ORIGINAL RESEARCH

A Study on Serum Pseudocholinesterase, Creatine Phosphokinase, and Amylase as Prognostic Markers in Organophosphorus Poisoning

¹Dr. K.Sunny Sanjay, ²Dr. Suresh M, ³Galeti Kalyan Kumar

¹Postgraduate, ^{2,3}Assistant Professor, Department of General Medicine, PES Medical College (PESIMSR), India

Corresponding Author

Dr. K.Sunny Sanjay Postgraduate, Department of General Medicine, PES Medical College (PESIMSR), India Email: sunnysanjaykalagadda@gmail.com

Received Date: 25 July, 2024

Acceptance Date: 28 August, 2024

ABSTRACT

Background: Organophosphate (OP) pesticide poisoning remains a critical public health issue in developing countries, including India, Pakistan, and Sri Lanka. These compounds, used extensively as insecticides, petroleum additives, and nerve agents, continue to pose significant health risks due to their widespread availability and limited public awareness. This study aims to assess the utility of biochemical biomarkers in evaluating the severity of OP poisoning. **Materials and methods:** An observational study at PES Institute of Medical Sciences and Research (PESIMSR) from January 2021 to June 2022 included 152 patients with confirmed OP poisoning. Exclusion criteria included co-existing conditions and recent interventions. Biochemical markers—serum amylase, plasma cholinesterase (CHE), and serum creatine kinase (CPK)—were analyzed at admission, and on Days 1, 3, and 5. Clinical outcomes were recorded, including ventilator support and mortality. Data were analyzed using IBM SPSS Statistics. **Results:** Patients were predominantly aged 31-40 years, with a male predominance (1.86:1). Of the 152 patients, 127 survived, and 25 died. Elevated serum amylase, CPK, and decreased CHE levels were associated with worse outcomes. Significant correlations were observed between high amylase and CPK levels with poor prognosis and increased need for ventilator support. The mean APACHE II score was higher in patients needing ventilation. **Conclusion:** Biochemical biomarkers, particularly serum amylase, CPK, and plasma CHE, effectively assess OP poisoning severity. Elevated levels are linked to higher mortality and ventilator dependency, underscoring their utility in managing OP poisoning.

Key words: Organophosphorus Compounds, Poisoning, Pesticide, Suicide, Plasma, cholinesterase, Serum amylase. This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution- Non Commercial-Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

INTRODUCTION

Organophosphate (OP) pesticide poisoning is a major public health challenge in developing nations like India, Pakistan, and Sri Lanka, as well as other parts of Asia. These compounds serve as insecticides, petroleum additives, and nerve agents in chemical warfare. They have been in use as pesticides for over five decades. Exposure to OP pesticides can occur accidentally, occupationally, or intentionally.^[1]

In 2012, the WHO reported 193,460 deaths from accidental poisoning and 370,000 from pesticiderelated suicides globally, with rural Asia being particularly affected. A study in Nepal by Shrestha R et al. found OPC poisoning to be the leading method of suicide and self-harm, accounting for 56.4% of cases.^[2] Laboratory analysis is crucial for confirming poisoning, detecting initial organ damage, and assessing severity. The most reliable lab test for OP poisoning is plasma cholinesterase measurement. Hyperamylasemia is a well-known metabolic disorder associated with OP exposure.^[3]Elevated serum amylase is also linked to the development of respiratory failure in OP poisoning.^[4]

Making prompt judgments on the transfer of patients for intensive care management and forecasting the clinical outcome may be aided by correlation. This study was conducted to determine the effectiveness of modern biochemical biomarkers for determining the severity of OP poisonings, such as lipase, amylase, and CPK.

Online ISSN: 2250-3137 Print ISSN: 2977-0122

DOI: 10.69605/ijlbpr_13.9.2024.35

MATERIALS AND METHODS

The study was an observational design conducted at PES Institute of Medical Sciences and Research (PESIMSR), Kuppam, over 18 months, from January 2021 to June 2022. The study population comprised PES patients admitted to Hospital with organophosphate poisoning, selected using purposive sampling. The sample size, calculated as 152, was based on previous study by Sumathi et al.^[5] Inclusion criteria included all confirmed organophosphate verified through poisoning cases history, circumstantial evidence, clinical examination, and basic laboratory investigations. Exclusion criteria involved patients with co-existing illnesses (e.g., myopathy, chronic renal disease, epilepsy), those with recent trauma or medical interventions, and those on specific medications or with alcohol use history.

