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Biochemical Abnormalities in OPS Poisoning and its Prognostic Significance

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Abstract *— Background: Organophosphorus insecticides are arguably one of the commonest causes of morbidity and mortality due to poisoning worldwide, especially in developing countries like India due to its easy availability. Though Serum cholinesterase can be a useful tool in the diagnosis of OP poisoning, its role in prognostication is very minimal. Our study is conducted to other biochemical abnormalities to predict the severity and prognosis in OP poisoning patients. Aims and Objectives: (1)To measure serum electrolytes, liver enzymes, amylase, CPK, CPK-MB, and Troponin I in acute organophosphorus poisoning (2) To analyse the correlation between these biochemical parameters and serum acetylcholinesterase levels (3) To analyse the validity of these biochemical parameters in prediction of severity and prognosis in op poisoning. Materials and Methods: This study is conducted over a period of one and half year in J.L.N Government Hospital attached to J.L.N Govt. Medical College, AJMER wherein 40 OP poisoning patients are selected using inclusion and exclusion criteria and their blood samples are collected on admission and analysed for the above said biochemical parameters. Results: 74% of the patients were male, and 62% of the patients fall in the age group of 20 to 30 years. Statistically significant elevation of ALT, CPK, CPK-MB and Troponin I were noted in the study group. A significant fall in serum potassium level in also noted. Most of the patients in the study had a cholinesterase level of 20% to 50%. Conclusion: AST, ALT, ALP, and Amylase increase in acute OP poisoning. Rise in CPK, CPK-MB, Troponin I and ALT indicate the severity of OP poisoning and is also statistically significant to predict the prognosis of the patient. Hypokalaemia and associated low cholinesterase levels indicate the requirement of ventilator support and also the poor prognosis of the patient. These findings can assist health professionals to better evaluate patient's prognosis and improve their treatment plan.*

Keyword *— **OPC, Serum Amylase , AST, ALT, ALP***

I. INTRODUCTION

The modern world thrives well on revolution in the agricultural practices that has resulted in a massive thrust in agricultural productivity. One of the most important step in green revolution is pesticides. Pesticides are a class of toxic substances that are intentionally released into the environment for the greater good it does that exceeds their toxicological concerns. In the developing world, Poisoning is a common method of suicide.[1] Pesticide poisoning is a major health hazard in the developing world.[2] Millions of people are exposed to these dangerous chemicals because of the occupational hazards and also because of unsafe storage practices.[3] However it is the deliberate self-poisoning that causes majority of the deaths and a difficult health strategy to manage among health services, especially in Asia. According to World health organisation report, about three million cases of pesticide poisoning occur every year worldwide and most of them are in Asia, among

which 50% of them are organophosphate poisoning. The exact rate of OP poisoning in India is not clear because of under reporting and lack of data. India is an agricultural country and OP compounds are used greatly for the agriculture in India. Therefore the access to these harmful pesticide substances is so easy. In many reports from India, rate of suicidal poisoning with Opc ranges from 10 to 43%[4]Among these patients mortality rate is as high as 20 to 70%[5] In developed countries like United Kingdom, the death due to OP compounds relate to only 1%. This is because in developing countries like India the facilities for early diagnosis and treatment are very limited.

