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A Study of Clinical Profile of Patients of Organophosphate Poisoning in a Teaching Hospital in Rural Maharashtra

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ABSTRACT

Background: *Organophosphates (OP) or phosphate esters are the most used compounds as pesticides in agriculture. Toxic exposure to these substances, accidental or intentional, is also not uncommon, especially in rural areas. Acute toxicity manifests as a cholinergic crisis, and the death is usually due to cardiovascular and respiratory failure.* ***Objectives:*** *This study was conducted to analyze the clinical profile of OP poisoning patients in a teaching hospital, where the majority of the clientele is from rural background.* ***Methods:*** *80 patients, above 16 years of age, admitted with OP poisoning were included in the study. The diagnosis was based on a history of exposure and characteristic clinical features and was supported by serum Pseudocholinesterase (PChE) levels. The clinical features, management, complications, and outcome were analyzed.* ***Results:*** *Deliberate self-harm was the leading cause of exposure. The mean age of patients was 32.3 years. The male-to-female ratio was 1.4:1. Four patients developed the intermediate syndrome. The average dose of atropine required was 26.84 mg and pralidoxime 24 gm. The serum cholinesterase level was <10% in 5, 10%-20% in 7, 20%-50% in 16, and >50% in 52 cases. The mean ICU stay was 4 days. The mortality rate in our study was 3.75%.* ***Conclusions:*** *The mortality was directly proportional to the amount of OP consumed, clinical severity, PChE levels, and delay in seeking medical attention. This study highlights the importance of rapid diagnosis and initiation of early treatment.*

Keywords: Organophosphate, Muscarinic, Nicotinic, Acetylcholinesterase, Atropine, Pralidoxime

INTRODUCTION

Poisoning is a global health challenge. Organophosphates and Carbamates, or 'organophosphorus compounds' (OP) are responsible for the poisoning in the majority of cases, especially in agrarian countries like India [1]. About 385 million cases of unintentional exposure to pesticides occur every year among farmworkers (about 44% of the estimated 860 million agricultural population worldwide) leading to 11,000 deaths [2]. In India, mortality rate attributed to unintentional poisoning was 0.26 (0.16-0.36), males 0.34 (0.2-0.46), females 0.17 (0.12-0.24) per 100,000 population in 2019 [3]. OPs are commonly employed in farming as a pesticide. The commonly used OPs in India are Chlorpyriphos, Dimethoate, Dichlorvos, Parathion, Malathion, Monocrotophos, and Glyphosate [4,5]. These agents are readily available over the counter. Their easy availability makes them an instrument of choice for deliberate self-harm. An estimated 168,000 pesticide self-poisoning deaths take place annually in India, that is, 19.7% of global suicides [6]. OP compounds are easily absorbed through the skin and mucous membranes. Absorption through gastric mucosa is the most common mode of poisoning. OPs are metabolized by Cytochrome P450s in the liver, either by deacylation to create an inactive metabolite, or through a desulfuration to produce an active oxon metabolite, and are essentially eliminated by the kidneys [7]. The OPs inhibit cholinesterase Acetylcholinesterase (AChE) and Butyrylcholinesterase (BChE) or Pseudocholinesterase or Plasma Cholinesterase (PChE). AChE Hydrolyses Ach and is primarily found at postsynaptic neuromuscular junctions, especially in muscles and nerves, while PChE

is mainly found in plasma is to the tune of 1000:1 [8]. The inhibition of AChE causes acetyl ACh accumulation at all nerve endings [9]. The excess ACh results in various signs and symptoms within minutes to hours; the common symptoms are Salivation, Lacrimation, Urination, Defecation, Gastric cramps, and Emesis (SLUDGE) [10,11]. OP poisoning (OPP) can manifest in three phases: acute cholinergic crisis, intermediate syndrome, and delayed neuropathy [11,12]. The OP-induced inhibition also causes low circulating PChE levels and is used for the diagnosis of OPP.

