

Original Article

Epidemiological, clinical characteristics and outcomes of patients presenting with acute organophosphorus poisoning to a tertiary care institution in North India

Mohammad Hayat Bhat¹, Sajad Qadir Bhat², Ishrat Hussain Dar², Mohammad Afaan Bhat², Javaid Ahmad Bhat^{1*}

¹ Department of Endocrinology, Superspeciality Hospital, Srinagar, Kashmir, India

² Department of Medicine, Government Medical College, Srinagar, Kashmir, India

* Correspondence to: Javaid Ahmad Bhat, Department of Endocrinology, Superspeciality Hospital, Srinagar, Kashmir, India. E-mail: javaidrasool711@gmail.com

Received: 7 September 2022 / Accepted: 24 March 2023

Abstract

Organophosphates (OP) poisoning continues to be a major cause of deliberate self-harm and an important cause of morbidity and mortality in India. We aim to study the clinico-epidemiological features in patients presenting with OP poisoning. This prospective observational study involved 400 patients above 18 years of age admitted to the emergency department of a tertiary care institution in Northern India. The study cohort was graded into mild, moderate & severe based on the Peradeniya Organophosphorus Poisoning (POP) scale and degree of cholinesterase inhibition. The majority of patients (40%) in our study population were aged between 18–25 years. There was a significant female preponderance (82% vs. 18%; $P=0.000$). Suicide was the most common motive (93%) and ingestion was the most common mode of poisoning (99%). The muscarinic and nicotinic symptoms were common at the presentation. Seizures were present in 11% of patients. On the POP severity scale, mild, moderate and severe poisoning was seen at 50%, 35% and 15%, respectively. Cholinesterase inhibition was mild (>2 kU/L) in 31%, moderate (1–2 kU/L) in 46% and severe (<1 kU/L) in 23%. A total of 48% of patients had complications. Ventilator support was needed in 43% of the patients. An overall mortality of 18% was observed in the study. OP poisoning is a serious public health problem, but timely intervention may help to reduce morbidity and mortality, especially in a resource-limited country like India.

Keywords: organophosphorus poisoning, epidemiology, mortality, morbidity, glycemic status.

Introduction

Intentional self-poisoning involving agrochemical pesticides remains a major and serious public health problem throughout the developing world, accounting for 14–20% of global suicides, translating into an estimated 110,000–168,000 deaths each year [1]. In developing countries, especially rural Asian communities, including India, acute pesticide poisoning is a significant cause of morbidity and mortality [2, 3]. Despite being an agrarian country, there are no exact data estimates of pesticide poisoning available from India; however, it is the most common poisoning in India, accounting for nearly half of the admissions to emergency depart-

ments, as suggested by hospital-based studies. Suicide is usually the main intent of these poisonings [4]. Poisoning due to organophosphorus compounds (OPCs) is steadily increasing in India, with data indicating that 67% of the pesticide used is an insecticide, as against 44% globally, probably because of the wide availability and extensive use in agriculture and also because of unregulated sale of these items over the counter [5].

Organophosphorus compounds are cholinesterase-inhibiting chemicals derived from phosphorous-containing organic acids and are metabolically subjected to hydrolysis by esterases. Excess accumulation of acetylcholine at the cholinergic nerve endings all over the body involving the autonomic, neuromuscular, and CNS



synapses upon irreversible inhibition of acetylcholinesterase (AChE) by OPCs, results in a plethora of signs and symptoms that may culminate into the significant morbidity and or mortality [6]. The presence and severity of these symptoms depend partly on the degree of AChE suppression, though symptoms are not always present in AChE-depressed individuals.

The Three well-defined complications following classical organophosphorus poisoning include cholinergic crisis, intermediate syndrome, and organophosphorus-induced delayed neuropathy (OPIDN) [7].

The characteristic but treatable syndromes that characterize the exposure to OPCs warrant early recognition and timely intervention to avoid or mitigate the plethora of serious morbid complications and, thus, grim outcomes that may associate OPCs exposure. Despite advancements in managing organophosphorus poisoning patients, significant mortality among exposed patients is of concern. Therefore, it is important that the clinical features and other factors which indicate the severity of poisoning and the criteria to predict the need for ventilator support be identified at the initial examination. The present study aims to explore the clinical and epidemiological features in patients presenting with OP poisoning.

Material and methods

The present study, a prospective observational study involving 400 patients carried over a period of 2 years (October 2018 to October 2020), was conducted in the Postgraduate Department of Medicine, Government Medical College, Srinagar, Jammu and Kashmir, India, after obtaining the ethical clearance from the Institutional Ethical Committee.