The morbidity and mortality in these patients depends on the time lag between the exposure and the onset of management. So it is very important to recognise the whole spectrum of symptoms in OP poisoning. Organophosphorus compounds inhibit acetyl cholinesterase and butyryl cholinesterase enzymes resulting in excess acetyl choline in the neuromuscular junction causing overstimulation at the cholinergic synapses. The symptoms are classified into muscarinic, nicotinic and central depending on the site of the compound over the respective receptors. Urination, lacrimation, emesis, miosis, excessive salivation, bradycardia, diarrhoea, and wheezing are the muscarinic features. Nicotinic features are paresis, fasciculation, tachycardia, and hypertension. Central features includes confusion, anxiety, seizures, ataxia and psychosis[7]. The need for newer biomarkers in relation to OP poisoning started a very long time ago. OP labelled albumin in plasma, blood beta-glucuronidase and paraxonase status were suggested by some scientists to be very reliable marker for both diagnosis of the poisoning and prognosis. But these assays are not available widely and are very costly. In a limited resourced country like India, we need cheap and easily measurable biomarkers. Many studies were conducted regarding this and were shown that Serum cholinesterase can be a useful tool in the diagnosis of OP poisoning. But its role in prognostication is very minimal. A number of recent studies were conducted using parameters like liver enzymes, serum amylase and serum CPK as newer markers and their correlation with severity and prognosis of OP poisoning[8-11]. Our study was conducted to assess parameters like CPK-MB, Serum potassium, Troponin I in correlation with Serum cholinesterase along with other liver enzymes, and serum amylase to predict the severity and prognosis in OP poisoning patients.

II. AIMS AND OBJECTIVES

AIM

To analyse the biochemical parameters (Serum acetyl Cholinesterase, Serum amylase, Serum lipase, CPK, CPK-MB, Liver enzymes, serum electrolytes) in cases of OPC poisoning.

OBJECTIVES

PRIMARY OBJECTIVES

1. To analyse biochemical alterations (Serum amylase Serum lipase, CPK, CPK-MB, Liver enzymes, serum electrolytes) in case of OPC poisoning.

2. To correlate the levels of these biochemical parameters to Plasma cholinesterase levels.

SECONDARY OBJECTIVES

1. To analyse the use of above biochemical parameters in predicting severity and prognosis of acute OPC poisoning.

III. MATERIALS AND METHODS

SOURCE OF DATA:

The present Study will done at J.L.N medical college and attached Hospital, Ajmer

STUDY DESIGN:

Cross-sectional observational Study

PERIOD OF STUDY

The study will conduct from April 2023 to March 2024.

ETHICS COMMITTEE APPROVAL

Approval willobtain from Institutional ethics committee.

INCLUSION CRITERIA

All the OP poisoning cases confirmed by history, circumstantial evidence of ingestion, admitted in our hospital within 12 hours of ingestion with characteristic clinical findings and basic laboratory investigations were included in the study

EXCLUSION CRITERIA

1. Patients with feature of exposure to another compound not relating to OP Poison.

2. Patients with mixed poisoning; OP poisoning and any other poison

3. Patients who has chronic alcoholism

4. Patients with history suggestive of liver disease

5. Patients with history of malignancy and autoimmune diseases

6. Patients with history of renal disease

7. Patients with history of cardiac disease

CONSENT

An informed consent was obtained from all the participants and their relatives wherever necessary.

SAMPLE COLLECTION

When the patient was admitted in our hospital, after obtaining informed consent, about 5ml of blood was collected in plain tube under aseptic precautions. The blood was allowed to clot and serum was separated by centrifugation and used for the analysis of following parameters.

SAMPLE SIZE:

Sample size was calculated at **95 % confidence level** expecting mean serum cholinesterase level of 2357 IU/L as one biochemical parameter among surviving patients of OPC poisoning as per the reference article (**Senthilnathan NK and Binny LA)** .biochemical abnormalities in OPC poisoning and its prognostic significance. Journal of Dental and Medical sciences.2017;16(7):116-9].

To detect mean difference of at least 500 IU/L in serum amylase among survivor and patients who died due to OPC poisoning as SD 2 to 4 at study power of 80%, the required sample size was **80 cases of OPC poisoning**. This sample size was enough to analyze all biochemical abnormalities in patients admitted with OPC poisoning.

Statistical Analysis

All the parameters are tabulated. Mean, Standard deviation will analyse using SPSS 20 software. All the biochemical parameters were correlated with serum cholinesterase using inter-correlations. Chi-square test was the test of significance used for qualitative variables to find the association between them. T test was the test of significance used for comparing quantitative variables with qualitative variable. One-way Anova is used as test of significance to assess various parameters with the compound used for poisoning.