The tools used were APACHE II, and qSOFA scoring. Blood samples were collected from patients admitted to the intensive care unit for biochemical analysis, including serum amylase (normal value: 45-130 U/L), plasma cholinesterase (normal value: 4659-14443 U/L), and serum creatine kinase (normal value: 20-200 IU/L). All analyses were performed using the DrychemistryVitros 250 Johnson and Johnson analyzer, with regular internal quality checks using Biorad controls.

Patients were followed for clinical outcomes, including recovery, acute respiratory distress syndrome, circulatory failure, CNS complications, renal failure, and death due to the aforementioned complications. Blood samples were collected on Day 1, Day 3, and Day 5, with serum amylase, plasma cholinesterase, and creatine kinase levels evaluated. The study adhered to ethical guidelines, ensuring informed consent and confidentiality throughout the process. The collected data were analysed with IBM SPSS Statistics for Windows, Version 26.0. (Armonk, NY: IBM Corp). To describe the descriptive data statistics frequency analysis, percentage analysis was used for categorical variables, and the mean & S.D were used for continuous variables. To find the significant relationship between the variables, the Chisquare test, t-test and Pearson coefficient are used. In all the above statistical tools, the probability value <0.05 is considered a significant level.

RESULTS

Among 152 patients with organophosphate (OP) poisoning, the majority were aged 31-40 years, and males predominated with a male-to-female ratio of 1.86:1. (Table 1) Of the total patients, 127 (83.6%) survived, while 25 (16.4%) died. The qSOFA scores among the patients showed that 100 (65.8%) had a score of two, 32 (21.1%) had a score of three, and 20 (13.2%) had a score of one.

There was a significant increase in mean serum Amylase levels from baseline in the study group, followed by a decreasing trend toward discharge (p < 0.05). The mean serum Amylase levels on Day 1, Day 3, Day 5, and at discharge are detailed in the table 2. Similarly, there was a significant increase in mean serum CPK levels from baseline, with levels also showing a decreasing trend towards discharge (p < 0.05). Additionally, there was a significant increase in mean plasma Cholinesterase (CHE) levels from baseline, followed by a decreasing trend toward discharge (p < 0.05). The mean plasma CHE levels at admission, Day 1, Day 3, Day 5, and at discharge are also presented in the table 2.

The mean serum Amylase levels in the survival group were 538 on Day 1, 357 on Day 3, 338 on Day 5, and 120 at discharge. The death group had significantly higher mean serum Amylase levels of 1299, 1095, 1104, 824, and 909 on the same days, with the differences being statistically significant (p < 0.05). Similarly, the mean serum CPK levels in the survival group were 318 on Day 1, 319 on Day 3, 316 on Day 5, and 255 at discharge, whereas the death group had higher mean levels of 422, 499, 636, 864, and 864, all statistically significant (p < 0.05). For plasma Cholinesterase (CHE), the survival group had mean levels of 956 on Day 1, 932 on Day 3, 970 on Day 5, and 962 at discharge, while the death group had significantly lower levels of 395, 326, 304, 435, and 729, respectively (p < 0.05). Additionally, the mean APACHE II score was significantly different between the groups, with 13.3 in the survival group and 24.2 in the death group (p < 0.05). Table 3 also highlights that the mean atropine doses required on Days 1, 2, and 3 differed significantly between the survival and death groups (p < 0.05). (Table 3)

At admission, both serum Amylase and Serum CPK showed a statistically significant negative correlation Plasma Cholinesterase, with correlation with coefficients of -0.744 and -0.237, respectively. On Day 1, this negative correlation persisted, with coefficients of -0.689 for serum Amylase and -0.265 for Serum CPK, both statistically significant. By Day 3, the negative correlation continued, with serum Amylase at -0.678 and Serum CPK at -0.308, again statistically significant. On Day 5, the correlation coefficients remained negative and statistically significant, with serum Amylase at -0.678 and Serum CPK at -0.360. Finally, at discharge, the negative correlation between serum Amylase and Serum CPK with Plasma Cholinesterase was still observed, with coefficients of -0.672 and -0.409, both statistically significant. (Table 4& Figure 1)