Atropine is an established specific antidote for the muscarinic effects of OPP. Atropine is a central and peripheral muscarinic receptor antagonist. However, atropine does not bind to nicotinic receptors and is ineffective in treating neuromuscular dysfunction. Pralidoxime (2-PAM) and other oximes, such as HI-6 and obidoxime, are cholinesterase reactivating agents that are effective in treating both muscarinic and nicotinic symptoms. Oximes may cause transient worsening of symptoms due to oxime-induced AChE inhibition and should not be administered without concurrent atropine. WHO recommends the use of pralidoxime (>30 mg/kg bolus followed by >8 mg/kg/hr infusion) along with atropine in the management of OPP [13].

AIM AND OBJECTIVE

This study was conducted to analyze the clinical profile and outcome of OP poisoning patients in a teaching hospital, where the majority of the clientele is from rural background.

METHODS

The study included 80 patients, admitted to medical ICU from July 2020 till July 2021.

Inclusion Criteria

All patients above 18 years of age, reporting a confirmed history of OP exposure/ingestion were included in the study.

Exclusion Criteria

The patients where the poison was not positively identified, had some pre-existing comorbidity, and those who received initial treatment from some other health care facility were excluded from the study.

The history of exposure, symptomatology, physical signs, and PCE levels were tabulated and analyzed. The diagnosis in each case was based on the history of exposure and clinical features and was confirmed by low serum PCE levels. The severity of poisoning was defined based on the POP scale and serum ChE level (latent poisoning serum ChE <50% of normal laboratory values, mild 20% to 50%, moderate 10% to 20%, and Severe <10% of normal laboratory value) [13]. Psychiatric consultation of each patient was carried out before discharge from the hospital (Table 1).

Table 1 Peradeniya Organophosphorus Poisoning (POP) scale

Parameters	Value	Points
Pupil size	>2 mm	0
	<2 mm	1
	Pinpoint	2
Respiratory rate	>20	0
	<20	1
	<20 with central cyanosis	2
Heart rate	>60	0
	41-60	1
	<40	2
Fasciculation	Absent	0
	Present-generalized/continuous	1

	Generalized and continuous	2
Level of consciousness	Conscious and coherent	0
	Impaired	1
	No response to verbal commands	2
Seizures	Absent	0
	Present	1

RESULTS

In the current study, data of 80 patients (58.75% male, 41.25% female) admitted with OPP were analyzed out of a total of 151 cases of poisoning. All patients had consumed the compound with the motive of self-harm. The OP consumed was identified in most cases from the used bottles. 22 of 80 (27.5%) patients used chlorpyriphos, and 19 (23.75%) used dimethoate for self-harm (Table 2).

Table 2 Type of OP consumed

S. No.	Name of OP	Number	Percentage
1	Chlorpyriphos	22	27.5
2	Dimethoate	19	23.75
3	Profenophos	9	11.25
4	Dichlorvos	8	10
5	Glyphosate	7	8.75
6	Monocrotophos	6	7.5
7	Dicrotophos	3	3.75
8	Azamethiphos	3	3.75
9	Temephos	2	2.5
10	Ethoprophos	1	1.25

Most of the patients were young, 40% from the age group of 16 years to 25 years, 32.5% from 26 years to 35 years, 12.5% from 36 years to 45 years, and 7.5% patients from the age group of 46 years to 55 years (Figure 1). The average time of presentation was 5.16 hours after consumption of OP (Figure 2).

