



Study of Serum Creatine Phosphokinase in Patients of Organophosphorous Poisoning and Its Correlation with the Severity of Organophosphorous Poisoning

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ABSTRACT

OBJECTIVE-

To estimate the serum levels of Creatine phosphokinase in acute organophosphorus poisoning cases.

To assess serum creatine phosphokinase level correlation with severity of organophosphorus poisoning.

METHODS- The observational study was carried out on 50 patients above the age of 17 years presenting with organophosphorus poisoning admitted in Emergency Ward (EW) of medicine department at Tertiary Health Center, South Gujarat.

RESULT- The POP score, Serum cholinesterase levels and serum creatine phosphokinase levels showed a significant association in predicting the need for ventilatory support. Lower grade of poisoning had a better outcome whereas higher severity of poisoning had a poorer outcome.

CONCLUSION- The elevated creatine kinase is commonly seen in OP compound poisoning and associated with high morbidity and higher mortality. High Serum levels of creatine kinase at admission indirectly indicate the severity of poisoning and poor prognosis.

KEY WORDS: Organophosphorus poisoning, Serum creatine phosphokinase

among young economically active group with a common fatality ratio of 20%. Insecticide compounds has caused many numbers of suicidal deaths all over India^[1]. OP poisoning causes what is called the “suicide impulse” which leads to high level of suicides in some sectors of the agricultural industry. The incidence of international OP-related human exposures appears to be underestimated. According to the World Health Organization (WHO), 1 million serious unintentional poisonings occur every year and an additional 2 million people are hospitalized for suicide attempts with pesticides^[2]. The commonly encountered OP compounds comprising of insecticides (such as malathion, parathion, diazinon, fenthion, dichlorvos, chlorpyrifos, ethion), nerve gases (such as soman, sarin, tabun, VX), ophthalmic agents (echothiophate, isofluorophate), antihelmintics (such as trichlorfon), herbicides [including tribufos (DEF), merphos such as tricresyl phosphate containing industrial chemicals]. Massive OP intoxication from suicidal and accidental events, such as the Jamaican ginger palsy incident in 1930, has led to the discovery of the mechanism of action of OPs. They act by inhibiting the acetylcholinesterase enzyme (AChE) at muscarinic and nicotinic receptors, producing an array of symptoms like miosis, bradycardia, increased gastrointestinal motility, emesis, sweating, tachypnoea, salivation, lacrimation, altered sensorium, fasciculation, bronchospasm, blurred vision, photophobia, urination and defecation. Complications of op poisoning include acidosis, respiratory paralysis, acute renal failure, seizures, arrhythmias, aspiration, coma and even death. OP poisoning leads to three main syndromes: Acute cholinergic syndrome, intermediate syndrome (IMS), and OP induced delayed neuropathy (OPIDN).^[3] IMS occurs 48–96 h after ingestion of OP compound and following recovery from the acute cholinergic crisis, characterized by skeletal

I. INTRODUCTION

Acute poisoning by Organophosphorous insecticide (OP) has reached epidemic proportions in most parts of the world, particularly in developing countries like India, where agriculture is the backbone. The toxicity of Organophosphorous poisoning and paucity of appropriate medical facilities accounts for a high fatality rate. Their ease of access and socio-cultural factors play important role in choice of OP as a self-poison and the incidence is higher



