

Original Research Article

A study of serum amylase levels in acute organophosphorous poisoning at Government Dharmapuri Medical College Hospital, Dharmapuri

P. Elango¹, G. Indumathi^{2*}

¹Assistant Professor, ²Assistant Professor

Department of General Medicine, Government Dharmapuri Medical College Hospital, Dharmapuri, India

*Corresponding author email: drindugo@gmail.com

	International Archives of Integrated Medicine, Vol. 4, Issue 9, September, 2017. Copy right © 2017, IAIM, All Rights Reserved. Available online at http://iaimjournal.com/ ISSN: 2394-0026 (P) ISSN: 2394-0034 (O)
	Received on: 12-08-2017 Accepted on: 17-08-2017 Source of support: Nil Conflict of interest: None declared.
How to cite this article: P. Elango, G. Indumathi. A study of serum amylase levels in acute organophosphorous poisoning at Government Dharmapuri Medical College Hospital, Dharmapuri. IAIM, 2017; 4(9): 6-11.	

Abstract

Introduction: In India, OP compounds cause more self-poisoning deaths in southern and central India. In Northern India, aluminum phosphide causes most deaths with a fatality ratio. Poisoning due to occupational exposure, accounted for about one fifth of the incidents, with a fatality ratio of less than 1%. More than 90% of the non-occupational incidents were suicidal, with a fatality rate more than 10% and the majority of the subjects are young males. Accidental exposures accounted for 8-10% of the incidents and homicidal use (less than 1%) were other forms of poisoning. The reported overall mortality following OP insecticide poisoning varies from 4-30% in different countries and institutions.

Aim: To estimate serum Amylase levels in acute organophosphorus compound poisoning and the find out the clinical outcomes.

Materials and methods: The study was conducted in Government Dharmapuri Medical College Hospital, Dharmapuri. Study duration was from January 2017 to June 2017. Of a total of 145 patients with organophosphorus compound poisoning admitted to the hospital during the study period, 40 were included in the study. 10 healthy (age matched) individuals were kept as a control. Biochemical evaluation which includes Serum Amylase Blood glucose, urea, creatinine, and Liver function tests were analyzed and matched among the two groups.

Results: The biochemical results have not shown much variation from the normal levels in our study.

In the study, the Amylase levels were significantly elevated at the time of admission (185.2 U/L) and have shown a gradual remission with proper treatment. The mean Amylase level in severely poisoned patients was 297.7 U/L which was significantly ($P < 0.01$) higher than the healthy control group. On comparing the Amylase levels in first 24 hours against control, the variations were considered to be significant ($P < 0.01$).

Conclusion: From the observation we made, it could be suggested that OP pesticide poisoning is a serious condition that needs rapid diagnosis and treatment. The mean Amylase level in first 24 hours of OP poisoning was 154 U/L which is significantly higher than the control groups.

Key words

Organophosphorus Poisoning, Serum Amylase, Blood sugar, Serum creatinine and urea.

Introduction

Acute poisoning by organophosphorus Pesticides (OP) has reached epidemic proportions in most parts of the world, particularly in developing agrarian countries, where the toxicity of available poisons and the paucity of appropriate medical facilities ensure a high fatality rate [1]. Their ease of access and socio-cultural factors plays important role in choice of OP as a self-poison and the incidence is higher in a young economically active group with a common fatality ratio of 20%. According to WHO, worldwide estimates of pesticide poisoning number 3 million each year, with 2 million hospitalized from suicide attempts and 2,20,000 deaths, the majority of which are actually intentional [2]. Poisoning due to occupational exposure, accounted for about one fifth of the incidents, with a fatality ratio of less than 1%. More than 90% of the non-occupational incidents were suicidal, with a fatality rate more than 10% and the majority of the subjects are young males. Accidental exposures accounted for 8-10% of the incidents and homicidal use (less than 1%) were other forms of poisoning. The reported overall mortality following OP insecticide poisoning varies from 4-30% in different countries and institutions [3]. In India, OP compounds cause more self-poisoning deaths in southern and central India. In Northern India, aluminium phosphide causes most deaths with a fatality ratio of 90%. Other Pesticides used for self-poisoning includes carbamates, Organochlorines, and pyrethroids. Organophosphorus compounds are principally used as pesticides, and their exposure