In our study, 36.8% of patients required ventilator support. Among these patients, the mean duration of ventilator use was 5.48 days with a standard deviation of 4.85. Out of the 152 patients studied, 56 required mechanical ventilation. Of these, 25 patients succumbed to their condition while 31 survived. The comparison between the mortality and survival groups yielded a chi-square statistic of 51.294 with a p-value <0.0001, indicating a statistically significant difference. Additionally, the mean APACHE II score for predicting the need for ventilator support was

22.29 in the ventilator group, which was statistically significant (p < 0.05).

Taplic characteristics of study participants								
Variables		Frequency (N)	Percentage (%)					
Age	< 20 years	13	8.6					
	21-30 years	46	30.3					
	31-40 years	66	43.4					
	41-50 years	14	9.2					
	> 50 years	13	8.6					
Gender	Male	99	65.1					
	Female	53	34.9					

 Table 1: Sociodemographic characteristics of study participants

Table 2: Comparison of Serum A	mylase, Serun	n CPK, Plasma	a CHE on	admission	n, D1, D3	and D5 by t
test.						

Parameter	Mean	SD	SE	T Score	P Value
Serum Amylase (SA)					
SA at admission	663.34	530.334	43.016	12.399	< 0.0001
SA 24hrs	478.91	457.057	37.072	9.412	< 0.0001
SA 72hrs	464.30	461.012	37.393	8.940	< 0.0001
SA 120hrs	314.43	322.037	26.836	6.872	< 0.0001
SA discharge	249.99	428.871	34.786	3.449	0.001
Serum CPK					
CPK at admission	335.80	194.447	15.772	8.610	< 0.0001
CPK 24hrs	348.90	244.103	19.799	7.521	< 0.0001
CPK 72hrs	368.80	300.939	24.409	6.915	< 0.0001
CPK 120hrs	403.42	444.581	37.048	5.491	< 0.0001
CPK discharge	345.19	413.174	33.849	4.289	0.000032
Plasma CHE					
PC at admission	1769.08	994.382	80.655	-38.199	< 0.00001
PC 24hrs	2106.18	1014.375	82.277	-33.349	< 0.00001
PC 72hrs	2320.59	1070.864	86.859	-29.121	< 0.00001
PC 120hrs	3008.05	1209.904	100.825	-18.269	< 0.00001
PC discharge	4356.18	1538.347	124.776	-3.958	0.00016

 Table 3: Comparison of Serum Amylase, Serum CPK, Plasma CHE and APACHE 2 with Outcome among patients

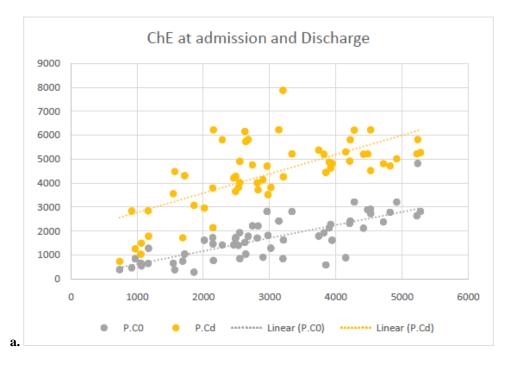
Parameter	Outcome	Mean	SD	SE	T Score	P Value		
Serum Amylase (SA)								
SA at admission	Survival	538.06	383.048	33.990	-7.739	< 0.00001		
	Death	1299.7	703.096	140.619				
SA 24hrs	Survival	357.48	239.045	21.212	-9.205	< 0.00001		
	Death	1095.8	734.781	146.956				
SA 72hrs	Survival	338.32	249.897	22.175	-9.625	< 0.00001		
	Death	1104.2	706.230	141.246				
SA 120hrs	Survival	222.39	148.145	13.412	-10.914	< 0.00001		
	Death	824.86	507.519	108.203				
SA discharge	Survival	120.13	51.316	4.554	-11.507	< 0.00001		
	Death	909.68	775.137	155.027				
		S	erum CPK					
CPK at admission	Survival	318.81	208.535	18.505	-2.468	0.015		
	Death	422.08	17.224	3.445				
CPK 24hrs	Survival	319.17	255.766	22.696	-3.510	0.001		
	Death	499.96	54.884	10.977				
CPK 72hrs	Survival	316.08	302.098	26.807	-5.285	< 0.0001		
	Death	636.64	35.106	7.021				
CPK 120hrs	Survival	320.20	432.005	39.112	-5.877	< 0.0001		
	Death	864.91	85.064	18.136				
CPK discharge	Survival	255.16	379.450	33.671	-7.485	< 0.0001		

