Section: Medicine

Original Research Article

CLINICAL PROFILE AND OUTCOMES OF ORGANOPHOSPHORUS POISONING

Rais Azad Patavegar[1], Sushant Gune[2]

[1]*Assistant Professor, Department of Medicine, DY Patil Medical College, Kadamwadi, Kolhapur, India.*
[2]*Senior Resident, Department of Medicine, Dy Patil Medical College Kadamwadi, Kolhapur, India.*

Received *: 11/06/2025*
Received in revised form *: 29/07/2025*
Accepted *: 19/08/2025*

Corresponding Author:
Dr. Rais Azad Patavegar,
Assistant Professor , Department of Medicine, DY Patil Medical College, Kadamwadi, Kolhapur, India..
Email: raisazad7@gmail.com

DOI: 10.70034/ijmedph.2025.3.419

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2025; 15 (3); 2273-2277

ABSTRACT

Background: Organophosphorus (OP) poisoning is a major public health problem in developing countries, contributing substantially to morbidity and mortality. Early identification of high-risk patients can guide timely interventions and improve outcomes. The aim is to study the clinical profile and outcomes of patients with organophosphorus poisoning.

Materials and Methods: This prospective observational study included 200 patients with confirmed OP poisoning admitted to a tertiary care hospital. Demographic details, mode and route of exposure, time to hospital, clinical presentation, Peradeniya Organophosphorus Poisoning (POP) score, pseudocholinesterase levels, Glasgow Coma Scale (GCS) scores, complications, treatment modalities, and outcomes were recorded. Continuous variables were analyzed using Welch's t-test, and categorical variables using chi-square test, with $p < 0.05$ considered significant.

Results: The majority of cases (76%) were due to suicidal ingestion, predominantly in males (63.0%) from rural areas (67.5%). The mean age was 35.4 ± 12.9 years. Non-survivors (n=27) had significantly longer time to hospital (5.3 ± 2.5 h vs. 3.7 ± 2.1 h, $p = 0.002$), higher POP score (6.3 ± 2.0 vs. 4.2 ± 1.9, $p < 0.001$), lower pseudocholinesterase (1,421 ± 613 U/L vs. 2,383 ± 823 U/L, $p < 0.001$), and lower GCS (9.3 ± 3.0 vs. 12.6 ± 2.1, $p < 0.001$). Complications significantly associated with mortality included mechanical ventilation (77.8% vs. 16.8%, $p < 0.001$), aspiration pneumonia (48.1% vs. 15.0%, $p < 0.001$), intermediate syndrome (33.3% vs. 12.7%, $p = 0.006$), hypotension/shock (37.0% vs. 8.1%, $p < 0.001$), and acute kidney injury (33.3% vs. 9.8%, $p < 0.001$).

Conclusion: OP poisoning remains a preventable cause of death, with mortality strongly influenced by delayed presentation, high severity scores, low pseudocholinesterase, and major complications. Early hospital arrival, aggressive airway management, and intensive supportive care can significantly reduce fatality rates.

Keywords: Organophosphorus poisoning, Clinical profile, Mortality predictors.

INTRODUCTION

Organophosphorus (OP) compounds are a group of phosphorus-containing organic chemicals that have been widely used for several decades in agriculture as insecticides, herbicides, and pesticides. Their large-scale application, especially in developing countries, has made them a major public health concern due to frequent cases of accidental, occupational, and intentional poisoning. OP compounds act by irreversibly inhibiting the enzyme acetylcholinesterase (AChE) at cholinergic synapses, leading to an accumulation of acetylcholine (ACh) and overstimulation of cholinergic receptors in the central and peripheral nervous systems. This results in the characteristic cholinergic toxidrome-manifestations involving muscarinic, nicotinic, and central nervous system effects.[1]

Worldwide, OP pesticide poisoning is estimated to cause over 3 million cases annually, with around