muscle weakness. Respiratory paralysis in IMS if identified early can reduce the need for ventilator support and appropriate treatment can be initiated at the earliest.^[4] Hence, identifying the patients at high risk for IMS may lead to a decrease in morbidity and mortality. The causes of death in OP poisoning may be either one or a combination of the above. Early diagnosis is a key to cure. A delay of initiation of treatments limits not only outcome, but also the opportunity to use 2-PAM (cholinesterase re-activator) which prevents “aging” of the enzyme. Till now, investigations were comprised of serum erythrocyte cholinesterase (EchE) and plasma cholinesterase (PchE) estimation, the levels of which are reduced in OP poisoning. But these are costly and not regularly performed in most laboratories of our country. Besides, the kinetic study of inhibition of human AchEs by Demeton-S-methyl has shown that cholinesterase-based titration methods are not suitable for the estimation of Ops.^[5] There are emerging options for newer, cheaper and/or easily quantifiable biochemical markers to determine the severity in OP poisoning like creatine phosphokinase (CPK), lactate dehydrogenase (LDH), serum immunoglobulins (IgG, IgA), circulating complements (C3, C4), etc.^[6] But the immunoglobulin assays – IgG, IgA, are costly and difficult to perform in most laboratories, are often unreliable. Several animal model studies conducted on rat liver and fresh-water snails have indicated the association between OP poisoning and CPK levels.^[7] In a study, it was proposed that serum level of CPK is often found to be elevated in OP poisoning and may be used as a biomarker. There will be the elevation of serum CPK in OP

poisoning due to myonecrosis caused by persistent depolarization at the neuromuscular junction and oxidative cellular damage to muscle membrane.^[8] Serum CPK level has also been studied as a predictor for the onset of IMS. With this background in mind, we are undertaking a study to assess the role of CPK as an alternative prognostic marker and to establish a correlation between CPK levels and the severity of OP poisoning.

II. MATERIAL AND METHODS –

The study was conducted among indoor patients admitted to our tertiary care hospital.

(i) Study Design- observational, cross sectional analytical study

(ii) Inclusion Criteria -

Patients who meet all the following criteria were included in the study-

1. Patients > 18 years
2. Patients with history of exposure to OP poisoning within 24 hours

(iii) Exclusion Criteria (All / any of the following)-

1. Patients having age less than 18 years.
2. History of Mixed poisoning
3. History suggestive of Myopathy, Epilepsy, Psychiatric illness, autoimmune disease, Malignancy, Trauma, Sepsis, Renal disease, Myocardial infarction and Myocarditis, recent IM injection.
4. History of drug intake like Statins, Fibrates, Dexamethasone, Frusemide and Amphotericin B.

III. OBSERVATION-

TABLE 1: DISTRIBUTION OF PATIENTS ACCORDING TO TYPE OF ORGANOPHOSPHOROUS COMPOUND CONSUMED

TYPE OF O.P. CONSUMED	NO. OF PATIENTS	PERCENTAGE (%)
MALATHION	8	16
ENDOSULPHAN	8	16
METHYL PARATHION	8	16
CHLORPYRIPHOS	11	22
DIAZINON	7	14
DICHLORVOS	8	16

In this study most commonly used poison was chlorpyriphos upto 22 % of total patients followed by Dichlorvos, Endosulphan , malathion, diazinon and Methyl parathion each having value of 16% of total patients.



TABLE 2: DISTRIBUTION OF PATIENTS ACCORDING TO THEIR PRESENTING SYMPTOMS

SYMPTOMS	NO. OF PATIENTS	PERCENTAGE (%)
VOMITING	43	86
NAUSEA	36	72
SALIVATION	36	72
SWEATING	35	70
BRONCHORHEA	28	56
LACRIMATION	25	50
BREATHLESSNESS	25	50
DIARRHOEA	20	40
CONVULSION	11	22
DIPLOPIA	8	16

In this study, the most common symptom reported by patients was vomiting (86%) followed by other common symptoms like nausea (72%), Salivation in 72% and excessive sweating in 70% patients.