IV. RESULTS

Table 1: Descriptive Statistics Liver Enzymes Amylase

	N	Min.	Max	Mean	S.D
Ast	50	20	220	113.48	50.287
Alt	50	18	220	109.62	50.004
Alp	50	48	362	165.96	82.646
Amylase	50	34	360	160.96	93.453

Table 2: Descriptive Statistics Serum Electrolytes

	Min.	Max	Mean	S.D
Sodium (InMeq/L)	126	144	134.90	3.370
Potassium (InMeq/L)	2.60	3.80	3.2140	.22769

Table 3: Descriptive Statistics Cpk, CpkMb

	Min.	Max	Mean	S.D
Cpk (Iu/L)	30	3738	802.38	955.396
Cpk-Mb (Iu/L)	15	758	191.76	207.497

Table 4: Compare The Outcome With TroponinI

TropI	Death		Live		Total		Statistical inference
	(N=21)	(100%)	(N=29)	(100%)	(N=50)	(100%)	
Negative	0	.0%	27	93.1%	27	54.0%	X^2=42.504Df=1 .000<0.05 **Significant**
Positive	21	100.0%	2	6.9%	23	46.0%	

Table 5: Enzymes With Outcome

Ast	**Mean**	**S.D**	**Statistical Inference**
Live(N=29)	102.24	50.026	T=-1.906Df=48 .063>0.05 Not Significant
Death(N=21)	129.00	47.494	
Alt	**Mean**	**S.D**	**Statistical Inference**
Live(N=29)	93.31	50.985	T=-2.909Df=48 .005<0.05 **Significant**
Death (N=21)	132.14	39.603	
Alp	**Mean**	**S.D**	**Statistical Inference**
Live(N=29)	157.90	76.705	T=-.808Df=48 .423>0.05 Not Significant
Death (N=21)	177.10	90.954	
Amylase	**Mean**	**S.D**	**StatisticalInference**
Live(N=29)	140.59	84.798	T=-1.856Df=48 .070>0.05 Not Significant
Death (N=21)	189.10	99.484	

Table 6: Electrolytes With Outcome

Sodium	**Mean**	**S.D**	**Statistical Inference**
Live(N=29)	135.41	3.123	T=1.275Df=48 .208>0.05 Not Significant
Death (N=21)	134.19	3.642	
Potassium	**Mean**	**S.D**	**Statistical Inference**
Live(N=29)	3.2690	.23468	T=2.072Df=48 .044<0.05 **Significant**
Death (N=21)	3.1381	.19869	

Table 7: S. AchE with outcome

S.Ache	**Mean**	**S.D**	**Statistical Inference**
Live(N=29)	2357.34	2242.477	T=3.096Df=48 .003<0.05 **Significant**
Death(N=21)	821.00	399.836	

Table 8: CPK & CPKMB with outcome

CPK	**Mean**	**S.D**	**Statistical Inference**
Live(n=29)	444.76	621.248	T=-3.436Df=48 .001<0.05 **Significant**
Death (n=21)	1296.24	1120.022	
CPK-MB	**Mean**	**S.D**	**Statistical Inference**
Live(n=29)	81.72	93.538	T=-5.613Df=48 .000<0.05 **Significant**
Death (n=21)	343.71	226.792	