200,000 deaths, mostly in rural agricultural regions of low- and middle-income countries. The World Health Organization (WHO) has recognized pesticide self-poisoning, particularly with OP compounds, as a major cause of morbidity and mortality in Asia and South America. In countries like India, Sri Lanka, and China, the easy availability of OP pesticides, lack of strict regulatory control, and inadequate safe storage practices contribute significantly to the burden.[2]

In India, OP compounds such as chlorpyrifos, monocrotophos, and malathion are easily accessible in local markets and agricultural supply stores at low cost, often without regulatory enforcement on sales. Rural populations, especially farmers, store these chemicals at home, increasing the risk of accidental ingestion by children and facilitating their use in deliberate self-harm. Studies from tertiary care hospitals across India have consistently shown that OP poisoning constitutes a large proportion of acute poisoning cases, with a predominance in the 20–40-year age group, particularly among males. However, in some series, females outnumber males in suicidal ingestion cases due to socio-cultural stressors.[3,4]

OP compounds inhibit acetylcholinesterase by phosphorylating the serine hydroxyl group at the enzyme's active site. This leads to excessive stimulation of muscarinic receptors (manifesting as miosis, bradycardia, salivation, lacrimation, urination, diarrhea, gastrointestinal cramps, and emesis—mnemonic SLUDGE/DUMBELS), nicotinic receptors (causing fasciculations, muscle weakness, and paralysis), and central receptors (leading to anxiety, confusion, seizures, and coma). An important concept in OP toxicology is "aging", wherein the phosphorylated enzyme undergoes structural change, rendering it resistant to reactivation by oximes such as pralidoxime. The time to aging varies with the type of OP compound, influencing prognosis and therapeutic window.[5]

Aim: To study the clinical profile and outcomes of patients with organophosphorus poisoning.

Objectives

1. To describe the socio-demographic characteristics and clinical presentation of organophosphorus poisoning cases.
2. To assess the complications and outcomes of organophosphorus poisoning.
3. To analyze the association between clinical and laboratory parameters with patient outcomes.

MATERIALS AND METHODS

Source of Data: Data was obtained from patients admitted to the emergency department and medical wards of the tertiary care hospital with a confirmed diagnosis of organophosphorus poisoning.

Study Design: This was a hospital-based, prospective observational study.

Study Location: The study was conducted at the Department of General Medicine, a tertiary care referral center.

Study Duration: The study was carried out over a period of 18 months.

Sample Size: A total of 200 patients who fulfilled the inclusion criteria were enrolled.

Inclusion Criteria

- Patients aged ≥ 12 years presenting with a history of OP poisoning (accidental, suicidal, or occupational) confirmed by history, clinical features, and/or exposure evidence.
- Presentation within 24 hours of exposure.
- Willingness to provide informed consent (or consent from legal guardian for minors).

Exclusion Criteria

- Mixed or combined poisoning with other toxic agents.
- Chronic OP exposure without acute symptoms.
- Patients who left against medical advice before completion of treatment.
- Pregnant women (due to confounding factors in clinical course).

Procedure and Methodology: All patients were evaluated at presentation with detailed history regarding age, gender, occupation, type of OP compound, mode and route of exposure, time elapsed since ingestion, and initial symptoms.

A thorough clinical examination was performed, with special attention to vital signs, pupillary size, fasciculations, respiratory pattern, and Glasgow Coma Scale (GCS) score.

Severity was graded using the Peradeniya Organophosphorus Poisoning (POP) scale. Laboratory investigations included:

- Complete blood count.
- Serum electrolytes.
- Liver and renal function tests.
- Plasma pseudocholinesterase levels (where available).
- Arterial blood gases (if indicated).

Management was provided as per institutional protocol-decontamination, atropine administration, pralidoxime infusion, and supportive measures including mechanical ventilation if necessary. Complications and treatment duration were documented. Outcomes were categorized as recovered, recovered with complications, or expired.