is highly prevalent in developing countries [4]. Toxic effects of OPs are associated with significant morbidity and mortality and are a major global clinical problem. Occupational, suicidal (or) homicidal exposure to OPs produces a characteristic but treatable syndrome in humans thus; early recognition and timely intervention of toxicity from these compounds are of great importance, to emergency physicians and patients [5]. Case reports of acute pancreatitis following acute organophosphorus compound ingestion have been reported now and then, but regular studies with reference to Pancreatitis is not available in a serial manner. Hence an attempt was made to study Pancreatic involvement through biochemical means [6]. The exact mechanisms of serum amylase metabolism are still not fully understood. Humans who have had a nephrectomy or have renal insufficiency have average serum amylase levels 50% higher than healthy individuals. Therefore, kidneys can be assumed to play a major role in amylase metabolism. However, the kidney is not the sole organ responsible for amylase clearance in humans [7]. The extrarenal mechanisms of amylase clearance have not been defined. Because of the high serum amylase levels also observed in hepatic necrosis and cirrhosis, the liver is thought to play a role in amylase metabolism. Many conditions have been reported to cause hyperamylasemia. Although hyperamylasemia is commonly assumed to be due to the release of amylase into the serum by the diseased organ, the precise relationship

between hyperamylasemia and an affecting condition is not entirely clear [8].

Materials and methods

The study was conducted in Government Dharmapuri Medical College Hospital, Dharmapuri. Study duration was from January 2017 to June 2017. Of a total of 145 patients with organophosphorus compound poisoning admitted to the hospital during the study period. 40 were included in the study. 10 healthy (age matched) individuals were kept as a control. Biochemical evaluation which includes Serum Amylase Blood glucose, urea, creatinine, and Liver function tests were analyzed and matched among the two groups. The Ethical committee approval was obtained to carry out the study in the hospital. Patients admitted in GRH were the study group. A previously designed proforma was used to collect the demographic and clinical details of the patients.

Inclusion criteria

40 patients with a history of exposure to OP poison were the study subjects.

Exclusion criteria

- Patients with indication of exposure to an entirely different poison other than OP poison.
- Patients with double poisoning
- Patients who have consumed poison along with alcohol.
- Patients who are chronic alcoholics
- Patients with a history suggestive of gall stone disease 6. Patients with known history of lipid disorders.

Sample collection

40 Patients satisfying the inclusion criteria were selected for the study. About 3 ml of venous blood were collected on two occasions from each subject first within 24 hours of consumption of poison (Sample I) and next after 24 hours of the first sample (Sample II). The samples were centrifuged at 3000 rpm for 15 minutes. The supernatant serum was separated and froze.

Serum Amylase was estimated with the help of kit manufactured by Diasys Diagnostic Systems GmbH Alte S Strasse g 65558 Holyheim Germany by using CNP-G3 method Autoanalyser AUTOPAK. Biochemical evaluation which includes Serum Amylase Blood glucose, urea, creatinine, and Liver function tests.

Statistical analysis

Data analysis was done with the help of computer using Epidemiological. Information Package (EPI 2002). Using this software, frequencies, percentages, means, standard deviations, chi square 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 test for qualitative variables. A 'p' value less than 0.05 is a relationship.

Results

Hematological parameters changes in organophosphorous poisoning were as per **Table – 1**. Clinical features and serum amylase levels in changes in organophosphorous poisoning was as per **Table – 2**. Outcome and amylase level was as per **Table – 3**.

