



ORIGINAL RESEARCH PAPER

A STUDY OF HYPOKALEMIA IN ORGANOPHOSPHORUS POISONING IN DAHOD GUJARAT

General Medicine

KEY WORDS: Hypokalemia,
OPC ,Mortality

Dr.Mohit Desai

Assistant Professor, Department Of Medicine, Zmch,dahod,gujrat,pin-389151.

Dr.Brajendra kumar*

Professor & Hod, Department Of Medicine, Zmch,dahod,gujrat,pin-389151.
*Corresponding Author

ABSTRACT

Organophosphorus compounds (OPCs) are common pesticides used in agriculture in India. In this study, totally 24 patients had hypokalemia. Of 39 male patients, 20 patients (51.3%) had hypokalemia. Of 11 female patients, 4 patients(36.4%)had hypokalemia. 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%. 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.

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 million cases of accidental and about 2 million cases of suicidal attempts using insecticides 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 our study, we tried to assess hypokalemia in OPC poisoning and its impact on the outcome.

MATERIAL AND METHODS

Our cross sectional study was conducted at ZMCH DAHOD GUJRAT in 2019 for 6 months. Patients admitted with history of organophosphorus compound poisoning in toxicology ward, Institute of Internal medicine. Fifty cases admitted with history of organophosphorus compound poisoning.

INCLUSION CRITERION:

Patients admitted with history of organophosphorus compound poisoning.

EXCLUSION CRITERIA:

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.

RESULTS

Of 50 patients studied, 24 patients had hypokalemia(serum potassium levels <3.6meq/dl) and 26 had normal serum potassium levels. The mean age for patients with hypokalemia was 38.38 and mean age for other was 34.12. This difference was statistically not significant(p value- 0.278). In this study, totally 24 patients had hypokalemia. Of 39 male patients, 20 patients (51.3%) had hypokalemia. Of 11 female patients, 4 patients(36.4%)had hypokalemia. This difference of 14.9% of increased incidence in male patients was not statistically significant.(p value0.382). In this study, totally 24 patients had hypokalemia which accounts for 42.9% of hyponatremia. Of 24 patients with hypokalemia this 6 cases of hyponatremia accounts for 25%. 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.

in male patients was not statistically significant.(p value0.382). 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.

DISCUSSION

In D.R.Mahadeshwara Prasad et al study, the author studied relationship between serum potassium levels with muscle twichings & fasciculations, respiratory distress, mortality and convulsions in 50 patients. In Lyzhnikov EA et al study, the relationship between plasma and RBC levels of potassium and sodium with mortality and ECG was analysed in 73 patients with OPC poisoning. Mean age of the patients in D.R. Mahadeshwara Prasad et al study was 27.14 years. In current study mean age for patients with normal potassium was 34.12 years and for patients with hypokalemia was 38.38 years.. In Lyzhnikov EA et al Study age difference was not studied. In D.R.Mahadeshwara Prasad et al study and Lyzhnikov EA et al Study the significance of gender and hypokalemia was not studied. In the current study, of 24 patients with hypokalemia, 20 were male patients and 4 were female patients. 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

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.

REFERENCES

- (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. *QJM*.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. CYPspecific 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
- (7) Casida JE, Quistad GB. Organophosphate toxicology: safety aspects of nonacetylcholinesterase secondary targets. *Chem Res Toxicol*.2004;17:983-998.[PMID:5310231]
- (8) Eddleston M, Eyer P, Worek F, et al. Differences between organophosphorus insecticides in human selfpoisoning:a prospective cohort study.*Lancet*. 2005; 366:1452-1459.[PMID:16243090]
- (9) Namba T, Nolte C, Jackrel J, Grob D. Poisoning due to organophosphate insecticides. *Am J Med*. 1971;50:475-492.[PMID:4324628]
- (10) 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].