TABLE 3: DISTRIBUTION OF PATIENTS ACCORDING TO THEIR CLINICAL SIGNS

SIGNS	No. of patients	Percentage (%)
MIOSIS	47	94
BRADYCARDIA	34	68
RESP RATE	33	66
FASCICULATION	18	36
NECK MUSCLE WEAKNESS	17	34
ALTERED CONSCIOUSNESS	12	24
CONVULSION	10	20
CYANOSIS	8	16

In this study, the most commonly found clinical sign was miosis in 94% of patients followed by bradycardia which was seen in 68 % of patients

TABLE 4: ASSOCIATION BETWEEN QUANTITY OF POISON CONSUMED AND MORTALITY

QUANTITY	NO. OF PATIENTS	EXPIRED	SURVIVED	p value <0.002
<30	15	0	15	
30-50	17	0	17	
>50	18	6	12	



*chi square test applied p value <0.002

None of patients expired in this study who consumed up to 50 ml of poison. 6 patients who expired out of 18 consumed more than 50ml of poison. This result was statistically significant (p value <0.002).

TABLE 5: OUTCOME OF PATIENTS

OUTCOME	NO. OF PATIENTS	PERCENTAGE (%)
SURVIVED	44	88
EXPIRED	6	12
TOTAL	50	100

In our study mortality was 12% and survival rate was 88%.

TABLE 6: POP SCORE: SHOWING SEVERITY ACCORDING TO PERADENIYA OP POISONING SCORE^[9]

POP SCORE	SEVERITY	NO. OF PATIENTS	PERCENTAGE (%)
0-3	MILD	28	56
4-7	MODERATE	16	32
8-11	SEVERE	6	12

In our study, 56% of patients belonged to mild grade of poisoning with a POP score 0-3. 32 % patient had a score between 4 to 7 and 12% patients had severe poisoning having score between 8 to 11.

TABLE 7: COMPARISON OF SEVERITY ACCORDING TO POP SCORE V/S SERUM CHOLINESTERASE LEVELS (IU/L)

POP SCORE	SERUM CHOLINESTERASE LEVELS (IU/L)			
	>4200	1681-4200	841-1680	<840
0-3 (MILD)	7	12	8	1
4-7(MODERATE)	0	6	5	5
8-11 (SEVERE)	0	0	0	6
NO. OF PATIENTS	7	18	13	12

TABLE 8: ASSOCIATION BETWEEN SERUM CHOLINESTERASE AND POP SCORE

POP SCORE	MEAN OF SERUM CHOLINESTERASE(IU/L)	STD. DEV. IN S.
0-3	2930	1533.41
4-7	1404	716.05



8-11	373	157.76
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*Anova p- value <0.00006

With the rise in severity of O.P. poisoning according to POP score, there is a decrease in levels of serum cholinesterase levels and its activity. In our study, there was significant statistical association found (p- value <0.00006).

TABLE 9: COMPARISON BETWEEN POP SCORE AND MEAN SERUM CREATINE PHOSPHOKINASE LEVELS (IU/L) (n=50)

POP SCORE	MEAN OF SERUM CREATINE PHOSPHOKINASE (IU/L)	StdDev of SERUM CREATINE PHOSPHOKINASE	NO. OF PATIENTS	P-value <0.0001
0-3	77.14	23.63	28	
4-7	386.56	286.36	16	
8-11	788.66	216.25	6	

*ANOVA P-VALUE <0.0001

The mean serum CPK levels are not same among the different levels of POP score. The mean S.CPK levels were in lower range with mild POP score and were in higher range with increasing severity of POP score. This result was statistically significant (PVALUE <0.0001).

TABLE 10: COMPARISON OF PATIENTS ACCORDING TO SEVERITY OF SERUM CHOLINESTERASE WITH SERUM CREATINE PHOSPHOKINASE

GRADE	CHOLINESTERASE ACTIVITY	SERUM CHOLINESTERASE (IU/L)	SERUM CREATINE PHOSPHOKINASE(IU/L)	
			NORMAL	ABNORMAL
			29-195	>195
NORMAL	>50%	>4200	7	0
MILD	20-50%	1680-4200	15	3
MODERATE	10-20%	840-1680	9	4
SEVERE	<10%	<840	2	10

As the severity of O.P. poisoning increases, there is decrease in cholinesterase levels with subsequent decrease in its activity, there was a rise in S. CPK levels. This was a statistically significant.

