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Original Research Paper

General Medicine

A STUDY OF SERUM AMYLASE LEVELS IN ACUTE ORGANOPHOSPHOROUS POISONING AT TERTIARY CARE HOSPITAL .

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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 Medical College and Hospital, Jammu. Study duration was from January 2018 to OCT.2018. Of a total of 350 patients with organophosphorus compound poisoning admitted to the hospital during the study period, 150 were included in the study. 50 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.

KEYWORDS : 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 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]. 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 [6,7]. 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 relationshipbetween hyperamylasemia and an affecting condition is not entirely clear [8].

Materials and methods

The study was conducted in Government Medical College Hospital, Jammu. Study duration was from January 2018 to October 2018. Of a total of 350 patients with organophosphorus compound poisoning admitted to the hospital during the study period. 150 were included in the study. 50 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. A previously designed proforma was used to collect the demographic and clinical details of the patients.

Inclusion criteria

1. Patients with a history of exposure to OP poison were the study subjects.
2. Age> 12 yrs, both sex

Exclusion criteria

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

Sample collection 150 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.

Table 1 & 2 depicts Age and Sex distribution in organophosphorous poisoning Serum amylase levels changes in organophosphorous poisoning as per Table – 3 & 4.

Clinical features in organophosphorous poisoning as per Table 5 & 6
Outcome and amylase level was as per Table – 7.

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 Organophosphorus 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%) .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].In current study majority of patients (78%) belonged to age group 26 to 55 yrs this is consistant with other studies [13,14]Males outnumbered the females.Among the total cases,100 (66.7)were the males and 50(33.3) were females.This data is similar to Present study found an association between the severity of poisoning and clinical manifestations. Age and sex of the patients have no significant relationship with the amylase levels. The most marked muscarinic signs in our study population were miosis (26.6%), excessive secretions (13.3%), and respiratory distress (18.66%) [14,15,16]. The most prominent of the nicotinic effect is muscular end plate block, resulting in muscle weakness and fasciculation's (16.7%). The CNS symptoms, like depressed mental status, were found in (13.33%) 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]. 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.[17,18]

However this hyperamylasemia in cases of OP poisoning, may be due to the fact that acute pancreatitis is caused by excessive cholinergic stimulation of pancreas by OP compounds [19,20]. Our study results were in accordance with the study done by Bhardwaj et al where they found that serum amylase is elevated in 47% of patients with organophosphorus poisoning [21]. In a prospective study by Singh S [22] amylase was elevated in 48.95% in patient with fenthion poisoning and serum amylase showed persistent elevation during serial estimation. Respiratory failure is the most common dreaded complication in organophosphorus poisoning leading to mechanical ventilation and death. In a study conducted in Japan by Sumiya et al [23], an increase in plasma amylase levels above the normal range have been found in 50% of the patients who developed respiratory failure. Lin CL et al., found that mean amylase levels were elevated in patients with respiratory support and serum amylase levels predicted ventilator support in OP poisoning [24]. Among the 150 patients in our study, death was reported in 6 (4%) patients and 28 (8.6%) patients survived after mechanical ventilation. The overall mortality in our study was 4% and this was compared with other studies [25].

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 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.

Conclusion

From the observation of present study, it may be concluded that the serum amylase levels considered as a marker of organophosphorous intoxication, since it enables the early recognition of severity and also helps to identify those at risk of developing the complications of Organophosphorous poisoning. In current study, age and sex of the patients have no significant relationship with the amylase levels but significant correlation found 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. However further analysis of a larger group would give an insight into the finer relationship between serum amylase level and clinical severity and outcome in OP poisoning.

TABLE NO: 1; AGE DISTRIBUTION

	AGE(Yrs)	Number of cases	Deaths
1	12 to 25	18	0
2	26 to 40	57	2
3	41 to 55	60	3
4	56 to 70	15	1
	TOTAL	150	6

TABLE NO: 2; SEX DISTRIBUTION

Gender	Cases	Deaths
Male	100	5
Female	50	1
TOTAL	150	6

TABLE NO: 3; AMYLASE LEVEL SAMPLE 1

AGE DISTRIBUTION	MEAN	S.D
12 -25	151.8	133.5
26-40	199.7	167.8
41-55	237.6	170.1
56-70	230.3	185.4

TABLE NO: 4; AMYLASE LEVEL SAMPLE 2

AGE DISTRIBUTION	MEAN	S.D
12-25	110.6	109.7
26-40	109.5	95.2
41-55	122	99
56-70	148.7	128.6

TABLE NO: 5; REASON FOR POISIONING

REASON	Number
Financial problem	20
Familial issue	130

TABLE NO: 6: CLINICAL FEATURES

A] SECRETIONS

SECRETIONS	CASES
+	6
++	120
+++	20
NIL	4
TOTAL	150

B] MENTAL STATUS

MENTAL STATUS	CASES
+	15
++	5
NIL	130
TOTAL	150

C] FASICULATIONS

FASICULATIONS	CASES
+	25
NIL	125
TOTAL	150

D] HEART RATE

HEART RATE	CASES
BRADYCARDIA	58
NORMAL	92
TOTAL	150

E] RESPIRATORY FAILURE

RESPIRATORY FAILURE	CASES
+	28
NIL	122
TOTAL	150

F] COMPARISION OF AMYLASE SAMPLE 1 AND 2 FOR PINPOINT PUPIL

PINPOINT PUPIL	AMYLASE SAMPLE 1		AMYLASE SAMPLE 2	
	MEAN	S.D	MEAN	S.D
PRESENT(40)	244.9	152.9	141.6	108
ABSENT(110)	141.5	133.1	63.2	26.2
	<0.001 Significant		< 0.001 Significant	

