e-ISSN: 0975-5160, p-ISSN: 2820-2651

Available online on www.ijtpr.com

International Journal of Toxicological and Pharmacological Research 2023; 13(10); 310-315

Original Research Article

An Evaluation of the Clinical Profiles of Organophosphate Poisoning Patients

Ashish Bhansali

Assistant Professor Dept. of Medicine Dr. Rajendra Gode Medical College, Amravati

Received: 12-07-2023 / Revised 14-08-2023 / Accepted 25-09-2023
Corresponding author: Dr. Ashish Bhansali
Conflict of interest: Nil

Abstract:

Background: In agriculture, phosphate esters, often known as organophosphates (OP), are the most commonly utilized chemicals as pesticides. Intentional or inadvertent toxic substance exposure is also not unusual, particularly in rural regions. Acute poisoning typically presents as a cholinergic crisis, with respiratory and cardiovascular failure accounting for the majority of fatalities. In the developing world, acute organophosphorus (OP) pesticide poisoning is common. Since the nation is mostly an agricultural one, pesticides and insecticides are widely employed in agriculture, and the general public has easy access to these hazardous chemicals. Determining the type of substance, clinical signs, demographic profile, and outcome of organophosphate poisoning cases that present to an Indian hospital was the aim of the study.

AIM: The aim of the study was to identify the nature of the demographic profile, type of compound, clinical manifestations, and outcome of organophosphate poisoning presenting.

Material and Method: This study was a descriptive cross-sectional investigation carried out in the intensive care units for patients admitted with a history of acute organophosphorus poisoning in the department of General Medicine. Ninety, rounded to ninety-five, would be the final needed sample size. The dichotomous outcome of the study was death or release from the hospital. Soon after admission, a thorough medical history was obtained in accordance with the proforma, and a systemic and general physical examination were conducted. Every four hours during the day, or more often depending on the situation, a general physical examination was performed along with the recording of vital parameters. OPC poisoning symptoms include fasciculations, miosis, diaphoresis, oronasal foaming, the smell of the poison, and the involuntary passage of feces and urine.

Results: Males made up 77% of the participants, with the age range of 21–40 years old accounting for the largest percentage (65%). Complications included cramping or soreness in the abdomen in 95% of cases, nausea and vomiting in 80%, and diarrhea in 15% of cases. Of the people experiencing respiratory system problems, seven (7%) experienced cyanosis, 25 (25%) had pulmonary edema, and 25 (25%) had oronasal foam. Of the persons experiencing cardiovascular problems, 6 (6%) had hypotension and 35 (35%) had bradycardia. 20% of subjects experienced urinary bladder incontinence, 58% sweated more than usual, 26% had normal pupils, and 70% of people had miosis. Of the participants that experienced nicotine symptoms, 25 (or 25%) had fasciculations in their skeletal muscles, and 37 (or 37%) suffered paralysis. Complications with the autonomic nervous system included tachycardia in 3 (3% of subjects) and hypertension in 4 (4%).

Conclusion: It is evident that OP poisoning is more common in younger people (those under 40), with a preponderance of men. Most of them had a high death rate and a moderate degree of poisoning. Patients exhibiting signs of moderate to severe OP poisoning require significantly more prompt delivery of an antidote with inadequate dosage and duration. Such individuals require close observation and monitoring along with high-quality supportive care.

Keywords: Organophosphorus, Pesticides, Muscarinic Manifestations, Nicotinic Manifestations and Poisoning

Introduction

Poisoning is a problem for world health. Most poisoning instances are caused by "organophosphorus compounds," also known as organophosphates and carbamates (OP), particularly in agricultural nations like India. [1] An estimated 385 million farmworkers (about 44% of the projected 860 million agricultural people worldwide) unintentionally come into contact with pesticides each year, resulting in 11,000 fatalities. [2] Because of their advantageous pharmacological characteristics, organophosphorus (OP) chemicals are frequently used as insecticides. [3] Due to their practically general use in farming and likely

ignorance of the potential severity of their effects, there is a marked increase in the risk of serious poisoning in rural regions. According to the WHO, there are three million cases of pesticide poisoning each year, with up to 300,000 fatalities. [4]