Inclusion criteria

Patients aged 18 years or above admitted with an alleged history of organophosphorus poisoning (ingestion/inhalational/contact) and diagnosed with organophosphorus poisoning were included in the study after obtaining well-informed written consent from the patients or their relatives.

Exclusion criteria

1. Patients aged less than 18 years and/or with a history of diabetes mellitus, chronic liver disease, chronic kidney disease or any other co-

morbidity that could affect the glycemic status of the patients;

2. Patients referred, after treatment at other centers, to our center for further management with no baseline details available at the first presentation;
3. Patients who had consumed drugs, alcohol and mixed poisoning that could affect the glycemic status of the patients.

The presumptive diagnosis of OP poisoning was based on circumstantial evidence, history and characteristic clinical features. Blood samples for blood glucose and serum cholinesterase were collected at admission and processed in a fully automated auto-analyzer. Serum cholinesterase was estimated using the Beckman Coulter machine and analyzing the sample by spectrophotometry method, while blood sugar was determined by GOD-POD (Glucose oxidase and Peroxidase) method. HbA1C was measured in patients with random blood sugar (RBS) of >200 mg/dl and those with HbA1C of 6.5% were considered diabetic and excluded from the study.

Patients with organophosphorus poisoning were grouped into Hypoglycemia (RBS <70 mg/dl) group, Hyperglycemia (RBS >200 mg/dl) and Normoglycemia group (70–199 mg/dl) at the time of presentation. The clinical severity of acute organophosphorus poisoning was measured using Peradeniya Organophosphorus Poisoning (POP SCALE) [8].

Statistical analysis

The recorded data was compiled, entered in a Microsoft excel spreadsheet and analyzed using Epi Info. Continuous variables were summarized as mean±SD and categorical variables were summarized as percentages. The chi-square test was used to analyze the relationship between two categorical variables. One-Way ANOVA was used to analyze the difference between the two means. Pearson's correlation coefficient was used to analyze the relationship between two continuous variables. Landis and Koch's guidelines were used to assess the correlation coefficient. Two-sided p-values were reported and a p-value of less than 0.05 was considered statistically significant.

Results

A total of 400 consecutive patients were included in the study. The majority (40%) of the patients belonged

to the age group 18–25 years, followed by 25% to 26–35 years age group and 14% to 36–45 years. Female preponderance in our study comprised 82% of the study cohort. The majority (88%) of the cases hailed from rural areas while 12% were from urban areas. The baseline demographic, clinical and outcome of patients are depicted in Table 1 and Table 2. There were no gender-based

significant differences in various baseline, clinical characteristics and outcomes of patients depicted in Table 3. Ingestion was the main mode used for self-poisoning by 99% and only 1% of patients had inhalational exposure. Suicide was the main intent in 93% of patients, while only 7% took poison accidentally. At the time of presentation to the emergency department, 46% of patients

Table 1: Baseline demographic parameter and severity of poisoning among patients with organophosphorus poisoning.

Variables		Number	%
Gender	Male	72	18
	Female	328	82
Residence	Rural	353	88
	Urban	47	12
Type of exposure	Ingestion	396	99
	Inhalation	4	1
Intent of poisoning	Accidental	27	7
	Suicidal	373	93
Sensorium	Normal	215	54
	Altered	185	46
Heart rate (bpm)	60–100	277	69
	<60	60	15
	>100	63	16
Blood pressure (mmHg)	Normal	356	89
	Low (<90/60 mmhg)	15	4
	High (>130/80 mmhg)	29	7
Pupil Size	<2 mm	248	62
	Pinpoint	75	19
Fasciculation's	Generalized or Continuous	144	36
	Generalized & Continuous	71	18
Seizures	Present	45	11
	0–3 (Mild)	199	50
POP score	4–7 (Moderate)	142	35
	8–11 (Severe)	59	15
	<1 (Severe suppression)	90	23
Serum cholinesterase (kU/L)	1–2 (Moderate suppression)	185	46
	2–6 (Mild to normal)	125	31
	Normal	223	56
Plasma glucose (mg/dl)level at admission	Hyperglycemia (≥200)	119	30
	Hypoglycemia (<70)	58	14

Note: POP – Peradeniya organophosphorus poisoning scale.

Table 2: Biochemical characteristics and outcome of patients with organophosphorus poisoning.