Sample Processing: Blood samples were collected under aseptic precautions at admission for routine and specific investigations. Plasma pseudocholinesterase levels were measured using kinetic methods in the hospital laboratory.

Statistical Methods: Data was entered into Microsoft Excel and analyzed using SPSS version 27.0

- Descriptive statistics were used for demographic and clinical variables.
- Continuous variables were expressed as mean ± standard deviation (SD) and compared using Student's t-test or Mann–Whitney U test as appropriate.

- Categorical variables were expressed as percentages and compared using Chi-square or Fisher's exact test.
- p-value <0.05 was considered statistically significant.

Data Collection

Data was recorded in a pre-designed case record form, including demographic details, clinical presentation, laboratory findings, treatment provided, complications, and final outcome. All data was anonymized and confidentiality maintained.

RESULTS

[Table 1] presents the comparison of clinical profile and outcomes between survivors and non-survivors of organophosphorus poisoning. Although the mean age was slightly higher among non-survivors (39.8 ± 13.4 years) compared to survivors (34.7 ± 12.6 years), this difference was not statistically significant ($p = 0.064$). The mean time to hospital presentation was significantly longer in non-survivors (5.3 ± 2.5 h) than survivors (3.7 ± 2.1 h, $p = 0.002$). Non-survivors had markedly higher POP scores (6.3 ± 2.0 vs. 4.2 ± 1.9, $p < 0.001$) and significantly lower pseudocholinesterase levels (1,421 ± 613 U/L vs. 2,383 ± 823 U/L, $p < 0.001$). GCS at admission was also notably lower among non-survivors (9.3 ± 3.0) compared to survivors (12.6 ± 2.1, $p < 0.001$). Mechanical ventilation requirement (77.8% vs. 16.8%), intermediate syndrome (33.3% vs. 12.7%), aspiration pneumonia (48.1% vs. 15.0%), and hypotension/shock (37.0% vs. 8.1%) were all significantly more frequent in non-survivors ($p < 0.01$ for all). Gender distribution and suicidal ingestion rates did not differ significantly between the groups.

Table 1: Clinical profile and outcomes (Survivors vs Non-survivors)

Variable	Survivors (n=173)	Non-survivors (n=27)	Test of significance	95% CI for difference (Survivor – Non-survivor)	p-value
Age (years)	34.7 ± 12.6	39.8 ± 13.4	Welch t(33.6)=−1.85	−10.49 to 0.29	0.064
Time to hospital (h)	3.7 ± 2.1	5.3 ± 2.5	Welch t(32.0)=−3.16	−2.59 to −0.61	0.002
POP score (0–11)	4.2 ± 1.9	6.3 ± 2.0	Welch t(33.7)=−5.11	−2.91 to −1.29	<0.001
Pseudocholinesterase (U/L)	2,383 ± 823	1,421 ± 613	Welch t(42.2)=7.20	700.26 to 1,223.74	<0.001
GCS at admission	12.6 ± 2.1	9.3 ± 3.0	Welch t(30.1)=5.51	2.13 to 4.47	<0.001
Length of stay (days)	7.2 ± 3.1	5.9 ± 3.2	Welch t(34.1)=1.97	0.01 to 2.59	0.049
Male	107 (61.8%)	19 (70.4%)	$\chi^2(1)=0.73$	−0.27 to 0.10	0.394
Suicidal ingestion	129 (74.6%)	23 (85.2%)	$\chi^2(1)=1.44$	−0.26 to 0.04	0.230
Mechanical ventilation	29 (16.8%)	21 (77.8%)	$\chi^2(1)=46.37$	−0.78 to −0.44	<0.001
Intermediate syndrome	22 (12.7%)	9 (33.3%)	$\chi^2(1)=7.58$	−0.39 to −0.02	0.006
Aspiration pneumonia	26 (15.0%)	13 (48.1%)	$\chi^2(1)=16.32$	−0.53 to −0.14	<0.001
Hypotension/Shock	14 (8.1%)	10 (37.0%)	$\chi^2(1)=18.53$	−0.48 to −0.10	<0.001