TABLE 11: COMPARISON BETWEEN POP SCORE, SERUM CREATINE PHOSPHOKINASE AND TOTAL AMOUNT OF ATROPINE GIVEN

POP SCORE	NO. OF PATIENTS (n)	MEAN OF TOTAL ATROPINE DOSE (mg) (+SD)	MEAN OF SERUM CREATINE PHOSPHOKINASE(IU/L) (±SD)
0-3	28	22.32(± 7.32)	77.14(± 23.63)
4-7	16	67.56 (±15.59)	386.56 (± 286.36)
8-11	6	104 (± 15.23)	788.66 (± 216.25)

*ANOVA p-value <0.002



There was an increase in requirement of atropine with rise in serum creatine phosphokinase levels and severity of poisoning determined by POP score. In our study, this was found to be statistically significant. (p-value <0.002)

TABLE 12: SHOWING ASSOCIATION BETWEEN POP SCORE, SERUM CREATINE PHOSPHOKINASE LEVELS, SERUM CHOLINESTERASE LEVELS AND OUTCOME

POP SCORE	MEAN OF SERUM CREATINE PHOSPHOKINASE (IU/L)	MEAN OF SERUM CHOLINESTERASE(IU/L)	EXPIRED	SURVIVED
0-3	77.14 (± 23.63)	2930 (± 1533.4)	0	28
4-7	386.56 (± 286.36)	1404 (± 716.05)	2	14
8-11	788.66 (± 216.25)	373 (± 157.76)	4	2

chi square test p value <0.00003 With the increasing severity of O.P poisoning, there was subsequent rise in S.CPK Levels and decrease in serum cholinesterase levels which was associated with increasing mortality rates. In our study, the result was statistically significant (p value <0.00003).

TABLE 13: SHOWING ASSOCIATION BETWEEN SERUM CREATINE PHOSPHOKINASE AND AMOUNT OF OP CONSUMED

Count of QUANTITY				
S.CPK(IU/L)	<30 ml	30-50 ml	>50ml	Grand Total
29-195	4	23	6	33
>195	1	4	12	17
Grand Total	5	27	18	50

*chi square test applied p value <0.0012 There was a rise in S. CPK levels in the patients who consumed higher amount of poison. The result was statistically significant (p value <0.0012).

TABLE 14: VENTILATORY SUPPORT TABLE 33: SHOWING ASSOCIATION BETWEEN SERUM CREATINE PHOSPHOKINASE LEVELS, POP SCORE AND REQUIREMENT OF VENTILATORY SUPPORT

POP SCORE	MEAN OF SERUM CREATINE PHOSPHOKINASE (± SD)	VENTILATORY SUPPORT		PERCENTAGE %
		NO	YES	
0-3	77.14(± 23.63)	27	1	3.57
4-7	386.56 (± 286.36)	9	7	43.75
8-11	788.66 (± 216.25)	0	6	100

There was an increase in need for ventilatory support in our study with the rise in severity of POP score with subsequent rise in levels of S.CPK levels.

TABLE 15: SHOWING THE ASSOCIATION BETWEEN POP SCORE, MEAN SERUM CREATINE PHOSPHOKINASE LEVELS AND COMPLICATIONS OF OP POISONING

OF O.P. PO ISO	POP SCORE	POP SCORE AND SERUM CREATINE PHOSPHOKINASE LEVELS (MEAN ± SD)		
		MILD (0-3) (n=28)	MODERATE (4-7) (n=16)	SEVERE (8-11) (n=6)



S.CPK	77.14(± 23.63)	386.56 (± 286.36)	788.66 (± 216.25)
RESPIRATORY PARALYSIS	0	6	6
ARDS	0	6	4
PARAPLEGIA	0	0	0
RENAL FAILURE	0	1	2
HYPOTENSION	0	4	5
ARRHYTHMIAS	0	0	0
DEEP COMA	0	0	0
SEIZURES	1	4	5

The most common complication seen among the patients was respiratory muscle paralysis (24%), followed by ARDS (20%) and seizures (20%). The rate of complications increased with the severity of POP score and rising S.CPK levels.