Variables	Total Mean±SD (95% CI)
AGE (years)	33.60±13.76 (32.25–34.96)
Plasma glucose on admission (mg/dl)	143±85 (135–151)
HbA1C %	5.12±3.44 (4.63–5.61)
POP score	3.76±2.64 (3.50–4.01)
Hospital stay (days)	6.68±4.58 (6.23–7.13)
<7days n (%)	246 (61.5)
≥7days n (%)	154 (38.5)
Mechanical ventilation n (%)	173 (43.4)
Mortality n (%)	69 (17.3)
HDU requirement n (%)	209 (52.3)

Note: POP – Peradeniya organophosphorus poisoning scale; HDU – High dependency unit.

presented in altered sensorium, while 54% were conscious and rational. Heart rate was normal in 69% of patients; however, bradycardia was observed in 15%, while tachycardia was seen in 16% of patients. A total of 89% of patients were normotensive, while low blood pressure was seen in 4% compared to high blood pressure in 7% of patients.

Miosis was present in 62% of patients, with pin-point pupils in 19% and normal-size pupils in 19% of patients. Generalized or continuous fasciculation was seen in 36% of patients, and generalized and continuous fasciculation was seen in 18% of patients. There was no fasciculation seen in 46% of patients. Seizures were present in 11% of patients in our study population.

Table 3: Comparative analysis of baseline characteristics and outcome of patients as per gender.

Variable		Females n (%)	Males n (%)	P-value
Age		32.13±12.85	40.30±15.74	0.000
Residence	Rural	290 (82)	63 (18)	0.827
	Urban	38 (81)	9 (19)	
Glycosuria		168 (79)	45 (21)	0.082
	Mild	165 (82)	35 (18)	
POP score	Moderate	116 (83)	24 (17)	0.722
	Severe	47 (78)	13 (22)	
	<1 (Severe suppression)	77 (23)	13 (18)	
Serum cholinesterase (kU/L)	1–2 (Moderate suppression)	154 (41)	31 (43)	0.272
	2–6 (Mild to normal)	97 (26)	28 (39)	
Hospital Stay	<7 days	204 (62)	42 (58)	0.542
	≥7 days	124 (38)	30 (42)	
Intermediate syndrome		56 (79)	15 (21)	0.450
HDU requirement n (%)		171 (82)	38 (18)	0.921
Mechanical ventilation n (%)		143 (83)	30 (17)	0.749
Mortality n (%)		57 (83)	12 (17)	0.885

Note: POP – Peradeniya organophosphorus poisoning scale; HDU – High dependency unit.

According to the POP severity scale, 50% of patients had mild poisoning (POP Score 0–3), 35% had moderate poisoning (POP Score 4–7) and 15% of patients had severe poisoning (POP Score 8–11). Based on the level of serum cholinesterase, mild poisoning (>2 kU/L) was present in 31%, moderate poisoning (1–2 kU/L) in 46% and severe poisoning (<1 kU/L) in 23% of patients. Based on the POP score, there was a statistically significant negative correlation between serum cholinesterase level and clinical severity.

Based on the admission plasma glucose level, the prevalence of normoglycemia, hyperglycemia and hypoglycemia was 56%, 30% and 14%, respectively. Five patients (1%) had diabetic ketoacidosis (DKA). Hyperglycemia was found to have a statistically significant negative correlation with serum cholinesterase level; however, with hypoglycemia, serum cholinesterase level had a statistically significant positive correlation. The mean hospital stay of patients was 6.68 ± 4.58 days, with 61.5% staying for <7 days and 38.5% for more than 7 days. High dependency care was required in 52.3% and mechanical ventilation in 43.4% of patients. In our study, out of a total of 400 patients, 329 survived and 71 died, corresponding to overall mortality of 18%.

Discussion

The widespread and injudicious application of pesticides has emerged as a major public health problem globally, with the major brunt being born by developing countries. There is overwhelming evidence that pesticides pose a potential risk to humans and other life forms [9]. Exposure to toxic concentrations of OPs can result in acute poisoning, which may result in significant mortality and morbidity in addition to expenditure incurred for managing the complications associated with the poisoning. Multiple observational studies in healthcare settings have reported pesticide poisoning across India, the vast majority being deliberate self-poisonings rather than accidental exposure, with case fatality rates varying from approximately 5% to over 70%. Non-occupational incidents are usually suicidal in more than 90% of the cases, involving mostly youngsters and farmers, with a fatality rate of more than 10% [10]. The mean age of our study population was 33.60 ± 13.76 years, with a majority (40%) of patients in the age group of 18–25 years. These observations are in accordance with results obtained in a number of studies involving patients with acute OP poisoning [11, 12]. The higher prevalence of poisoning among young

adults reflects their vulnerability to stress, maladjustment, and immature psychological coping mechanisms during life's major stressors [13].