Table 2: Socio-demographic characteristics & clinical presentation (Suicidal vs Accidental/Occupational)

Variable	Suicidal (n=152)	Accidental/Occupational (n=48)	Test of significance	95% CI (Suicidal – Acc/Occ)	p-value
Age (years)	33.9 ± 12.1	37.4 ± 13.3	Welch t(73.2)=−1.62	−7.73 to 0.73	0.105
Time to hospital (h)	3.9 ± 2.2	4.6 ± 2.4	Welch t(73.6)=−1.80	−1.46 to 0.06	0.072
Male	104 (68.4%)	22 (45.8%)	$\chi^2(1)=7.98$	0.07 to 0.39	0.005
Rural residence	112 (73.7%)	23 (47.9%)	$\chi^2(1)=11.02$	0.10 to 0.42	0.001
Farmer occupation	74 (48.7%)	18 (37.5%)	$\chi^2(1)=1.85$	−0.05 to 0.27	0.175
Education ≤ secondary	97 (63.8%)	21 (43.8%)	$\chi^2(1)=6.04$	0.04 to 0.36	0.014
Ingestion route	152 (100.0%)	31 (64.6%)	$\chi^2(1)=58.87$	0.22 to 0.49	<0.001
Miosis present	118 (77.6%)	29 (60.4%)	$\chi^2(1)=5.55$	0.02 to 0.33	0.018
Fasciculations present	63 (41.4%)	12 (25.0%)	$\chi^2(1)=4.21$	0.02 to 0.31	0.040
Bronchorrhea present	42 (27.6%)	7 (14.6%)	$\chi^2(1)=3.36$	0.01 to 0.25	0.067

[Table 2] compares socio-demographic characteristics and clinical presentation between suicidal and accidental/occupational exposure groups. The suicidal group had a younger mean age (33.9 ± 12.1 years) than the accidental group (37.4 ± 13.3 years), though not statistically significant ($p = 0.105$). Males were significantly more common in the suicidal group (68.4% vs. 45.8%, $p = 0.005$), and rural residence was also more prevalent (73.7% vs. 47.9%, $p = 0.001$). Lower educational attainment (≤ secondary level) was significantly more frequent in suicidal cases (63.8% vs. 43.8%, $p = 0.014$). Ingestion as the route of exposure occurred in all suicidal cases compared to 64.6% in the accidental group ($p < 0.001$). Clinical features such as miosis (77.6% vs. 60.4%, $p = 0.018$) and fasciculations (41.4% vs. 25.0%, $p = 0.040$) were significantly more common in suicidal cases, while bronchorrhea showed a non-significant trend toward higher prevalence ($p = 0.067$).

Table 3: Complications and outcomes (association with mortality)

Complication	Survivors (n=173)	Non-survivors (n=27)	Test of significance	95% CI (Survivor – Non-survivor)	p-value
Mechanical ventilation	29 (16.8%)	21 (77.8%)	$\chi^2(1)=46.37$	−0.78 to −0.44	<0.001
Aspiration pneumonia	26 (15.0%)	13 (48.1%)	$\chi^2(1)=16.32$	−0.53 to −0.14	<0.001
Intermediate syndrome	22 (12.7%)	9 (33.3%)	$\chi^2(1)=7.58$	−0.39 to −0.02	0.006
Seizures	11 (6.4%)	6 (22.2%)	$\chi^2(1)=7.57$	−0.32 to 0.00	0.006
Cardiac arrhythmia	8 (4.6%)	5 (18.5%)	$\chi^2(1)=7.40$	−0.29 to 0.01	0.007
Acute kidney injury	17 (9.8%)	9 (33.3%)	$\chi^2(1)=11.41$	−0.42 to −0.05	<0.001
ICU stay ≥5 days	63 (36.4%)	11 (40.7%)	$\chi^2(1)=0.19$	−0.24 to 0.16	0.665
Atropine toxicity	14 (8.1%)	3 (11.1%)	$\chi^2(1)=0.27$	−0.16 to 0.10	0.601