	Death	864.91	85.064	18.136			
Mean Plasma CHE							
PC at admission	Survival	1967.4	956.103	84.840	-7.739	< 0.00001	
	Death	761.56	395.521	79.104			
PC 24hrs	Survival	2342.8	932.489	82.745	-9.205	< 0.00001	
	Death	903.88	326.509	65.302			
PC 72hrs	Survival	2580.4	970.944	86.157	-9.625	< 0.00001	
	Death	1000.4	304.524	60.905			
PC 120hrs	Survival	3327.0	1010.712	91.506	-10.924	< 0.00001	
	Death	1233.8	435.851	92.924			
PC discharge	Survival	4899.5	962.019	85.365	-11.507	< 0.00001	
	Death	1596.0	729.590	145.918			
		APA	CHE 2 scol	re			
Survival 13.30 5.673 0.503 -8.794						< 0.0001	
	Death	24.20	5.627	1.125			
Atropine							
Day 1	Survival	16.75	9.533	0.846	-11.330	< 0.0001	
	Death	39.60	7.348	1.470			
Day 2	Survival	10.43	6.156	0.546	-11.753	< 0.0001	
	Death	26.20	6.000	1.200			
Day 3	Survival	5.98	3.399	0.302	-13.308	< 0.0001	
	Death	19.20	8.251	1.650			

 Table 4: Correlation of Serum amylase and Creatinine Kinase with plasma Cholinesterase at admission, at 24 hours, at 72 hours, at 120 hours and discharge

Time Point	Serum Amyla	se	Serum Creatinine Kinase		
	Pearson Correlation	p-value	Pearson Correlation	p-value	
At Admission	-0.744**	< 0.0001	-0.237**	0.003	
24 Hours After Admission	-0.689**	< 0.0001	-0.265**	0.001	
72 Hours After Admission	-0.678**	< 0.0001	-0.308**	< 0.0001	
120 Hours After Admission	-0.678**	< 0.0001	-0.360**	< 0.0001	
At Discharge	-0.672**	< 0.0001	-0.409**	< 0.0001	

**Correlation is significant at the 0.01 level (2-tailed).



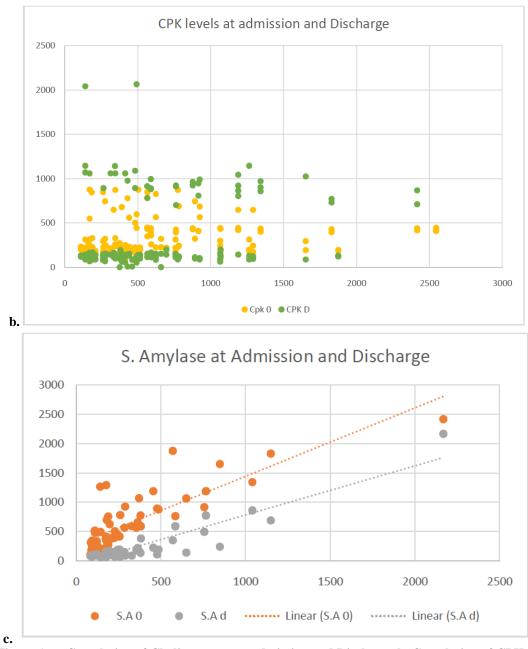


Figure 1: a. Correlation of Cholinesterase at admission and Discharge b. Correlation of CPK at admission and Discharge c. Correlation of S. Amylase at admission and discharge