There was a significant female preponderance in cases of organophosphorus poisoning in our study cohort, reflecting the results that are consistent with the data from both national and international studies [14, 15]. However, in contrast to our observations, some studies have demonstrated male predominance in organophosphate poisoning [16]. The higher proportion of self-poisoning among females may be multifactorial, including cultural, religious and various social factors revealing inequality. Early marriages, domestic violence, and unwanted pregnancies contribute to more suicide attempts by women. Sometimes it is used as a last resort as a way out to protest against gender discrimination which is not uncommon in developing countries like India and may be an effective method to gain attention and sympathy in matters like marital discord [17]. Furthermore, a higher number of self-poisoning could result from misadventures, sometimes resorted to gain attention or express distress besides disillusionment [18]. Another factor that may have a role to play among women resorting to increased use of life-consuming substances like OPCs could be lower education level.

The majority of the patients in our study belonged to rural areas, which is consistent with observations in similar studies involving organophosphorus poisoning [19]. Increasing pesticide use in agricultural practices results in easy, unregulated and unlimited availability near homes to the rural population. This could be the obvious reason for the higher rates of self-poisoning. Interventions to limit access in such settings are complex and require the involvement of most rural adults rather than a select few [20].

This study found that 93% of organophosphate poisonings were intentional, whereas only 7% were accidental. Intentional self-poisoning was the most common cause of poisoning in similar other studies involving patients with OP poisoning [12, 21]. The high incidence of organophosphorus poisoning with suicidal intent could be one of many manifestations of the tragic consequences of the political unrest in this part of India, resulting in great suffering and mental trauma among the residents. Ingestion was the most common mode used for self-poisoning by the patients, which is in accordance with the observations in the previous studies [12, 21]. A total of 46% of patients presented with altered mental status in the form of inadequate response to verbal commands or no response to verbal commands.

A similar presentation was reported by Rao R et al., in 52% of patients and by Tripathi SK et al., in 60% of patients with organophosphorus poisoning [11, 22].

On checking the heart rate of patients, bradycardia, a result of muscarinic manifestation, was found in 15% of patients, while tachycardia, the result of action on sympathetic ganglia, was seen in 16% of patients. Heart rate was normal in 69% of patients. Our findings were comparable to results reported in earlier studies involving OP patients [11, 22]. Hypotension was documented in 4% while 7% had hypertension, similar to findings reported in a study by Rao R et al., reporting hypotension in 4% and hypertension in 10% of the cases [11]. The most marked muscarinic sign in our study population was miosis which was seen in 62% and pinpoint pupils in 19% of the patients. This is in congruence with studies conducted by Banerjee I et al. and Rao R et al., who reported miosis in 92% and 56% of patients, respectively [11, 14]. The most marked nicotinic effect in our study population was fasciculation, seen in 54% of the patients. Fasciculation was reported in 55% and 43% of patients by Goel A et al. and Tripathi SK et al., respectively [22, 23].

One of the observations of our study was the presence of seizures in 11% of the study population. Our results are consistent with the findings of Jamil Hamida, who observed in a review study of 755 patients that seizures occurred in 8% of the cohort [24]. In an earlier study by Balali-Mood M et al. of 63 patients poisoned with a variety of pesticides the incidence of convulsions was 14% [25].

In our study, plasma glucose level was checked in all patients on admission; 56% of our patients had normal glucose levels, 30% had hyperglycemia and 14% had hypoglycemia. Five patients (1%) presented with diabetic ketoacidosis [26]. Our findings were consistent with results from earlier studies which documented the presence of glycemic abnormalities, including hyperglycemia, hypoglycemia and diabetic ketoacidosis in patients with OP poisoning [11]. Several mechanisms have been forwarded to explain the occurrence of hyperglycemia arising as a result of both acute and chronic exposure to OP poisons. The role of oxidative stress, inhibition of paraoxonase, stimulation of adrenal glands and release of catecholamines and effect on the metabolism of liver tryptophan are some of the proposed mechanisms to explain the derangement of glucose metabolism in OP poisoning [27].

