HYPOKALEMIA IN ORGANOPHOSPHORUS COMPOUND POISONING-CASE CONTROL STUDY

Dr. K.A. Sharath Chandra [1] , Dr. Ravi Shankar.V [2]

1, Junior Resident, Department of Emergency Medicine Sree Mookambika Institute of Medical Sciences College Kanyakumari, Tamil Nadu, India.

2.Associate Professor, Department of Emergency Medicine, Sree Mookambika Institute of Medical Sciences Kanyakumari, Tamil Nadu, India.

Corresponding Author: Dr. K.A. Sharath Chandra ,Junior Resident, Department of Emergency Medicine Sree Mookambika Institute of Medical Sciences College Kanyakumari, Tamil Nadu, India.

ABSTRACT :

Background: organophosphorus compound(opc) poisoning is very common in india and it is a very important cause of morbidity and mortality in such patients. many factors determine the outcome in opc poisoning. but so far, there are no properly conducted case control studies to analyse individual parameters in opc poisoning. hypokalemia is common in this scenario, and case reports are available which quote its significance.

Aim: In this study of 50 patients, the impact of hypokalemia in OPC poisoning, has been studied.Diseases or drugs that can modify serum potassium levels have been excluded from the study.

Results: In this study hypokalemia was associated with increased mortality and prolonged duration of mechanical ventilation, both of which are statistically significant with p values of 0.039 and 0.037 respectively.

Conclusion: Hypokalemia can be used as a reliable and cost effective marker of worse outcomes in the setting of OPC poisoning.

Keywords: OPC poisoning,Hypokalemia,mechanical ventilation,

INTRODUCTION:

Organophosphorus compounds (OPCs) are common pesticides used in agriculture in India. Inappropriate handling, easier availability, and lack of adequate knowledge contribute to increased incidence of poisoning with these compounds in India which are also responsible for associated worse outcomes. Poisoning holds fourth position in leading causes of death in India. According to statistics given by WHO, approximately a millioncases of accidental and about 2 million cases of suicidal attempts usinginsecticides occur worldwide annually.
Many factors influence outcome in OPC poisoning like severity of poisoning, development of respiratory failure, availability of mechanical ventilation and so on.
In the current study, i am trying to assess hypokalemia in OPC poisoning and its impact on the outcome.

AIM AND OBJECTIVES OF THE STUDY:

- To assess hypokalemia in the setting of OPC poisoning
- To correlate hypokalemia with various clinical parameters and outcome.atropine, pralidoxime and respiratory failure.

MATERIALS AND METHODS:

Patients admitted in the emergency department those who are consumed organophosphorus poisoning included in this study. Inclusion criteria are Patients admitted with history of organophosphorus compound poisoning.Exclusion criteria are Known kidney disease patients, heart disease patients, patients on diuretics.

Informed consent was obtained from each patient or the relative,Patients had their history taken according to a Questionnaire and were subjected to clinical examination,Renal function tests were done in all patients,All the data were entered in the proforma(enclosed),SPSS package and ANOVA was used to analyse the data.

Statistical analysis was done using the statistical package for social sciences (SPSS).Different statistical methods were used as appropriate. Mean ± SD was determined for quantitative data and frequency for categorical variables. The independent t- test was performed on all continuous variables. The normal distribution data was checked before any t-test. The Chi-Square test was used to analyze group difference for categorical variables.A p- value < 0.05 was considered significant.

RESULTS:

CHI-SQUARE TESTS

SEX AND SERUM POTASSIUM LEVELS

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)	P value
Pearson Chi-Square	.765(b)	1	.382			
Continuity Correction(a)	.284	1	.594			
Likelihood Ratio	.774	1	.379			.382
Fisher's Exact Test				.501	.298	
Linear-by-Linear Association	.750	1	.387			
N of Valid Cases	50					

CHI-SQUARE TESTS

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)	P value
Pearson Chi-Square	4.276(b)	1	.039			

Square						
Continuity Correction(a)	3.072	1	.080			
Likelihood Ratio	4.369	1	.037			.039
Fisher's Exact Test				.059	.039	
Linear-by-Linear Association	4.191	1	.041			
N of Valid Cases	50					