IV. DISCUSSION-

A total 50 no. of cases were studied. The clinical and diagnostic findings of this study were compared with other studies in literature here.

Organophosphorus (op) compounds are most widely used pesticides in agriculture and due to their easy accessibility, OP toxicity is important health problem especially in developing countries like India.

In our study, majority of patients were in the age group of 18-30 years (44%). 90% of patients were within 40 years of age.

TABLE 16: COMPARING THE AGE GROUPS WITH MAXIMUM INCIDENCE IN VARYING STUDIES.

AUTHORS	AGE GROUPS (in years)	PERCENTAGE (%)
PRESENT STUDY	18-30	44
GOEL et al^[10]	12-30	86.4
REIHMAN et al^[11]	15-25	70
BASAVARAJ et al^[12]	21-30	46

TABLE 17: COMPARING MALE TO FEMALE RATIO IN VARYING STUDIES

SEX	PRESENT STUDY	SHANKAR P. S. et al^[13]	A. GOEL et al^[10]
FEMALE	52%	59.87%	60%
MALE	48%	40.20%	40%
M : F	0.92:1	1.48:1	2.5:1

In present study, out of 50 patients, 48% were males and 52% were females. The male to female ratio in this study is 0.92:1. In other similar study gender distribution reported by Shankar et al(1.48:1) and A Goel et al(2.5:1).

TABLE 18: COMPARING MARRIED AND UNMARRIED RATIO IN VARYING STUDIES

MARITAL STATUS	PRESENT STUDY	BASAVARAJ et al^[12]
MARRIED	70%	74%
UNMARRIED	30%	26%
RATIO	2.33:1	2.85:1



In our study, married patients were 35 (70%) and unmarried patients were 15 (30%).
The ratio of married to unmarried in this study is 2.33:1, comparison to 2.85:1 in Basavaraj et al^[12]

TABLE 19: COMPARING SOCIO ECONOMIC CLASS IN VARYING STUDIES.

SOCIOECONOMIC STATUS	PRESENT STUDY	A GOEL et al ^[10]	CHATTERJEE et al ^[14]
LOWER	52%	75.73%	88%
MIDDLE	44%	24.27%	11%
UPPER	4%	0%	1%

52% of patients in this study were from a lower socioeconomic group. In other studies done by A Goel et al^[10] and Chatterjee et al^[14], 75% of patients were from low socioeconomic group. The likely reason of rise in the incidences of OP

poisoning in middle socioeconomic class, in compare to low socioeconomic class as per previous study, can be given by the increase prevalence of psychiatric illness like depression in community.

TABLE 20: COMPARING OCCUPATION IN VARYING STUDIES.

OCCUPATION	PRESENT STUDY	REIHMAN et al ^[11]	HONNAKATTI V et al ^[15]
AGRICULTURAL LABOURERS	28%	14%	38%
HOUSE WIFE	32%	28%	25%

In our study, majority of patients who consumed O.P. poison had suicidal intention were house wives in their 30s and majority belonged to lower socioeconomic class.

TABLE 21: COMPARING INTENTION OF SUICIDE IN VARYING STUDIES.

STUDY	SUICIDAL INTENTION
PRESENT STUDY	92%
NOIURA et al ^[16]	90%
GOEL et al ^[10]	96.10%
GUPTA et al ^[17]	91%

Almost all cases in our study (92%) had consumed poison with a suicidal intent. This is in comparison to values reported by Noiura et al(90%), Goel et al(96.1%), and Gupta et al(91%).

The commonest route of exposure was oral route and the commonest manner of poisoning was suicidal and the commonest compound being

used Chlorpyrifos (22%). WeissmannBrenner et al^[18] reported that 66% of patients with OP poisonings were males and 34% were females, 39% were less than 10 years old, 64% of exposure was accidental, 36% was suicidal and the most common route of intoxication was oral (67%).