Organophosphorus compounds (OPCs) are widely employed as pesticides in horticulture and agriculture as well as in homes to manage vector-borne illnesses including dengue, malaria, and others. In the developing world, OPC poisoning claims over 200,000 lives annually. [5] The World Health Organization (WHO) said in 2012 that suicidal pesticide poisoning caused 370,000 deaths worldwide while accidental poisoning caused about 193,460 deaths. [6] The issue is more noticeable in Asia's rural areas. OPC poisoning-related deaths are more common in southern and central India. In rural India, poisoning ranks as the fourth most common cause of death. Poisoning from organophosphates and aluminum phosphide is widespread in North India. Organophosphates, also known as aluminum phosphide, were created to manage insects and other pests and are now a significant factor in poisoning fatalities. [7]

Compounds containing organophosphorus (OP) are employed as nerve gasses, herbicides, and chemical warfare agents. Since the nation is mostly an agricultural one, pesticides and insecticides are widely employed in agriculture, and the general public has easy access to these hazardous chemicals. [8] There are already about a hundred distinct OP chemicals in synthesis. Because highly toxic pesticides are easily accessible at stressful times, the majority of OP pesticide poisoning cases and the deaths that follow happen in developing nations as a result of intentional self-ingestion, especially in young, productive age groups. [9]

Organophosphorus chemicals are anti-acetylcholine esterase agents that cause toxicity by disrupting acetylcholine's normal function, which is crucial for the autonomic and central nervous systems as a neurotransmitter. Acetylcholine builds up at synapses and myoneural junctions due to OP's inhibition of the cholinesterase enzyme, which causes cholinergic overactivity. [10] Although there is variation in the clinical symptoms and indications according on the type of chemicals, amount consumed, severity, time interval between exposure, and hospital admission, respiratory failure and lung injury account for the majority of patient deaths. Clinicians could stratify patients based on their likelihood of worsening by using predicted prognostic characteristics. This is because vigorous care and early detection can often save lives. Several clinical and laboratory indicators have been employed to evaluate the extent of OPC poisoning and forecast the clinical course of patients who are hospitalized for the condition. [11]

According to reports, the death rate from OP poisoning in India is between 4 and 30%, with respiratory failure being the most common cause of death. Therefore, in certain situations, prompt and occasionally preventive mechanical ventilation may be essential. [12] Hence, the present study was conducted with the objective of correlating serum cholinesterase levels and POP scale scores with the requirement of ventilation support and with mortality.

Material and Methods

This study was a descriptive cross-sectional investigation carried out in the intensive care units for patients admitted with a history of acute organophosphorus poisoning in the department of General Medicine. Ninety, rounded to ninety-five, would be the final needed sample size. The dichotomous outcome of the study was death or release from the hospital. Soon after admission, a thorough medical history was obtained in accordance with the proforma, and a systemic and general physical examination were conducted. The demographic details (age, gender, occupation, socioeconomic status, marital status), as well as the histories (mode of poisoning, type of poison, route of poisoning, time of onset of symptoms after poison intake, history of alcoholism and other addictions, psychiatric illness, and specific symptoms of OPC poisoning), were recorded on a preformed proforma. Every four hours during the day, or more often depending on the situation, a general physical examination was performed along with the recording of vital parameters. The scent of the poison, miosis, diaphoresis, oronasal foaming, the involuntary passage of urine and stools, level of awareness, and fasciculations are symptoms unique to OPC poisonings.

Inclusion Criteria:

All patients in whom a provisional diagnosis of OPC poisoning was made based on the patient's clinical presentation/history as recorded from the patient's attendant/details of poison containers were included in the study.

Exclusion Criteria:

Exclusions from the study included patients with a history of bronchial asthma, cardiac illness, neuromuscular diseases, multiple compound/tablet poisoning, contradictory diagnoses regarding the compound, and patients who passed away within minutes of being admitted to the hospital, even before the first course of treatment could begin.