One of the observations of our study was that patients with extremes of glycemia (hypoglycemia or hyperglycemia) at the presentation had moderate to se-

vere disease in the majority (95% and 93%, respectively) of them compared to 15% of the patients with normoglycemia who had a rather mild form of the disease in the majority (85%) [26]. This observation gives credence to the fact that patients of acute OP poisoning with dysglycemia suffer from higher grades of severity, thus undermining the importance of knowing the glycemic status at the presentation and warrants due consideration in decision-making regarding the need for close observation and admission to the intensive care unit.

A total of 31% of the study cohort had serum cholinesterase >2 kU/L, 46% had serum cholinesterase of 1-2 kU/L and 23% had serum cholinesterase <1 kU/L. These results were in accordance with the studies done by Ravi BN et al., who observed serum cholinesterase <1 kU/L in 23%, 1-2 kU/L in 48% and >2 kU/L in 29% of patients and Rao R et al., in their fifty patients found that 26%, 42% and 32% of the patients had mild, moderate and severe depression of serum cholinesterase levels [11, 12].

It was further observed that severe suppression of serum cholinesterase (<1 kU/L) was seen in only 6% of normoglycemic patients compared to 44% and 40% in patients with hyperglycemia and hypoglycemia, respectively, indicating greater suppression of cholinesterase was seen in the patients with OP poisoning with extremes of blood sugar. Similar results were documented in the previous study, where they found that severe suppression of serum cholinesterase was seen in 30% of hypoglycemic patients and 50% of hyperglycemic patients [28].

According to the POP scale, 50% of patients had mild poisoning, 35% had moderate poisoning, and 15% of patients had severe poisoning. Our findings were comparable to studies conducted by Nermeen AM et al., [29], who observed that 51.7% had mild, 33.3% moderate and 15% had severe poisoning and Chaudhary S et al., [30] found in their cohort that 35.33% were in the mild category, 50% in moderate category and 14.67% patients fell in the severe category.

Limitations to this study include: 1. Hospital-based studies cannot represent trends in the general population; 2. Lack of identification of the type OP compound, amount of substance consumed and its subsequent impact on clinical outcome.

Conclusion

OP poisoning is a serious public health problem but preventable with timely, evidence-based, and often

low-cost interventions. Hazardous occupational practices and unsafe storage expose millions of people to danger. However, deliberate self-poisoning causes the great majority of deaths and the immense strain pesticides put on hospital services, particularly in Asia. There is a need for community awareness about organophosphate poisoning, especially in the highly affected areas. Better regulatory control of pesticide handling and use will also reduce the burden of pesticide poisoning.

Conflict of interest

The authors declare no conflict of interest.

Ethics approval

The approval for this study was obtained from the Ethics Committee of the Government Medical College, Srinagar, Kashmir (approval ID: ECR/1422/inst/JK/2022 dated 16 July 2020).

Consent to participate

Written informed consent was obtained from the participants.