CHI-SQUARE TESTS

Value	df		Asymp. Sig. (2-sided)	P value
12.765(a)	14		.545	.545
Likelihood Ratio	17.393	14	.236	
N of Valid Cases	50			

SERUM POTASSIUM LEVELS AND DURATION OF MECHANICAL VENTILATION (IN DAYS)

	Serum potassium levels meq/dl	N	Mean	Std. Deviation	Std. Error Mean
Duration of mechanical ventilation days	Hypokalemia	24	2.79	2.449	.500
	Normal	26	1.42	2.062	.404

In this study, totally 15 different poisons were involved. Of 24 cases of hypokalemia, monochrotophos is responsible for maximum number of cases being involved in 8 cases followed by chlorpyriphos in 6 cases.Acephate, anilphos, diazinon, dimethoate, phosphomidon, and temephos did not cause hypokalemia in any cases. However this increased incidence with individual poisons are not statistically significant(p value- 0.545).

Of 50 patients totally, 14 had hyponatremia(<135 meq/dl) which constitutes 28%. Of this 6 patients had hypokalemia which accounts for 42.9% of hyponatremia. Of 24 patients with hypokalemia this 6 cases of hyponatremia accounts for 25%. This association is not significant statistically (p value- 0.650). Hypernatremia did not occur in any patient.

In the current study mean duration of hospital stay in hypokalemia patients was 6.29 days, whereas in patients with normal potassium was 6.08 days. Hypokalemia did not prolong hospital stay. (p value- 0.802).

Out of 50 patients, 28 patients needed mechanical ventilation.All 14 patients who died had required mechanical ventilation. Of 28 patients needed mechanical ventilation, 18 patients had hypokalemia.The mean duration of mechanical ventilation in hypokalemia patients was 2.79 days which is almost double as the mean duration in patients with normal potassium which was 1.42 days. This prolongation in mechanical ventilation was statistically significant (p value- 0.037).

DISCUSSION:

In Lyzhnikov EA et al Study, severe arrhythmia and cardiac arrest leadind to death occurred in 29 patients who are found to have hypernatremia and hypokalemia. In D.R.Mahadeshwara Prasad et al study, death occurred in patients with a mean potassium levels of 2.90 +/- 0.057 meq/dl(p value < 0.001). In the current study, out of 14 deaths totally, 10 patients(71.4%) had hypokalemia .

In Lyzhnikov EA et al Study, patients had poisoning with chlorophos, carbophos ,and thiophos.

In D.R.Mahadeshwara Prasad et al study, significance to individual poison was not analysed. In the current study there is no statistically significant association between an individual poison and hypokalemia.

In the current study, 14 patients had hyponatremia(<135 meq/dl).Of which, 6(41.6%) had hypokalemia but this was not significant statistically.(p value-0.650) None of the patients had hypernatremia. In D.R.Mahadeshwara Prasad et al study sodium disturbance was not studied. In Lyzhnikov EA et al Study, hypernatremia was associated with hypokalemia and icreased mortality.

In the current study mean duration of hospital stay in hypokalemia patients was 6.29 days, whereas in patients with normal potassium was 6.08 days. Hypokalemia did not prolong hospital stay.(p value-0.802).
In both previously done studies, duration of hospital stay was not assessed.

In D.R.Mahadeshwara Prasad et al study, both respiratory distress and mechanical ventilation taken together and statistically significant(p value < 0.001). In the current study, mean duration of mechanical ventilation was prolonged(p value-0.037). In Lyzhnikov EAet alStudy, mechanical ventilation was not assessed.

CONCLUSION:

Seasonal variation was present in snake bite.Incidence and mortality more during January, Hypokalemia increases both morbidity and mortality in organophosphorus compound poisoning significantly.Hypokalemia can be used as a reliable and a cost effective marker of morbidity and mortality in organophosphorus compound poisoning.

LIMITATIONS OF THE STUDY

- Because there is no randomisation, the influence of confounding factors like infection, and development of multi organ dysfunction cannot be eliminated.
- As included in the criteria, patients with renal failure have been excluded. So electrolyte disturbance in this population cannot be assessed.
- Number of female patients in the study group is small.