Based on the following criteria, patients were diagnosed with Intermediate condition (IMS): a history of acute OP poisoning, the existence of clinical manifestations of the condition, and the ability to recover from the acute cholinergic crisis associated with the syndrome. [13] The

aforementioned selection criteria were used to assess patients with acute organophosphorus poisoning who were admitted to the intensive care unit. Once the participant or responsible next of kin provided written informed consent, the eligible participants underwent a comprehensive history as well as a clinical and biochemical assessment in accordance with the pre-tested proforma. Based on the clinical score (POP scale) recorded at admission, the patients were divided into three categories: mild poisoning (POP score 0-3), moderate poisoning (POP score 4-7), and severe poisoning (POP score 8-11). Before beginning any intervention, blood samples were taken and sent for an assessment of the plasma cholinesterase level. The acute OP poisoning cases were graded as normal (>50%), mild (20-50%), moderate (10-20%) & severe (<10%) as per the noted cholinesterase activity (pseudocholinesterase). Case follow-up continued until the patient passed away or was released from the hospital, at which point several connections between mortality and the degree of poisoning (measured by POP scale and pseudocholinesterase levels) were examined.

Treatment Protocol:

The standard protocol for treating OPC poisoning was followed for all patients, which included decontamination, correction of metabolic abnormalities, reversal of cholinesterase inhibition with oxime nucleophiles (pralidoxime), and blockade of muscarinic hyperactivity with atropine or glycopyrrolate. Endotracheal intubation was used to maintain a sufficient airway in unconscious patients, and ventilatory support was provided as needed.

The length of stay (LOS), complications, necessary atropine dose, need for ventilatory support, length of support, and mortality were all considered clinical outcomes. Studies were conducted on correlations between the admission scores and the above indicated outcome variables. At the time of admission, laboratory tests included a complete blood count, a random blood sugar test, a renal function test, and a liver function test. The patients were routinely observed until the conclusion.

Statistical Analysis

The data were entered in MS Excel and SPSS (version 18.0) was used for data analysis. The chi-Square test was employed for analysis and $p<0.05$ was considered for defining the level of significance.

Result: -

A total of 95 subjects were included in the final analysis.

Table 1: Descriptive analysis of age and gender distribution. (N=95)

Parameter	Number (%)
Age group	
<20	7(7%)
21-40	65(65%)
41-60	21(21%)
>60	2(2%)
Gender	
Male	77(77%)
Female	18 (18%)

The majority of the subjects were males (77%) with the maximum belonging to the age group of 21-40 years (65%). The most common OPC to be consumed was Methyl parathion (27%), followed by Chlorpyrifos (22%) The least common compound was Phorate (4%).

Table 2: Descriptive analysis of clinical presentation in the study population

Clinical manifestations			
Muscarinic manifestations		**Nicotinic manifestations**	
GIT	Number (%)	Skeletal muscle	Number (%)
Nausea and vomiting	80 (80%)	Fasciculations	25(25%)
Abdominal pain/cramps	95 (95%)	Paralysis	37(37%)
Diarrhea	15 (15%)	Autonomic nervous system	
Respiratory system		Tachycardia	3(3%)
Oronasal froth	25 (25%)	Hypertension	4(4%)
Cyanosis	7 (7%)	CNS manifestations	
Pulmonary edema	25 (25%)	Altered consciousness	46(46%)
Cardiovascular system		Coma	17(17%)
Bradycardia	35(35%)	Intermediate syndrome	4(4%)
Hypotension	6 (6%)	OPIDPN	0(0%)
Eyes			

Miosis	70 (70%)		
Normal pupil	26 (26%)		
Exocrine glands			
Increased sweating	58 (58%)		
Urinary bladder			
Incontinence	20(20%)		

complications were abdominal pain/cramps in 95%, followed by nausea and vomiting in 80% of participants, and diarrhea in 15% of participants. Among respiratory system complications, 25 (25%) participants had Oronasal froth, 25 (25%) participants had Pulmonary edema and 7 (7%) participants had Cyanosis. Of the persons experiencing cardiovascular problems, 6 (6%) had hypotension and 35 (35%) had bradycardia. Twenty percent of subjects experienced urinary bladder incontinence, 58% sweated more than usual, 26% had normal pupils, and 70% of people had miosis. Of the participants that experienced nicotine symptoms, 25 (or 25%) had fasciculations in their skeletal muscles, and 37 (or 37%) suffered paralysis. Complications with the autonomic nervous system included tachycardia in 3 (3% of subjects) and hypertension in 4 (4%). The following are examples of CNS complications: 4(4%) have Intermediate syndrome, 17(17%) have coma, and 46(46%) have altered consciousness.