G] COMPARISION OF AMYLASE SAMPLE 1 AND 2 FOR MENTAL STATUS

MENTAL STATUS	AMYLASE SAMPLE 1		AMYLASE SAMPLE 2	
	MEAN	S.D	MEAN	S.D
PRESENT	342.2	78.8	264.2	74.3
ABSENT	163.5	138.7	74.5	51.9
	<0.001 Significant		<0.001 Significant	

H] COMPARISION OF AMYLASE SAMPLE 1 AND 2 FOR FASICULATIONS

FASICULATIONS	AMYLASE SAMPLE 1		AMYLASE SAMPLE 2	
	MEAN	S.D	MEAN	S.D
PRESENT	355.2	105.8	251.6	94.8
ABSENT	176.5	145.1	95.4	68.1
	<0.001 Significant		<0.001 Significant	

I] COMPARISION OF AMYLASE SAMPLE 1 AND 2 FOR RESPIRATORY FAILURE

RESPIRATORY FAILURE	AMYLASE SAMPLE 1		AMYLASE SAMPLE 2	
	MEAN	S.D	MEAN	S.D
PRESENT	330.7	137.7	226.3	122.5
ABSENT	156.8	126.3	59.6	36.7
	<0.001 Significant		<0.001 Significant	

J] PANCREATITIS AND ITS OUTCOME

PANCREATITIS	DEATHS
8	4
142	2

TABLE 7. RELATIONSHIP OF SERUM AMYLASE LEVELS WITH OUTCOME

OUTCOME	SERUM AMYLASE LEVELS {MEAN(SD)}	
	DAY 1	DAY 2
ALIVE	148.34(78.06)	67.70(36.98)
DIED	482.46(59.1)	166.96(11.32)
P VALUE	0.0001 Significant	0.0001 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 Poisonning – A Placebo – Controlled Trial using Pralidoxime. JAP1 May 2005; 53: 427430.
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/extoxnet/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-speakernotes.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. Gupta et al. Organophosphorus poisoning-facts and mights, Medicine Update, 1999; 1345-48. [15]. Kamath PG, Dalgi AJ, Patel BM. Diazinon poisoning, JAPI 1964; 14:477-81.
16. Vishwanathan M. and Shrinivasan K. Poisoning by bug poison - A preliminary study. Journal of Indian Medical Association 1962; 39:345-349.
17. Adhil SM and Sudharsan S. Estimation of serum amylase and lipase levels in correlation with clinical outcome of organophosphorus poisoning. Int. J. Modn. Res. Revs. 2015; 3(10):849-851.
18. Matsumiya N, Tanaka M, Iwai MN, Kondo T, Takahashi S, Sato S. Elevated amylase is related to the development of respiratory failure in organophosphate poisoning. Human Experimental Toxicology 1996; 15:250-253.
19. Tietz NW, Huang WY, Rauh DF, Shuey DF. Laboratory tests in differential diagnosis of hyperamylesemia. Clin Chem 1986; 32:301-7.
20. Ahmed A, Begum I, Aquil N, Atif S, Hussain T, Vohra E. Hyperamylasemia and acute pancreatitis following organophosphate poisoning. Pak J Med Sci 2009; 25:957-61.
21. Bhardwaj SSU, Verma SK, Bhalla A, Gill K. Hyperamylasemia and acute pancreatitis following anticholinesterase poisoning. Hum Exp Toxicol 2007; 26(6): 467-71.
22. Singh S, Bhardwaj U, Verma SK, Bhalla A, Gill K. Hyperamylasemia and acute pancreatitis following anticholinesterase poisoning. Hum Exp Toxicol 2007; 26:467-71.
23. Sumiya MN, Tanaka M, Iwai M, Konda T, Takahashi S, Sato S. Elevated serum amylase is related to development of respiratory failure in organophosphate poisoning. Hum Exp Toxicol 1996; 15(3): 250-53.

24. Lin CL, Yang CT, Pan KY, Huang CC. Most common intoxication in nephrology ward organophosphate poisoning. Ren Fail 2004; 26: 349-54.
25. Kavya S.T, Srinivas V, Chandana, Madhumati R. Clinical Profile of Patients with Organophosphorus poisoning in an intensive care unit in a tertiary hospital. International Journal of Clinical cases and investigations 2012; 4 (3): 24- 31.