Discussion

Organophosphates and Carbamates are frequently used pesticides which can produce life-threatening intoxication. Well over 50,000 organophosphorus compounds have been synthesized since the first one by Clermont in 1857. All these compounds act by irreversible inactivation of acetylcholinesterase (ACh) [10]. The clinical symptoms range from the classic cholinergic syndrome to flaccid paralysis and intractable seizures. About 99% of fatal poisoning occurs in developing countries, particularly among farm workers. Despite an increased incidence of organophosphorus insecticide poisoning, the exact micro molecular changes that take place remain elusive. Till date, atropine and oxime continue to occupy the prime position in the specific management of OP

poisoning [11]. With the ease of availability, it is not surprising that the use of OP compounds in suicide attempts has mushroomed from a disturbing early trend to being one of the commonest modes of suicidal poisoning which accounted for 100% in our study. This rate was consistent with other study (94.3%) whereas it was reported to be 67%. There was no accidental exposure in our study [12]. This alarming incidence of suicidal attempts may be probably because of the uncontrolled sale and use of these agents all over the country. The vast majority of poisonings followed oral ingestion of liquid form and almost for all patient's gastric lavage was immediately done [13]. The incidence was higher (40%) in the age group of 21-30 followed by (35%) in the age group of 31-40. The accumulation of ACh in nerve terminals results in continued stimulation with subsequent paralysis of receptors and accounts for the clinical signs of muscarinic, nicotinic and CNS effects. Present study found an association

between the severity of poisoning and clinical manifestations. The most marked muscarinic signs in our study population were miosis (55%), excessive secretions (60%), and respiratory distress (25%) [14]. The most prominent of the nicotinic effect is muscular end plate block, resulting in muscle weakness and fasciculation's (30%). The CNS symptoms, like depressed mental status, were found in (27.5%) patients. OP insecticides increase the intraductal pressure and exocrine pancreatic flow. The increase in pressure leads to extravasation of pancreatic fluid. This increased pancreatic exocrine flow could be due to direct cholinergic hyper stimulation of pancreatic acinar and ductal cells. In the study, the Amylase levels were significantly elevated at the time of admission (185.2 U/L) and have shown a gradual remission with proper treatment. The mean Amylase level in severely poisoned patients was 297.7 U/L which was significantly ($P < 0.01$) higher than the healthy control group [14].

Table - 1: Hematological parameters changes in organophosphorous poisoning.

Parameters	Case		Control		P
	Mean	SD	Mean	SD	
Blood sugar	96.9	24.2	102.3	11.2	0.2535 Not significant
Blood urea	29.8	7.4	31.4	5.0	0.5035 Not significant
Serum creatinine	0.95	0.3	0.83	0.21	0.281 Not significant

On comparing the Amylase levels in first 24 hours against control, the variations were significant ($P < 0.01$). From our observation, it can be suggested that estimation of Amylase levels would be extremely useful to assess the clinical severity.

Age and sex of the patients have no significant relationship with the amylase levels. The mean Amylase level in first 24 hours was 154 U/L which is significantly higher than the control groups. In our study, there was no significant correlation between elevated Amylase levels and the outcome. From the observation, we made, it could be suggested that OP pesticide poisoning is a serious condition that needs rapid diagnosis and treatment [15].

Conclusion

Our study also showed that there was a significant correlation between markedly elevated Amylase level and respiratory failure and therefore poor outcome. A significant rise in Serum Amylase level also portends various complications that include convulsions, CNS depression, fasciculations and respiratory failure. However, as the study was limited to a small population due to financial and laboratory constraints, analysis of a larger group would give an insight into the further finer relationship between serum amylase level and clinical severity and outcome in OP poisoning.

Table – 2: Clinical features and serum amylase levels in changes in organophosphorous poisoning.