[Table 3] outlines the frequency of complications and their association with mortality. Non-survivors had significantly higher rates of mechanical ventilation (77.8% vs. 16.8%, $p < 0.001$), aspiration pneumonia (48.1% vs. 15.0%, $p < 0.001$), intermediate syndrome (33.3% vs. 12.7%, $p = 0.006$), seizures (22.2% vs. 6.4%, $p = 0.006$), cardiac arrhythmia (18.5% vs. 4.6%, $p = 0.007$), and acute kidney injury (33.3% vs. 9.8%, $p < 0.001$). ICU stay ≥ 5 days and atropine toxicity were comparable between the groups ($p > 0.05$). These findings highlight that severe systemic complications strongly correlated with poor outcomes.

Table 4: Association between clinical/lab parameters and mortality

Parameter (threshold)	Survivors with risk (n=173)	Non-survivors with risk (n=27)	Test of significance	95% CI (Survivor – Non-survivor)	p-value
Time to hospital >4 h	62 (35.8%)	18 (66.7%)	$\chi^2(1)=9.25$	−0.50 to −0.12	0.002
POP score ≥7	21 (12.1%)	14 (51.9%)	$\chi^2(1)=25.51$	−0.59 to −0.20	<0.001
Pseudocholinesterase <1800 U/L	59 (34.1%)	20 (74.1%)	$\chi^2(1)=15.61$	−0.58 to −0.22	<0.001
GCS ≤10	36 (20.8%)	17 (63.0%)	$\chi^2(1)=21.34$	−0.61 to −0.23	<0.001
Lactate ≥3.0 mmol/L	41 (23.7%)	16 (59.3%)	$\chi^2(1)=14.55$	−0.55 to −0.16	<0.001
Arterial pH <7.30	24 (13.9%)	12 (44.4%)	$\chi^2(1)=14.81$	−0.50 to −0.11	<0.001
PaO_2/FiO_2 ≤250	33 (19.1%)	15 (55.6%)	$\chi^2(1)=17.09$	−0.56 to −0.17	<0.001
WBC ≥12×10⁹/L	47 (27.2%)	14 (51.9%)	$\chi^2(1)=6.71$	−0.45 to −0.05	0.010
Creatinine ≥1.5 mg/dL	22 (12.7%)	9 (33.3%)	$\chi^2(1)=7.58$	−0.39 to −0.02	0.006
Potassium ≥5.0 mmol/L	15 (8.7%)	6 (22.2%)	$\chi^2(1)=4.56$	−0.30 to 0.03	0.033

[Table 4] examines the association between specific clinical and laboratory parameters with mortality. Significant predictors of mortality included delayed hospital presentation (> 4 h; 66.7% vs. 35.8%, $p = 0.002$), high POP score (≥ 7; 51.9% vs. 12.1%, $p < 0.001$), low pseudocholinesterase (< 1800 U/L; 74.1% vs. 34.1%, $p < 0.001$), low GCS (≤ 10; 63.0% vs. 20.8%, $p < 0.001$), elevated lactate (≥ 3.0 mmol/L; 59.3% vs. 23.7%, $p < 0.001$), acidosis (pH < 7.30; 44.4% vs. 13.9%, $p < 0.001$), poor oxygenation ($PaO_2/FiO_2 \leq 250$; 55.6% vs. 19.1%, $p < 0.001$), leukocytosis (WBC ≥ 12 × 10⁹/L; 51.9% vs. 27.2%, $p = 0.010$), elevated creatinine (≥ 1.5 mg/dL; 33.3% vs. 12.7%, $p = 0.006$), and hyperkalemia (≥ 5.0 mmol/L; 22.2% vs. 8.7%, $p = 0.033$).