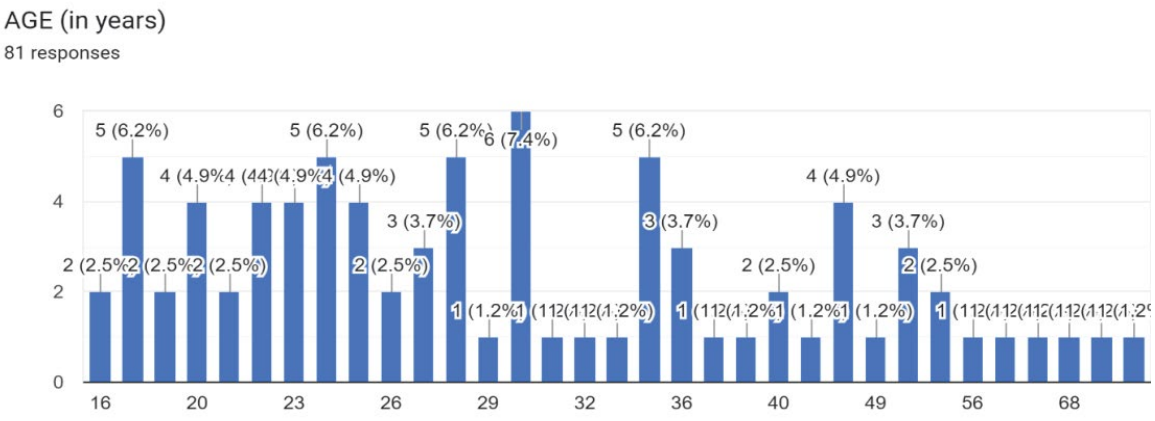

Figure 1 Age distribution of the patients

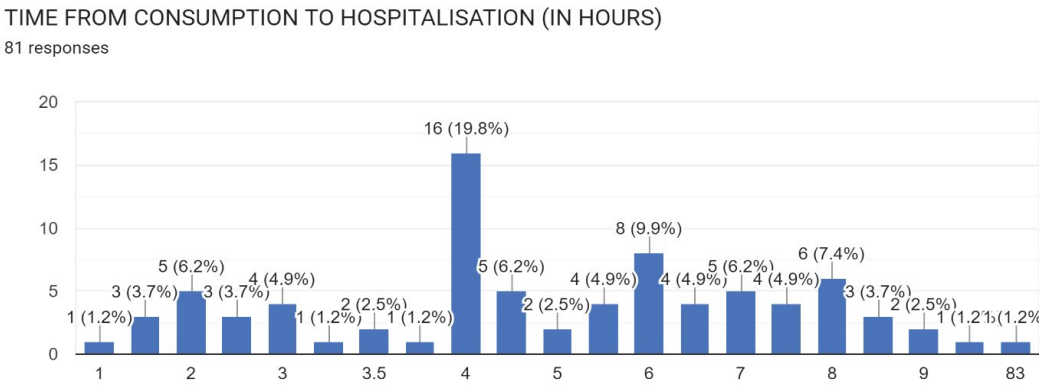

Figure 2 Interval from OP consumption to hospitalization

The most common signs and symptoms were salivation (38.3%), lacrimation (35.8%), and diaphoresis (16%) apart from nausea and vomiting that was present in all the patients. The other features in decreasing order occurrence were weakness, diarrhea, sweating, bradycardia, hypotension, bronchospasm, abdominal pain, miosis, fasciculations, cramps, and paralysis respectively (Figure 3 and Figure 4). Serum cholinesterase was <10% in 5, 10% to 20% in 7, 20% to 50% in 16, and >50% in 52 cases.