Discussion

Since Clermont created the first organophosphorus molecule in 1857, over 50,000 other organophosphate compounds have been synthesized. Organophosphates are widely used pesticides that have the potential to cause significant illness and mortality. With a mortality rate ranging from 10 to 22%, the clinical signs include the typical cholinergic syndrome, flaccid paralysis, and intractable seizures. The validation of clinically meaningful markers that may be directly helpful in cases of acute OP poisoning was the main emphasis of the current investigation. According to earlier research of a similar nature, young adults had a noticeably higher prevalence of OP poisoning. [14]

Laudari et al2011 [15] further observed an increasing amount of OP poison intake (>40 ml) to be having a significant correlation with the increase in the mortality rate. Thungs et al 2010 [16] also reported the incidence of death (6.3%) to be higher in participants having taken >30 ml compound than those with<30 ml intake (2.7%) during acute OP poisoning episodes. Ozone poisoning is more common in developing nations like India due to the extensive use and accessibility of OP in agriculture. Accidental poisoning is less prevalent than suicidal ingestion. It is becoming more common among rural residents and is widespread. Every patient in our research took OP with the intention of hurting themselves. [17]

A study done by Agarwal et al.2007 [18] reported 67.4% of the cases had suicidal intent, whereas 15.8% of the cases were due to accidental poisoning. However, a study by Khan FY et al.2006 [19] reported a much higher incidence of accidental exposure in 87.3% of patients in Qatar. No example of homicidal poisoning was found in our analysis; instead, 76% of cases were suicidal, and 24% of cases were accidental poisoning. This is likely due to the fact that these compounds are inexpensive, readily accessible over-the-counter, and frequently employed as a significant pesticide in agricultural cultivation across India. Studies done by Gannur DG et al.2008 [20] Nigam M et al.2004 [21], revealed the highest incidence of poisoning in the young age group of 16–30 years. These investigations and our observation were similar. The younger generation, which makes up the working class and has greater responsibility, is more impetuous and ambitious. They are therefore particularly susceptible to different emotional conflicts during this stage of life.

A similar trend was also observed by Padmanaba et al 2011 [15] and Joshi et al 2016 [7] where the male-to-female ratio is 1.2:1. This could be because men make up the majority of those working in outdoor fields; that is, they are more likely to be the ones spraying crops in farms. In this study, methyl parathion was ingested more frequently than any other toxin (27%) and chlorpyrifos (22%) came second. Methyl parathion was also the most common poison detected in the studies of Banerjee et al. 2012 [17]. This variation in the type of poison consumed can be attributed to the regional availability of pesticides in different regions.

Sungur et al.2001 [22] reported a mortality rate of 50% for the patients who were mechanically ventilated and 21.6% for the patients who are not mechanically ventilated in their study. Kavya et al.2012 [12] observed a significant correlation between the severity of poisoning and the serum cholinesterase at the time of initial presentation of the patients. Additionally, there was a positive correlation between lower Pseudocholinesterase levels and POP scores and the requirement for ventilation.

The longer hospital admission wait time in the current study may be the cause of the elevated death rate. The majority of the time between the

poisoning and the start of treatment was spent getting to the hospital and making transportation arrangements. Reducing the amount of time that passes between ingesting a poison and starting a certain treatment could potentially lower the risk of dying. The primary weakness of the study was its cross-sectional and descriptive design, which hindered us from conducting any hypothesis testing. There would have been bias if the compound's nature had been assumed based only on its history and description in a few instances. Because of the smaller sample size and smaller catchment region, the study's conclusions are not as broadly applicable. Two significant limitations of the current study are the comparatively smaller sample size and the cases being primarily from rural populations. Larger sample sizes and well-proportioned urban areas are suggested for studies to support the current study's conclusions.

When medication is administered correctly and promptly, symptoms go away; when treatment is delayed, there is a considerable risk of morbidity and mortality. Therefore, in order to start treatment as soon as possible and appropriately, the primary care physician has to be aware of the clinical signs and symptoms of OP compound poisoning.

Conclusion:

It is evident that OP poisoning is more common in younger people (those under 40), with a preponderance of men. Most of them had a high death rate and a moderate degree of poisoning. Patients exhibiting signs of moderate to severe OP poisoning require significantly more prompt delivery of an antidote with inadequate dosage and duration. Such individuals require close observation and monitoring along with high-quality supportive care. The negative effects of such poisoning may also be lessened by stringently enforcing the Pesticide Act and implementing a new government program aimed at educating the general public and young people about the potentially fatal effects of organophosphorus chemicals.