DISCUSSION

A total of 152 OP compound poisoning patients satisfying the inclusion and exclusion criteria were taken for study. Our study observed that 56.1% of patients were in the 18-30 age group, aligning with findings from Dilip M Rampure et al.^[6], Goel et al.^[7], Reihman et al.^[8], who reported similar age distribution among patients. Murat Sungur et al.^[9]reported a mean age of around 30 years in Turkey. Studies by Karalliedde et al.^[10] and Malik et al.^[11] indicated a majority under 30 years of age, with a significant proportion under 25. Bhattacharyya et al.^[12] reported a mean age of 25.5 years in Kolkata, suggesting a younger demographic affected by organophosphate exposure. Our findings are

potentially because this age group was vulnerable to various emotional conflicts that occur during this phase of life, are described to be most stressful, emotionally weak and vulnerable to minor conflicts, failures or disappointments during this phase of life. We found male predominance with a ratio of 1.86:1, consistent with Raghu G et al.^[13],Biwas S et al.^[14], Subhash L.Patil et al.^[15], and Edwin J George et al.^[16]. Conversely, Sen R et al.^[17], and Sungur et al.^[9] reported a female predominance. The reason behind males commonly affected was due to as they are main working group in outdoor field, i.e. they are more involved in spraying crops in the farm and have the whole responsibility of their family.

According to WHO estimates, there are approximately 1 million accidental poisonings and 2 million suicide attempts involving pesticides annually. Studies by Murat Sunguret al.^[9] and Karalliedde L et al.^[10], observed high rates of suicidal exposure to organophosphates. Malik et al.^[11] reported 74.4% of cases were suicidal, while Palimar et al.^[18]found 98.7% were suicidal, often linked to crop failure and debt. In the UK, 2700 people seek hospital treatment for self-poisoning each week, highlighting the global scale of the issue.^[19] A study from West Bengal observed that 8 patients had a12% death rate associated with the ingestion route. Malik et al.^[11] 58 reported 140 cases of ingestion, 7 of inhalation, and 17 of topical exposure in Kashmir.

Nausea and vomiting were the most common symptoms in our study, similar to findings by Selvaraj et al.^[20], Edwin et al.^[16], and Dayanand et al.^[21]. Goswamy R et al.^[22] highlighted breathlessness as a common feature, potentially due to muscarinic receptor stimulation. Joshi et al. noted symptom onset within 30 minutes to 1 hour, with delayed treatment correlating with increased severity.^[5] Study from Turkey reported gastrointestinal and CNS symptoms respectively.^[9] Sen R et al. observed salivation and vomiting as the most common symptoms.^[17]

Our study found a significant decrease in plasma cholinesterase levels (P<0.001), consistent with findings by Bobba et al.^[23]. Agarwal et al. found no relationship between cholinesterase levels and severity.^[24] Sequential estimations of cholinesterase did not show significant rises despite clinical improvement in this study. Sen R et al. found a negative correlation between severity and cholinesterase levels, suggesting it is a useful diagnostic tool but less reliable for prognosis.^[17]

A study by Rabha et al.^[25] reported elevated creatine phosphokinase (CPK) levels among 70.97% of patients, with higher mortality rates associated with levels above 1000 U/L. Sen R et al.^[17] and Agarwal et al.^[24] also found significant CPK elevation in severe cases, indicating cardiac and muscle impairment. Bhattacharya et al, 2011 in a study conducted in Kolkata had also commented the same.^[12]Vanneste et al.^[26]and John et al.^[27] linked elevated CPK levels to muscle injury and severity. Our study also found elevated CPK levels correlated with increased severity and mortality, suggesting CPK levels can be an alternative to cholinesterase for monitoring.Matsumiya et al.[28] found high serum amylase levels associated with acute pancreatitis and clinical severity. Elevated amylase levels were correlated with mortality in our study, aligning with findings from Lee et al.^[29] and Sahin et al.^[30], although no cases of pancreatitis due to OP poisoning were observed.

Among 152 patients, 36.8% required ventilator support, with a mortality rate of 44.6%. Murat Sungur et al.^[9] and S. Shivakumar reported similar findings, with high mortality among ventilated patients.