Clinical features	Amylase levels					
	I		II		I – II	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Pinpoint pupil						
Present	204.1	136.1	52.4	33.2	151.1	111.8
Absent	67	38.5	25.4	25.1	41.6	27.6
‘p’	0.0001 Significant		0.0009 Significant		0.0001 Significant	
Depressed mental status						
Yes	261	151.8	63	31	198	131.1
No	97.4	75.6	31.7	29	65.8	54.9
‘p’	0.0003 Significant		0.0023 Significant		0.0004 Significant	
Secretions						
Mild	83	59.2	30.3	25.7	52.7	33.8
Moderate	108.9	90.6	30.3	26.8	78.6	67.4
Severe	242.2	157.5	59.7	32.3	182.5	135.2
NS	84.5	72.8	67.5	67.2	17	5.7
‘p’	0.0168 Significant		0.0219 Significant		0.0062 Significant	
Fasciculation						
Present	272.3	149.9	67.5	33.4	204.8	127.2
Absent	86.6	50.8	29	24.7	57.6	17.5
‘p’	0.0001 Significant		0.0001 Significant		0.0001 Significant	
Heart Rate						
Bradycardia	209.1	142.8	51.1	31.8	157.9	119.5
Tachycardia	-	-	-	-	-	-
Normal	93.3	83.4	33	31.7	60.4	59.8
‘p’	0.0001 Significant		0.0321 Significant		0.0001 Significant	
Convulsions						
Present	156	-	38	-	118	-
Absent	142.1	126.9	40.3	32.9	101.7	102
‘p’	-		-		-	
Respiratory Failure						
Yes	297.7	151.8	69.8	36.4	227.9	126.7
No	90.6	50.8	30.4	24.6	60.2	37.3
‘p’	0.0001 Significant		0.0016 Significant		0.0001 Significant	

Table – 3: Outcome and Amylase levels.

Outcome	Amylase levels					
	I		II		I – II	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Alive	134.6	122	39.9	33.4	94.7	96.3
Dead	213	142	44	25.3	169	130.7
‘p’	0.1762 Not Significant		0.443 Not Significant		0.1428 Not Significant	

References

1. M. Eddleston, L. Sinicize, P. Eyer. Oximes in acute organophosphorus pesticide poisoning: a systematic review of clinical trials. *QJ Med. J.*, 2002; 275 – 283.
2. M.A. Cherian, C. Roshini, J. Visalakshi. Biochemical and Clinical Profile After Organophosphorus Poisoning – A Placebo – Controlled Trial using Pralidoxime. *JAP1* May 2005; 53: 427-430.
3. Cholinergic Toxicity Syndrome. Accessed from <http://www.fpnotebook.com/Neuro/Pharm/ChlnrgcTxcty.htm>
4. Michotte A, Van Dijck I, Mals V. Organophosphorus Insecticide Poisoning. *JIFCC*, 1999; 11(2).
5. C.H. Srinivas Rao, V. Venkateswaralu, T. Surender. Pesticidepoisoning in South India: Opportunities for prevention and improved medical management. *Tropical Medicine and International Health*, 2005; 10(6): 581-588.
6. Extonet (Extension Toxicology Network). Toxicology Information Briefs – Cholinesterase Inhibition. Accessed from <http://pmep.cce.cornell.edu/profiles/extonet/TIB/cholinesterase.html>
7. K. D. Tripathi. *Essentials of Medical Pharmacology*, 4th Edition, p. 71, 83-85.
8. Vidyasagar J, Karunakar N. Reddy M.S. Oxidative Stress and antioxidant status in acute organophosphorus insecticide poisoning. *Indian Journal of Pharmacology*, 2004, 36(2): 76-79.
9. Gurayten Ozyrut, Hillaya Biligin, Melda Gedic Kutsal. Atropine Aerosol Spray (AAS) by Nasal Application in Organophosphate poisoning. Accessed from WWW.Spingerlink.com.
10. Hardman J.G., Limbird L.E., Molinoff. Goodman and Gilman's *The Pharmacological Basis of Therapeutics*, 12th edition, McGraw Hill, 2011.
11. Pesticide Illness. Accessed from Aoec.org/pesticide-illness/2-speaker-notes.doc.
12. Kushik Jaga, Chandrabhan Dharmani. Sources of exposure to and public health implications of organophosphate pesticides. *Rev. Panam Salud Publica.*, 2003; 14(3): 171-185.
13. Singh S, Sharma N. Neurological Syndromes following organophosphate poisoning. *Neurol Indian (Serial online)*, 2000; 48: 308-13.
14. Murat Sungur, Muhammed Given. Intensive care management of organophosphate insecticide poisoning. *Crit Care*, 2001; 5(4): 211-215.
15. K. Futagami, N. Tanaka, M. Nishimura. Relapse and elevation of blood urea nitrogen in Acute fenitrothion and malathion poisoning. *International Journal of clinical pharmacology and Therapeutics*, 1996; 34(10): 453-456.