References:

1. Gunnell D, Eddleston M, Phillips MR, Konradsen F. The global distribution of fatal pesticide self-poisoning: systematic review. BMC Public Health. 2007;7:357.
2. Jeyaratnam J, de Alwis Seneviratne RS, Copplestone JF. Survey of pesticide poisoning in Sri Lanka. Bull World Health Organ. 1982;60 (4):615–9.
3. Taylor P. Anticholinesterase agents. Goodman and Gilman's The Pharmacological basis of Therapeutics. 11th ed. United States of America: Taylor and Francis; 2006:176-82.
4. Mancini F, Janice LS, O'Malley M. Reducing the incidence of acute pesticide poisoning by educating farmers on integrated pest management in South India. Int J Occup Environ Health. 2009;15(2):143- 51.
5. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet 2008; 371:59 7-607.
6. WHO Public health impact of pesticides used in agriculture Geneva, Switzerland: World Health Organization; 1990.
7. Casida JE, Quistad GB. Organophosphate toxicology: safety aspects of no acetylcholinesterase secondary targets. Chem Res Toxicol. 2 004;17:983–998.
8. Sharma B, Harish D, Sharma V, Vij K. The epidemiology of poisoning- An Indian viewpoint. J Foren Med Toxicol. 2002;19(2)5-11.
9. Behere PB, Behere AP. Farmers' suicide in Vidarbha region of Maharashtra state- A myth or reality? Indian J Psychiatry. 2008;50(2)124-27.
10. Zawar S. Correlation between plasma cholinesterase levels and clinical severity of acute organophosphate and carbamate poisoning. JAPI. 2001;149;91
11. Aygun D, Doganay Z, Altintop L, Guven H, Onar M, Deniz T, et al. Serum acetylcholinesterase and prognosis of acute organophosphate poisoning. J Toxicol Clin Toxicol. 2002; 40(7) 903-910.
12. Eddleston M. Patterns and problems of deliberate self-poisoning in the developing world. QJM. 2000;93:715–731.
13. De Bleecker JL. Intermediate syndrome: Prolonged cholinesterase inhibition. J Toxicol Clin Toxicol. 1993;31(1):197-9
14. Cherian M, Roshini C, Visalakshi J, Jeyaseelan L, Cherian A. Biochemical and clinical profile after organophosphorus poisoning A placebo-controlled trial using pralidoxime. J Assoc Physicians India. 2005;53;427-431.
15. Laudari S, Patowary BS. Analysis of Organophosphorus compound poisoning patients attending CMS-TH, Bharatpur, Nepal. J Coll Med Sci-Nepal. 2011;7:9-19.
16. Thungs G, Sam KG, Khera K, Pandey S, Sagar SV. Evaluation of organophosphorus poisoning cases in a tertiary care hospital. J Tox Env Health Sci. 2010; 2(5):73-6.
17. Banerjee, Indranil, S. K. Tripathi, and A. Sinha Roy. "Clinico-epidemiological characteristics of patients presenting with organophosphorus poisoning." North American journal of medical sciences, 2012;4(3):147
18. Agarwal SB, Bhatnagar VK, Agarwal A, Agarwal U, Venkaiah K, Nigam SK, et al. Impairment in clinical indices in acute

organophosphate insecticide poisoning patients in India. Internet J Toxicol 2007;4.
19. Khan FY, Kamha AM, Ibrahim AS, D'souza A. One-year study of patients with acute organophosphate insecticide poisoning admitted to the intensive care unit of Hamad General Hospital, Doha, State of Qatar. J Emerg Med Trauma Acute Care 2006; 6:16-20.
20. Gannur DG, Maka P, Narayan Reddy KS. Organophosphorus compound poisoning in Gulbarga region-A five-year study. Indian J Forensic Med Toxicol 2008; 2:1-6.
21. Nigam M, Jain AK, Dubey BP, Sharma VK. Trends of organophosphorus poisoning in Bhopal region an autopsy-based study. J Indian Acad Forensic Med 2004; 26:61-5.
22. Sungur M, Guven M. Intensive care management of organophosphate insecticide poisoning. Crit Care. 2001;5(4)211-215.