Dayanand et al.^[21] found elevated CPK levels associated with the need for ventilation and increased mortality. Goel A et al.^[7] and Soni P et al.^[31] highlighted the impact of timely therapy on ventilatory support requirements. Our study found that a mean therapy delay of 9.64 hours was significantly associated with the need for mechanical ventilation. Karalliedde et al. reported an 18% mortality rate in 92 organophosphate poisoning (OPC) cases in Sri Lanka.^[10] In Turkey, Sungur et al. reported a 32% mortality rate.^[9]Out of 93 cases studied by Rabha et al.^[25]. 86 (92.47%) survived, while 7 (7.53%) died in our study. A study by Yuri Gagarin et al. noted that most deaths occurred in patients aged 41-60, with no significant gender disparity.^[32] Our study found a 22% mortality rate, with 48.9% of ventilated patients dying, especially those ventilated for over seven days.Our overall mortality rate was 16.4%, which is relatively low. Significant associations were found between serum CPK, cholinesterase, amylase levels, and outcomes.

CONCLUSION

Our study emphasizes that acute organophosphorus poisoning predominantly affects males in the 31-40 age group, with qSOFA and APACHE II scores serving as reliable indicators of illness severity and patient outcomes. The marked elevation of serum amylase and creatine kinase (CPK) levels at baseline, followed by their decline during hospitalization, coupled with the increase in plasma cholinesterase levels, suggests that these biomarkers are valuable in assessing poisoning severity. Additionally, the correlation between mechanical ventilation duration and higher APACHE II scores further supports the role of these metrics in predicting the need for intensive interventions. While these findings offer important insights into the management of organophosphorus poisoning, further randomized controlled trials are warranted to validate these predictive tools and enhance clinical decision-making.

REFERENCES

- Sundaray K, Ratheesh KJ. Organophosphorous poisoning: Current Management guidelines. 2010;420– 6.
- Amir A, Raza A, Qureshi T, Mahesar GB, Jafferi S, Haleem F, et al. Organophosphate Poisoning: Demographics, Severity Scores and Outcomes From National Poisoning Control Centre, Karachi. Cureus 12(5):e8371.
- Amanvermez R, Baydin A, Yardan T, Başol N, Günay M. Emergency Laboratory Abnormalities in Suicidal Patients with Acute Organophosphate Poisoning. Turkish Journal of Biochemistry 2010;35:29–34.
- 4. Thundiyil JG, Stober J, Besbelli N, Pronczuk J. Acute pesticide poisoning: a proposed classification tool. Bull World Health Organ 2008;86(3):205–9.
- Sumathi ME, Kumar SH, Shashidhar KN, Takkalaki N. Prognostic significance of various biochemical parameters in acute organophosphorus poisoning. Toxicol Int 2014;21(2):167–71.

- Rampure DM, Ganiger IB, Ellareddy. A Study on Serum Amylase Levels in Acute Organophosphorus Poisoning And Its Relationship with Clinical Severity and Outcome.
- Goel A, Joseph S, Dutta T. Organophosphate poisoning: predicting the need for ventilatory support. The Journal of the Association of Physicians of India 1998;46(9).
- Rehiman S, Lohani S, Bhattarai M. Correlation of serum cholinesterase level, clinical score at presentation and severity of organophosphorous poisoning. JNMA; journal of the Nepal Medical Association 2008;47(170).
- Sungur M, Güven M. Intensive care management of organophosphate insecticide poisoning. Crit Care 2001;5(4):211–5.
- Karalliedde L, Baker D, Marrs TC. Organophosphateinduced intermediate syndrome: aetiology and relationships with myopathy. Toxicol Rev 2006;25(1):1–14.
- Malik GM, Mubarik M, Romshoo GJ. Organophosphorus poisoning in the Kashmir Valley, 1994 to 1997. N Engl J Med 1998;338(15):1078.
- Bhattacharyya K, Phaujdar S, Sarkar R, Mullick OS. Serum creatine phosphokinase: a probable marker of severity in organophosphorus poisoning. Toxicol Int 2011;18(2):117–23.
- G R, Gangannavar AA, K MKB, Rajoor U. Study of serum creatinine phosphokinase and serum lactate dehydrgenase as prognostic markers in organophosphorus poisoning. International Journal of Advances in Medicine 2020;7(7):1125–30.
- Biswas S, Ghosh R, Mandal A, Lapeña J, Roy D, Benito-León J. Is Serum Creatine Phosphokinase Level Useful to Predict the Severity of Organophosphate Poisoning? Medical research archives 2022;10(9).
- 15. Patil DSL, Vasepalli DP. Prognostic value of clinical and lab parameters in assessing the severity of organophosphorous compound poisoning. 4(1).
- 16. George EJ. Clinical profile and outcome of organophosphate poisoning cases in a tertiary care hospital in central Kerala. 2021;11(4).
- Sen R, Nayak J, Khadanga S. Study of serum cholinesterase, CPK and LDH as prognostic biomarkers in organophosphorus poisoning. International Journal of Medical Research and Review 2014;2(3):185–9.
- Palimar V, Arun M, Saralaya KM, Bhoopendra S. Spectrum of organophoshorous poisoning in Manipal. Medico-Legal Update 2005;5(2):55–7.
- Hawton K, Fagg J, Simkin S, Bale E, Bond A. Trends in deliberate self-harm in Oxford, 1985-1995. Implications for clinical services and the prevention of suicide. Br J Psychiatry 1997;171:556–60.