TABLE 22: OF SYMPTOMATOLOGY IN VARYING STUDIES.

SYMPTOMS	PRESENT STUDY	REIHMAN et al ⁵⁶	APN KUMAR et al ⁶⁴	GOEL et al ⁵⁵	KUMAR et al ⁶⁵
VOMITING	86%	80%	93%	97.08%	62.50%



SALIVATION	72%	32%	85%	28.15%	36.25%
SWEATING	70%	-	-	-	36.25%
NAUSEA	72%	-	-	-	77.50%
LACRIMATION	50%	-	80.60%	-	7.50%

In the present study, vomiting was the commonest symptom seen in 86%, followed by nausea (72%) and Salivation (72%).

TABLE 23: COMPARISON OF CLINICAL SIGNS IN VARYING STUDIES.

CLINICAL SIGNS	PRESENT STUDY	REIHMAN et al^[11]	A GOEL et al^[10]	APN KUMAR et Al^[19]	KUMAR et al^[20]
MIOSIS	94%	60%	95%	62%	93.75%
FASCICULATION	36%	8%	55%	38.60%	65%
TACHYPNOEA	66%	34%	42.50%	81.30%	73.50%
BRADYCARDIA	68%	52%	-	39%	27.50%
ALTERED SENSORIUM	24%	30%	75%	-	25%

The common clinical signs were miosis (94%), tachypnoea (66%), Fasciculations (36%). These results are comparable to the studies of Reihman et al^[11], A Goel et al^[10], APN Kumar et al^[19] and Kumar et al^[20].

TABLE 24: COMPARISON OF MORTALITY IN VARYING STUDIES.

STUDY	MORTALITY
PRESENT STUDY	12%
DAS B. WET et al^[21]	13.30%
ARUP KUMAR KUNDU et al^[22]	13.30%
NOIURA et al^[23]	10%
REIHMAN et al^[11]	14%

In present study, mortality of 12%. which is in comparison with Das. B.Wet al^[21](13.3%),Arup kumar kundu et al^[22](13.3%), Noiura et al^[23](10%), Reihman et al^[11](14%). 100% of patients with mild grade of poison according to POP scale survived. 2 out of 6 patients

who had expired had moderate grade and 4 patients expired out of 6 who had severe grade of poisoning according to POP scale. POP scale had a statistically significant correlation with mortality. (p value < 0. 00003).

TABLE 25: COMPARISON OF S.CPK IN VARYING STUDIES.

	S. CPK LEVELS (MEAN ± SD)	
	PRESENT STUDY	BHATTACHARYA et al^[24]



MILD (0-3)	77.14(± 23.63)	273.53 (± 108.71)
MODERATE (4-7)	386.56 (± 286.36)	456.06 (± 77.02)
SEVERE (8-11)	788.66 (± 216.25)	1032.57 (± 205.65)

In our study, there is positive correlation between CPK and POP score ($r=0.8393$). These results are in correlation with **Bhattacharyya et al**^[24] who confirmed the presence of a high degree of correlation between initial CPK value and POP scale. Muscle fiber necrosis and consequently increase in CPK levels occur in severely acute OP poisoned cases. So, cheaper, easily quantifiable and more available biochemical markers in relation to OP poisoning like serum CPK, serum amylase, serum lactate dehydrogenase etc. can be used in predicting and assessing the prognosis of patients with OP poisoning.

V. CONCLUSION

The elevated creatine kinase is commonly seen in OP compound poisoning and associated with high morbidity and higher mortality. We also concluded that higher the clinical grade of poisoning at initial presentation, more is the incidence of respiratory failure and need for mechanical ventilator support. High Serum levels of creatine kinase at admission indirectly indicate the severity of poisoning and poor prognosis. Early estimation of creatine kinase should be routinely considered as it is a good prognostic marker as well as marker in intermediate syndrome with cost benefits and easy availability.

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