BIBLIOGRAPHY

1. Gunnell D, Eddleston M, Phillips MR, Konradsen F. The global distribution of fatal pesticide self-poisoning: systematic review. *BMC Public Health.*2007;7:357.[PMID: 18154668]
2. Eddleston M. Patterns and problems of deliberate self-poisoning in the developing world. *Q J Med.* 2000;93:715-731.[PMID: 11077028]
3. Eyer F, Meischner V, Kiderlen D, et al. Human parathion poisoning. A toxicokinetic analysis.*Toxicol Rev.* 2003;22:143-163.[PMID: 15181664]
4. Gallo MA, Lawryk NJ. Organic phosphorus pesticides. In: Hayes WJ, Laws ER, eds. *Handbook of Pesticide Toxicology*. San Diego, CA: Academic Press; 1991:917-1123.
5. Buratti FM, Volpe MT, Meneguz A, Vittozzi L, Testai E. CYP- specific bioactivation of

four organophosphorothioate pesticides by human liver microsomes. *Toxicol Appl Pharmacol.* 2003;186:143- 154.[PMID:12620367]
6. Thiermann H, Szinicz L, Eyer P, Zilker T, Worek F. Correlation between red blood cell acetylcholinesterase activity and neuromuscular transmission in organophosphate poisoning. *Chem Biol Interact.* 2005;157- 8:345-347.[PMID: 16263103]
7. Casida JE, Quistad GB. Organophosphate toxicology: safety aspects of nonacetylcholinesterase secondary targets. *Chem Res Toxicol.*2004;17:983-998.[PMID: 15310231]
8. Eddleston M, Eyer P, Worek F, et al. Differences between organophosphorus insecticides in human selfpoisoning:
9. a prospective cohort study. *Lancet.*2005;366:1452-1459.[PMID: 16243090]
10. Namba T, Nolte C, Jackrel J, Grob D. Poisoning due to organophosphate insecticides. *Am J Med.* 1971;50:475-492.[PMID: 4324629]
11. Hruban Z, Schulman S, Warner NE, Du Bois KP, Bunnag S, Bunnag SC. Hypoglycemia resulting from insecticide poisoning. Report of a case.*JAMA.*1963;184:590-593.[PMID: 13955258]
12. Harputluoglu MMM, Kantarceken B, Karincaoglu M, et al.
13. Acute pancreatitis: an obscure complication of organophosphate intoxication.
14. *Hum Exp Toxicol.* 2003;22:341-343.[PMID: 12856957]
15. Pach D.The usefulness of scintigraphic examination for the evaluation of hepatotoxic impact of cholinesterase inhibitors.*Przegl Lek.* 1996;53: 313-323.[PMID: 8711183]
16. Roth A, Zellinger I, Arad M, Atsmon J. Organophosphates and the heart. *Chest.*1993;103:576-582.[PMID:8432156]
17. Senanayake N, Karalliedde L. Neurotoxic effects of organophosphate insecticides: an intermediate syndrome.
18. *N Engl J Med.* 1987;316:761-763.[PMID: 3029588]
19. Wadia RS, Bhirud RH, Gulavani AV, Amin RB. Neurological manifestations of three organophosphate poisons.
20. *Indian J Med Res.* 1977;66: 460-468.[PMID: 598919]
21. Karalliedde L, Baker D, Marrs TC. Organophosphate-induced intermediate syndrome: aetiology and relationships with myopathy. *Toxicol Rev.* 2006;25:1-14.[PMID: 16856766]
22. Benson B, Tolo D, McIntire M. Is the intermediate syndrome in organophosphate poisoning the result of insufficient oxime therapy? *J Toxicol Clin Toxicol.*1992;30:347.
23. Thiermann H, Szinicz L, Eyer P, Zilker T, Worek F. Correlation between red blood cell acetylcholinesterase activity and neuromuscular transmission in organophosphate poisoning. *Chem Biol Interact.* 2005;157- 8:345-347.[PMID: 16263103]
24. Thiermann H, Szinicz L, Eyer P, Zilker T, Worek F. Correlation between red blood cell acetylcholinesterase activity and neuromuscular.