V. DISCUSSION

Organophosphate ingestion is one of the supreme cause of suicidal deaths in India. Inhibition of acetylcholinesterase is the main mechanism by which organophosphates act leading to excessive cholinergic stimulation. The clinical features of cholinergic storm develops fast, which helps in diagnosing clinically that is established by detailed history and biochemical demonstration of cholinesterase inhibition. This study is undertaken to analyse the biochemical abnormalities in organophosphate poisoning and to assess their prognostic significance. In this study the total number of patients were 50. Among them 37 of them were male (74%) and 13 of them were female (26%), showing that the incidence of poisoning is more in males. The mean values of AST-113.48 IU/L, ALT-109.62 IU/L, ALP- 165.96 IU/L, and Amylase- 160.96 IU/L is noted among the poisoning patients in this study. According to Lohitnavy & Vijayaraghavan[1,2], there is elevation of Serum AST and ALT because of degeneration of hepatocytes and further necrosis, causing damage to cell organelles like mitochondria and pouring these enzymes into blood stream[3,4]. The present study also demonstrates elevation in liver enzymes following OP poisoning (Table 1). Among the liver enzymes ALT elevation shows a statistical significance when compared with the outcome and thus can be used as a prognostic indicator (Table 5).Singhetal[5] found elevationofamylase and acute pancreatitis inOP poisoning. Matsumiya N et al[6] and Li T Nagayama N et al[7] stated that elevated serum amylase in the absence of clinical pancreatitis could be attributed to hypoxemia. Dressel et al[8] showed that

OP intoxication causes increase in intraductal pressure and increase in exocrine pancreas flow rate resulting in extravasation of fluid. The mean serum potassium in our study is3.2meq/l(Table2).According to Devanur RMM and Prasadet al[9], poor out come in OP poisoning was noted in patients having respiratory distress with hypokalemia and very low serum cholinesterase. They also found that there was a remarkable fall in serum potassium relating to OP toxicity induced weakness of muscle and paralysis finally leading to death. The fall in serum potassium was proportional to the onset of detrimental signs and symptoms. In our study the mean potassium in patients with successful outcome is 3.2 and in patients who had a negative endpoint is 3.13. The p value for statistical significance is <0.05(Table 6). The mean CPK in our study is 802.38 IU/L and CPK MB is 191.76 IU/L (Table 8). In a study conducted in Egypt by Nermeen AM et al [10] they have demonstrated increases in serum CPK and a proportionate fall in serum cholinesterase. The excess of acetylcholine in OP poisoning causes reversible muscle injury and rise invarious muscle enzymes including CPK[11]. Dayanand Raddi et al conducted a study regarding CPK and OP poisoning and concluded escalation of CPK is evident of respiratory-failure and timely estimation of CPK has to be customarily taken as a prognostic indicator in OP poisoning[12]. The mechanism behind cardio toxicity of Organophosphates is not known. It is postulated that parasympathetic and sympathetic over activity causes myocardial damage. Parasympathetic over activity has significant role in coronary artery spasm. In our study also there is a significant correlation between CPK, CPK MB (Table 8), and Troponin I (Table 4) and the outcome of patient. Hence these three parameters can be used as a prognostic indicator in OP poisoning.

In our study serum cholinesterase mean value in patients who had successful outcome is 2357 IU/L and it is 821 IU/L in those who had a negative outcome. In our study the serum cholinesterase measured on the day of admission predicts the outcome with high statistical significance (Table 7).

VI. CONCLUSION

1. Incidence is high in male patients
2. Measurement of Serum potassium and serum cholinesterase is helpful in predicting the outcome and prognosis in OP poisoning. Hypokalemia associated with reduced cholinesterase level is related with a negative outcome.
3. Increase in serum creatine kinase is commonly seen in organophosphate poisoning.
4. Increase in CPK MB and TroponinI is commonly seen in organophosphate poisoning because of the cardio toxicity involved in OP poisoning
5. CPK, CPKMB and Troponin-I have high statistical significance
6. Prognosis of the patient.
7. The significant increase of liver enzyme activity and serum amylase appears to correlate with clinical severity of the patient in OP poisoning.
8. Among the liver enzymes and serum amylase, ALT elevation has a statistical significance to predict the outcome of the patient.

VII. LIMITATIONS

1. The period of study is minimal, huge data would have been collected if done for long time.
2. Study group is just adequate.
3. Serial estimation of the biochemical parameters during the course of hospital stay is not done.
4. TroponinI is done in qualitative method and not quantitative method.
5. Previously undiagnosed cardiac disease is not ruled out in the study patients.

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