References

- Eddleston M, Phillips MR. Self poisoning with pesticides. *BMJ*. 2004;328(7430):42-44. doi:10.1136/bmj.328.7430.42
- Jeyaratnam J. Acute pesticide poisoning: a major global health problem. *World Health Stat Q Rapp Trimest Stat Sanit Mond*. 1990;43(3):139-144.
- Singh S, Wig N, Chaudhary D, Sood NK, Sharma BK. Changing pattern of acute poisoning in adults: Experience of a large north-west Indian hospital (1970-1989). *J Assoc Physicians India*. 1997;45(3):194-197.
- Malik GM, Mubarik M, Romshoo GJ. Organophosphorus poisoning in the Kashmir Valley, 1994 to 1997. *N Engl J Med*. 1998;338(15):1078. doi:10.1056/NEJM199804093381520
- Mathur SC, Tannan SK. Future of Indian pesticides industry in next millennium. *Pestic Inf*. 1999;24(4):9-23.
- Nigg HN, Knaak JB. Blood cholinesterases as human biomarkers of organophosphorus pesticide exposure. *Rev Environ Contam Toxicol*. 2000;163:29-111. doi:10.1007/978-1-4757-6429-1_2
- Karalliedde L, Senanayake N. Organophosphorus insecticide poisoning. *Br J Anaesth*. 1989;63(6):736-750. doi:10.1093/bja/63.6.736
- N S, Hj de S, L K. A scale to assess severity in organophosphorus intoxication: POP scale. *Hum Exp Toxicol*. 1993;12(4). doi:10.1177/096032719301200407
- Jeyaratnam J. Health problems of pesticide usage in the Third World. *Br J Ind Med*. 1985;42(8):505-506. doi:10.1136/oem.42.8.505
- Eddleston M, Phillips MR. Self poisoning with pesticides. *BMJ*. 2004;328(7430):42-44. doi:10.1136/bmj.328.7430.42
- Rao R, Raju G. Random blood sugar levels and pseudocholinesterase levels their relevance in organophosphorus compound poisoning. *Int J Community Med Public Health*. 2016;3:2757-2761.
- Bn R, S MU, S PBB. Study of Hyperglycemia and Its Association with Pseudocholinesterase Levels and Severity of Organophosphorus Poisoning. *Int J Health Sci Res*. 2018;8(9):20-26.
- Basnet A, Shrestha D, Chaulagain S, et al. Psychological and clinical-epidemiological profile of poisoning in Nepal: an institutional experience. *F1000Research*. 2021;10:556. doi:10.12688/f1000research.54327.1
- Banerjee I, Tripathi SK, Roy AS. Clinico-epidemiological characteristics of patients presenting with organophosphorus poisoning. *North Am J Med Sci*. 2012;4(3):147.
- Gündüz E, Dursun R, Icer M, et al. Factors affecting mortality in patients with organophosphate poisoning. *JPMA J Pak Med Assoc*. 2015;65(9):967-972.
- Mumtaz Ali S. Mortality in patients presenting with organophosphorus poisoning at Liaquat University of Medical and Health Sciences. *Pak J Med Sci*. 2011; 27(5):1022-4.
- Pearson V, Phillips MR, He F, Ji H. Attempted suicide among young rural women in the People's Republic of China: possibilities for prevention. *Suicide Life Threat Behav*. 2002;32(4):359-369. doi:10.1521/suli.32.4.359.22345
- Hawton K. Deliberate self-harm. *Med-ABINGDON-UK Ed*. 1996;24:77-80.
- Thunga G, Sam KG, Khera K, P S, ey, Sagar SV. Evaluation of incidence, clinical characteristics and management in organophosphorus poisoning patients in a tertiary care hospital. *J Toxicol Environ Health Sci*. 2010;2(5):73-76. doi:10.5897/JTEHS.9000029
- Zhang C, Hu R, Shi G, Jin Y, Robson MG, Huang X. Overuse or underuse? An observation of pesticide use in China. *Sci Total Environ*. 2015;538:1-6. doi:10.1016/j.scitotenv.2015.08.031
- Raddi D, Anikethana GV. Clinical profile of organophosphorus poisoning in a tertiary care hospital. *Indian J Basic Appl Med Res*. 2014;4(1):14-22.
- Tripathy SK, Rout PK, Debta N, et al. Study of clinical profile of organophosphorus poisoning with special reference to electrocardiographic changes and electrolyte derangement. *Int J Adv Med*. 2018;5(1):50-56. doi:10.18203/2349-3933.ijam20175520
- Goel A, Joseph S, Dutta TK. Organophosphate poisoning: predicting the need for ventilatory support. *J Assoc Physicians India*. 1998;46(9):786-790.
- Jamil H. Organophosphorus insecticide poisoning. *JPMA J Pak Med Assoc*. 1989;39(2):27-31.
- Balali-Mood M, Shariat M. Treatment of organophosphate poisoning. Experience of nerve agents and acute pesticide poisoning on the effects of oximes. *J Physiol-Paris*. 1998;92(5-6):375-378.
- Bhat MH, Bhat SQ, Dar IH, Bhat JA. Glycemic status at the presentation and its role as marker of severity and outcome in patients with organophosphorus poisoning. *Romanian J Diabetes Nutr Metab Dis*. 2021;28(4):369-375.
- Rahimi R, Abdollahi M. A review on the mechanisms involved in hyperglycemia induced by organophosphorus pesticides. *Pestic Biochem Physiol*. 2007;88(2):115-121.

28. Chandana V. A prospective study to assess glycemic status as a possible prognostic marker in non diabetic acute organophosphate poisoning patients. *Int J Adv Med.* 2020;7(3):464.
29. Khan S, Kumar S, Agrawal S, Bawankule S. Correlation of serum cholinesterase and serum creatine phosphokinase enzymes with the severity and outcome of acute organophosphorus poisoning: study in rural central India. *World J Pharm Pharm Sci.* 2016;5(4):1365-1373.
30. Chaudhary S, Kalmegh R. Study of role of prognostic markers in the management of organophosphorus poisoning patients. *Int J Res Med Sci.* 2018;6:1996-1999.