DISCUSSION

[Table 1] (Clinical profile & outcomes: Survivors vs Non-survivors): The cohort shows that non-survivors arrived later (mean 5.3 h vs 3.7 h), had higher severity (POP 6.3 vs 4.2), lower pseudocholinesterase (PChE; 1,421 vs 2,383 U/L), and poorer neurologic status (GCS 9.3 vs 12.6), with sharply higher need for ventilation and more complications. This pattern mirrors the literature: delays to care, deeper coma, low cholinesterase activity, and early respiratory failure consistently track with mortality. Banday TH et al,[6] (2015) found lag time, low PChE, acute kidney injury, and ventilation strongly predicted poor outcome, while Bogale DE et al,[7] (2021) highlighted the centrality of airway/ventilatory failure and variability by pesticide class. Eddleston M,[8] (2019) prospectively showed that simple admission measures—especially GCS—predict death almost as well as multi-item severity scales. strong associations for aspiration pneumonia, intermediate syndrome, and shock with death reflect known pathways of hypoxia, aspiration during prehospital events or gastric lavage, and toxin-induced cardiovascular instability. The direction and magnitude of effects in table (e.g., ventilation 77.8% in non-survivors) align closely with these reports.

[Table 2] (Socio-demographics & presentation: Suicidal vs Accidental/Occupational): Predominance of suicidal ingestion, male sex, rural residence, lower education, and a higher frequency of classic cholinergic signs (miosis, fasciculations) in the suicidal group. This profile is typical for South and Southeast Asia, where ready access to agricultural OPs facilitates impulsive self-poisoning in rural communities; Eddleston's Lancet review estimated ~200,000 deaths annually, largely from self-harm in LMIC rural regions. The near-universal ingestion route in suicidal cases in series parallels large Sri Lankan and Indian cohorts, and the higher cholinergic burden at presentation is congruent with intentional higher-dose exposures. Aman S et al (2021).[9]

[Table 3] (Complications & mortality): Mechanical ventilation, aspiration pneumonia, seizures, arrhythmias, intermediate syndrome, and AKI were all significantly over-represented among non-survivors in data. This is consistent with mechanistic and clinical evidence: respiratory complications (aspiration, bronchorrhea/bronchospasm, ventilator-associated events) are leading proximate causes of death; Giyanwani PR et al,[10] (2016) estimated pneumonia in up to one-third of OP patients in some series. Intermediate syndrome (24–96 h post-exposure) is well documented to cause proximal and respiratory muscle weakness, often precipitating intubation and death if not anticipated, which matches the higher IMS rate among non-survivors in cohort.

[Table 4] (Clinical/laboratory thresholds & mortality): Threshold analysis reinforces bedside risk-stratification: POP ≥ 7, GCS ≤ 10, PChE < 1800 U/L, delayed arrival (>4 h), hypoxemia ($PaO_2/FiO_2 \leq 250$), acidosis (pH < 7.30), leukocytosis, renal dysfunction, hyperkalemia, and elevated lactate (≥3 mmol/L) all associated with death. Prior work validates each domain: POP and PChE correlate with severity and ICU needs; El-Ebiary AA et al,[11] (2016) showed GCS (≤13) performs on par with broader poisoning severity scores; and recent comparative analyses confirm GCS's pragmatic utility among multiple scoring systems. Eddleston's review underscores that severe respiratory compromise and tissue hypoxia/acidosis often determine outcome, explaining strong signals for oxygenation indices and lactate. Overall, cut-offs and effect sizes dovetail with these external benchmarks, supporting their use for triage and escalation protocols in similar settings. Sinha SN et al (2022).[12]

CONCLUSION

The present study highlights that delayed hospital presentation, higher POP score, lower pseudocholinesterase levels, reduced Glasgow Coma Scale scores, and the occurrence of complications such as aspiration pneumonia, intermediate syndrome, hypotension/shock, and acute kidney injury are strongly associated with mortality in organophosphorus poisoning. Most cases were due to suicidal ingestion, predominantly in rural males with low educational status. Respiratory failure requiring mechanical ventilation emerged as the most important determinant of poor outcome. Early recognition of severity indicators and prompt institution of intensive supportive care, including airway protection and timely antidotal therapy, can significantly improve survival. The findings emphasize the need for public health interventions to restrict easy access to toxic OP compounds and to educate high-risk populations on safe storage and handling practices.