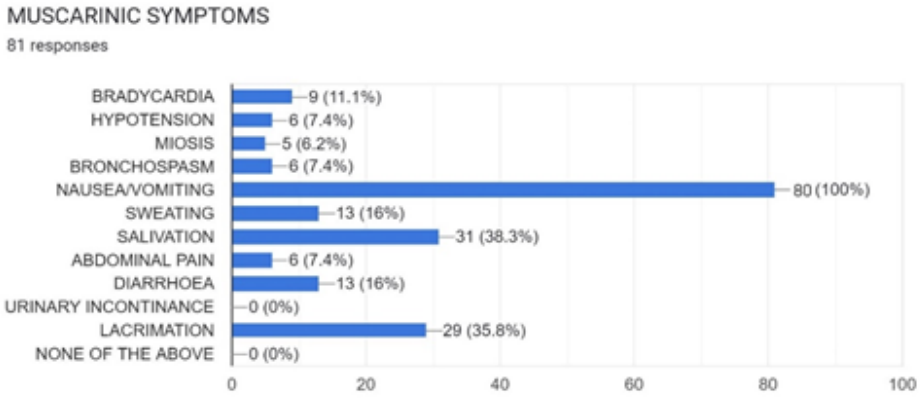

Figure 3 Frequency of muscarinic features

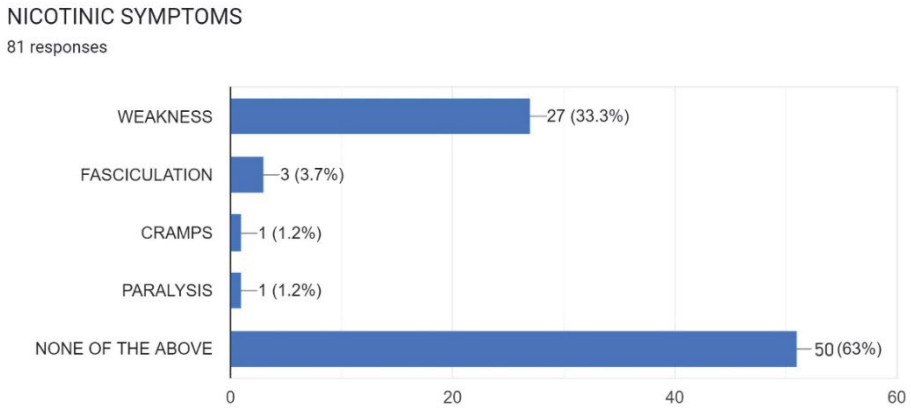

Figure 4 Frequency of nicotinic features

consumed, clinical severity, PChE levels, and delay in seeking medical attention. This study highlights the importance of rapid diagnosis and initiation of early treatment.

DECLARATIONS

Conflict of Interest

The author's declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

REFERENCES

[1] Narang, Udit, Purvasha Narang, and OmPrakash Gupta. "Organophosphorus poisoning: A social calamity." *Journal of Mahatma Gandhi Institute of Medical Sciences*, Vol. 20, No. 1, 2015, p. 46.

[2] Boedeker, Wolfgang, et al. "The global distribution of acute unintentional pesticide poisoning: estimations based on a systematic review." *BMC public health*, Vol. 20, No. 1, 2020, pp. 1-19.

[3] Nicholls, Ella, et al. "Staying at home: the potential cost savings related to triage advice provided by the New Zealand National Poisons Centre." *Clinical toxicology*, Vol. 60, No. 1, 2022, pp. 115-21.

[4] Gupta, S. K., et al. "An epidemiological pattern of poisoning in India." *Pharmacoepidemiology and drug safety*, Vol. 11, No. 1, 2002, pp. 73-74.

[5] Banday TH., et al. "Predictors of Morbidity and Mortality in Organophosphorus Poisoning: A Case Study in Rural Hospital in Karnataka, India". *Research Gate*, 2015; Vol. 7, No. 6, pp. 259-65.

[6] Mew, Emma J., et al. "The global burden of fatal self-poisoning with pesticides 2006-15: systematic review." *Journal of affective disorders*, Vol. 219, 2017, pp. 93-104.

[7] Ellison, Corie A., et al. "Human hepatic cytochrome P450-specific metabolism of the organophosphorus pesticides methyl parathion and diazinon." *Drug Metabolism and Disposition*, Vol. 40, No. 1, 2012, pp. 1-5.

[8] Tunsaringkarn, Tanasorn, Kalaya Zapuang, and Anusorn Rungsiyothin. "The correlative study of serum pseudo-cholinesterase, biological parameters and symptoms among occupational workers." *Indian Journal of Clinical Biochemistry*, Vol. 28, No. 4, 2013, pp. 396-02.

