
14. 14 S. Laudari, B.S. Patowary, analysis of organophorus compound poisoning patient attending CMS-TH, Journal of College of Medical sciences- Nepal. 2011, vol-7, No. 4, 9-19.
15. 15 Dr. M. S. Sagunadevan, Dr. A. Warnakulasuria, Audit on organophosphate poisoning requiring intensive care unit admission, SAARC Journal of Anaesthesia, July 2008;1(2) :p-112-115.