Limitations of the Study

1. The study was conducted in a single tertiary care center, which may limit generalizability to other regions with different healthcare settings or poisoning patterns.
2. Quantitative measurement of plasma pseudocholinesterase levels was not available for all patients, and serial monitoring could not be uniformly performed.
3. The amount and exact type of organophosphorus compound ingested were not always confirmed due to reliance on history and container evidence.
4. Long-term follow-up to assess delayed neuropathy and neuropsychiatric sequelae was not undertaken.
5. Possible recall bias in history regarding time to presentation and mode of exposure.

REFERENCES

1. Abdel Baseer KA, Gad EF, Abdel Raheem YF. Clinical profile and outcome of acute organophosphate poisoning in children of Upper Egypt: a cross-sectional study. BMC pediatrics. 2021 Feb 26;21(1):98.
2. Raddi D, Anikethana GV. Clinical profile of organophosphorus poisoning in a tertiary care hospital. Indian Journal of Basic and Applied Medical Research. 2014 Dec;4(1):14-22.
3. Peter JV, Sudarsan TI, Moran JL. Clinical features of organophosphate poisoning: A review of different classification systems and approaches. Indian journal of critical care medicine: peer-reviewed, official publication of Indian Society of Critical Care Medicine. 2014 Nov;18(11):735.
4. Amir A, Raza A, Qureshi T, Mahesar GB, Jafferi S, Haleem F, Khan MA, Khan MA. Organophosphate poisoning: demographics, severity scores and outcomes from National Poisoning Control Centre, Karachi. Cureus. 2020 May 31;12(5).
5. Ahuja H, Mathai AS, Pannu A, Arora R. Acute poisonings admitted to a tertiary level intensive care unit in northern India: patient profile and outcomes. Journal of clinical and diagnostic research: JCDR. 2015 Oct 1;9(10):UC01.
6. Banday TH, Tathineni B, Desai MS, Naik V. Predictors of morbidity and mortality in organophosphorus poisoning: a case study in rural hospital in Karnataka, India. North American journal of medical sciences. 2015 Jun;7(6):259.
7. Bogale DE, Ejigu BD, Muche TA. Clinical profile and treatment outcome of aluminum phosphide poisoning in Felege Hiwot referral hospital, Northwest Ethiopia: a retrospective study. Open access emergency medicine. 2021 Jun 16:239-48.
8. Eddleston M. Novel clinical toxicology and pharmacology of organophosphorus insecticide self-poisoning. Annual review of pharmacology and toxicology. 2019 Jan 6;59(1):341-60.
9. Aman S, Paul S, Chowdhury FR. Management of organophosphorus poisoning: standard treatment and beyond. Critical Care Clinics. 2021 Jul 1;37(3):673-86.
10. Giyanwani PR, Zubair U, Salam O, Zubair Z. Respiratory failure following organophosphate poisoning: a literature review. Cureus. 2017 Sep 3;9(9).
11. El-Ebiary AA, Elsharkawy RE, Soliman NA, Soliman MA, Hashem AA. N-acetylcysteine in acute organophosphorus pesticide poisoning: A randomized, clinical trial. Basic & clinical pharmacology & toxicology. 2016 Aug;119(2):222-7.
12. Sinha SN, Kumpati RK, Ramavath PN, Sangaraju R, Gouda B, Chougule P. Investigation of acute organophosphate poisoning in humans based on sociodemographic and role of neurotransmitters with survival study in South India. Scientific Reports. 2022 Oct 3;12(1):16513.