[9] Weinbroum, Avi A. "Pathophysiological and clinical aspects of combat anticholinesterase poisoning." *British Medical Bulletin*, Vol. 72, No. 1, 2004, pp. 119-33.

[10] Sert, Ali İhsan, et al. "Retrospective analysis of organophosphate poisonings in an intensive care unit in Turkey: a single-center study." *Dubai Medical Journal*, Vol. 1, 2018, pp. 1-4.

[11] Kumar, Subhas V., et al. "Current review on organophosphorus poisoning." *Archives of applied science research*, Vol. 2, No. 4, 2010, pp. 199-15.

[12] Kumar, Subash V., et al. "A study on poisoning cases in a tertiary care hospital." *Journal of natural science, biology, and medicine*, Vol. 1, No. 1, 2010, p. 35.

[13] Gupta, Rishab, and Mayur Parmar. "Pralidoxime." 2020.

[14] Eyer, Peter. "The role of oximes in the management of organophosphorus pesticide poisoning." *Toxicological reviews*, Vol. 22, No. 3, 2003, pp. 165-90.

[15] Senanayake, Nimal, H. J. De Silva, and Lakshman Karalliedde. "A scale to assess severity in organophosphorus intoxication: POP scale." *Human and experimental toxicology*, Vol. 12, No. 4, 1993, pp. 297-99.

[16] Michael E. et al., " Management of acute organophosphorus pesticide poisoning". *PMC Pubmed Central*, 2014.

[17] Umakanth, Maheswaran. "Intermediate Syndrome Following Organophosphate Poisoning; Review Article." *Asia Pacific Journal of Medical Toxicology*, Vol. 8, No. 1, 2019, pp. 19-24.

[18] Banerjee, Indranil, S. K. Tripathi, and A. Sinha Roy. "Clinico-epidemiological characteristics of patients presenting with organophosphorus poisoning." *North American journal of medical sciences*, Vol. 4, No. 3, 2012, p. 147.

[19] Panchal, Manisha, and Dipti Trivedi. "Clinical profile in patients of organophosphorus poisoning." *International Journal of Science and Researchm* Vol. 5, 2016, pp. 97-99.

[20] Reddy, B. Shrikar, et al. "Factors associated with outcomes in organophosphate and carbamate poisoning: a retrospective study." *Toxicological research*, Vol. 36, No. 3, 2020, pp. 257-66.

[21] Prasad, Devanur RMM, et al. "Relevance of plasma cholinesterase to clinical findings in acute organophosphorous poisoning." *Asia Pacific Journal of Medical Toxicology*, Vol. 2, No. 1, 2013, pp. 23-27.

[22] Hiremath, Pradeepkumar, et al. "Pseudocholinesterase as a predictor of mortality and morbidity in organophosphorus poisoning." *Indian Journal of Critical Care Medicine*, Vol. 20, No. 10, 2016, p. 601.

[23] Giyanwani, Pirthvi Raj, et al. "Respiratory failure following organophosphate poisoning: a literature review.", *Cureus*, Vol. 9, No. 9, 2017.

[24] DeBleecker, Jan L. "The intermediate syndrome in organophosphate poisoning: an overview of experimental and clinical observations." *Journal of Toxicology: Clinical Toxicology*, Vol. 33, No. 6, 1995, pp. 683-86.

[25] Syed, Sumaya, et al. "Is the World Health Organization-recommended dose of pralidoxime effective in the treatment of organophosphorus poisoning? A randomized, double-blinded and placebo-controlled trial." *Saudi journal of anaesthesia*, Vol. 9, No. 1, 2015, p. 49.

[26] Youssef, Carl, et al. "Compressive cervical myelopathy in patients with demyelinating disease of the central nervous system: improvement after surgery despite a late diagnosis." *Cureus*, Vol. 13, No. 2, 2021.

[27] Eddleston, Michael, et al. "Oximes in acute organophosphorus pesticide poisoning: a systematic review of clinical trials." *Qjm*, Vol. 95, No. 5, 2002, pp. 275-83.