- Selvaraj T, Sudharson T. Demographic and Clinical Profile of Organophosphorus Poisoning cases in a Medical College Hospital, Tamil Nadu. Indian Journal of Forensic and Community Medicine 3(2):124–7.
- 21. Raddi D, V AG. Clinical profile of organophosphorus poisoning in a tertiary care hospital. Indian Journal of Basic & Applied Medical Research 2014;4(1):14–22.
- 22. Goswamy R, Chaudhuri A, Mahashur AA. Study of respiratory failure in organophosphate and carbamate poisoning. Heart Lung 1994;23(6):466–72.
- 23. Bobba R, Venkataraman BV, Pais P, Joseph T. Correlation between the severity of symptoms in organophosphorus poisoning and cholinesterase activity (RBC and plasma) in humans. Indian J Physiol Pharmacol 1996;40(3):249–52.
- Agarwal S, Bhatnagar VK, Agarwal A, Agarwal U. Impairment in Clinical Indices in Acute Organophosphate Insecticide Poisoning Patients in India. IJTO 2007;4(1).
- 25. Rabha M, Baruah C, Boro M. Clinical Profile of Acute Organophosphorous Poisoning with Special Reference to Serum Creatine Phosphokinase and Serum Lactate Dehydrogenase Levels as Prognostic Markers: A Tertiary Care Hospital Based Observational Study in North-East India. 3(6):83–8.
- 26. Vanneste Y, Lison D. Biochemical changes associated with muscle fibre necrosis after experimental organophosphate poisoning. Hum Exp Toxicol 1993;12(5):365–70.
- 27. John M, Oommen A, Zachariah A. Muscle injury in organophosphorous poisoning and its role in the development of intermediate syndrome. Neurotoxicology 2003;24(1):43–53.
- Matsumiya N, Tanaka M, Iwai M, Kondo T, Takahashi S, Sato S. Elevated amylase is related to the development of respiratory failure in organophosphate poisoning. Hum Exp Toxicol 1996;15(3):250–3.
- 29. Lee WC, Yang CC, Deng JF, Wu ML, Ger J, Lin HC, et al. The clinical significance of hyperamylasemia in organophosphate poisoning. J Toxicol Clin Toxicol 1998;36(7):673–81.
- Sahin I, Onbasi K, Sahin H, Karakaya C, Ustun Y, Noyan T. The prevalence of pancreatitis in organophosphate poisonings. Hum Exp Toxicol 2002;21(4):175–7.
- Soni P, Solu MG, Garg V, Pathria A, Shah S, Mundra A. Organophosphate Poisoning Predicting the Need for Mechanical Ventilator Support. 2016;4(6).
- 32. Gagarin PY, Rajagopal RL. Relation of Serum Cholinesterase with Clinical Severity and Treatment Outcomes of Organophosphorus Poisoning in a Tertiary Care Center, a Prospective Observational Study. 2020;7(5):7–11.