

ORIGINAL RESEARCH

Clinical Profile of Organophosphorus Poisoning

Abhishek Singhai¹, Sagar Dembla²**ABSTRACT**

Introduction: Organophosphorus poisoning can occur after dermal, respiratory or oral exposure to pesticides (e.g. chlorpyrifos, dimethoate, sarin, tabun). The aim of this study was to characterize organophosphorus poisoning, inclusive of exposure route, clinical symptoms, therapy given and consecutive outcomes.

Methods and Material: This is a retrospective study. Medical records of all patients presenting to Emergency Department (ED) of a tertiary care centre with OP poisoning from January 2011 to December 2014 were reviewed.

Results: The most eminent route of exposure was ingestion in 40 patients (67%), followed by inhalation in 16 patients (27%). Majority of exposures: Forty nine cases (82%) were intentional; eleven cases (18%) were accidental. The most common clinical presentation in 43 patients (72%) was vomiting. Ten patients (17%), underwent intubation in Emergency Department because of respiratory failure. Mean length of hospital stay was 6.7 days. Two patients died. Predominantly patients were discharged in absence of any neurological deficit in Fifty-eight patients (97%).

Conclusion: Management of agrochemicals poisoning (like organophosphates) is a challenge, peculiarly in a resource limited circumstance. These are widely being used in developing countries, where most cases are intentional ingestion. An uptight law making for trading, marketing and stocking of these chemicals is crucial. These measures will conceivably reduce the incidence of poisoning.

Keywords: Organophosphorus poisoning, clinical characteristics, intentional

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INTRODUCTION

Organophosphates are chemicals which were originally synthesized by the reaction of alcohols with phosphoric acid. In early 1930, organophosphates were commercially used as insecticides, whereas these chemical molecules were aggrandized as neurotoxins in World War II by German military. They principally affect neuromuscular transmission by inhibiting Acetyl Cholinesterase.

Organophosphate insecticides, such as diazinon, chlorpyrifos, disulfoton, azinphos-methyl, and fonfos, are widely in agriculture and household applications as pesticides. Organophosphorus poisoning can occur after dermal, respiratory or oral exposure to pesticides (e.g. chlorpyrifos, dimethoate, sarin, tabun). These agents lead to a major health care burden by suicidal ingestion in various parts of the world^{1,2} nearly causing about 300,000 death every year.^{3,4} Despite the superlative number of deaths arise in agronomic rural areas of developing countries,² pesticide poisoning is a predicament in industrialized countries, where suicidal ingestion is anecdotic for a substantial proportion of deaths.⁵

Pesticide exposure is seen before spraying because of effortless availability mixing and inadequate labelling. Thus making these compounds as a cheap and readily available poison in market.⁶

Clinical symptoms and signs are versatile on the nature of the compound, amount ingested and duration of presentation to hospital. The typical treatment comprises supportive measures, administration of antimuscarinic agents and acetylcholinesterase reactivator.

Thus, the aim of our study was to delineate organophosphorus poisoning based on the route of exposure, clinical characteristics, management and later outcomes.

MATERIAL AND METHODS

This is a retrospective study spanning over 4 years in Sri Aurobindo Medical College & Postgraduate Institute,

a tertiary care hospital in Indore. The Sri Aurobindo Medical College & Postgraduate Institute is a 1000-bed teaching hospital. Medical records of all patients presenting to Emergency Department (ED) of a tertiary care centre with OP poisoning from January 2011 to December 2014 were thoroughly reviewed. The subsequent variables were abstracted from files inclusive of age, sex, occupation, agent consumed, route of poisoning, clinical sign & symptoms, laboratory findings, site of care and final outcome. All patients of either sex presented with organophosphorus poisoning were included in the study. Patients, who have consumed other poison or mixed poison, were excluded from study. The data from all case sheets were entered into a database (MS Access) to calculate descriptive statistics on 20 presentations. Statistical analyses were performed using SPSS Statistics 17.0 (SPSS Inc., Chicago, USA).

After resuscitation in a casualty ward, patients were admitted to the intensive care unit, a 16 beds well equipped unit and is monitored by specialist doctors and nurses. Patients were treated on standard protocol. The clothing were removed immediately and discarded. All traces of residue were removed by careful washing with alkaline soap or bleach solution. If patient came after oral ingestion, than he received gastric lavage and activated charcoal. Initial management consists of intravenous fluid and oxygen inhalation. Further, patients were treated with atropine and pralidoxime. Patients suffering from respiratory failure were immediately intubated and put on ventilator.

STATISTICAL ANALYSIS

Discriptive statistics was used to calculate the results with the help of SPSS version 21.

RESULTS

Patient characteristics are shown in Table 1. A total of 60 patients (44 males [73%] and 16 females [27%]), around 18% patients were below 18 years (i.e. 11/60), remaining 82% were above 18 years (i.e. 49/60 years). Most prevailing route of exposure being ingestion (accidental & intentional) seen in 67% (40/60 patients) followed by inhalation seen in 27% (16/60) patients. There were two cases of dermal exposure and in one case the route of exposure was unknown. The majority of exposures: Forty nine cases (82%) were intentional; eleven cases (18%) were accidental. The most common clinical presentation in 43 patients (72%) was vomiting; 21 cases (35%) had excessive salivation, 2 (3%) cases had cardiac arrest and 42 patients (70%)

Patient Characteristics	
Age in years (Mean \pm SD)	30 \pm 2.4
Sex (%)	73% Male 27% Female
Route of Exposure	Ingestion 67% Inhalation 27%
Type of Poisoning	Intentional 82% Accidental 18%
Clinical features	Vomiting 72% Excessive salivation 35% Non-specific 70%
Ventilator required	17%
Final outcome	Survived 97% Mortality 3%
Table-1: Patient Characteristics	

presented with clinical features non-specifically relating to cholinergic crisis. Ten patients (17%) were given respiratory support in ED for respiratory failure. The mean length of stay in hospital was 6.7 days. Two patients died. Fifty-eight patients (97%) were discharged home without any neurological deficits.

DISCUSSION

In early 19th century organophosphate was first synthesized by reaction between alcohol and phosphoric acid. Physostigmine was one of the cardinal agent used to treat glaucoma in 1870s. By the year 1930, these were used as synthetic cholinesterase inhibitors for autonomic and skeletal muscle disorder. Some of these agents were tried in the therapy of Parkinson's disease. In year 1986, tacrine experimentation were instituted, to be the first anti-cholinesterase to be tried for Alzheimer's, hence was approved for clinical use in 1993. But was discontinued in US in 2013 due to safety concerns. The major concern in the development of these agents was blood-brain barrier penetration for being used in dementia. Drugs like rivastigmine are widely being used; they primarily act as parasympathomimetic or cholinergic agents by irreversible inhibition of acetylcholinesterase (AChE) on both target and nontarget sites. They are administered either orally or by transdermal patch. Reported adverse effects like gastro-intestinal disturbance, significant weight loss because of the increased cholinergic activity.

Acetylcholinesterase is seen in red blood cells; Nicotinic and muscarinic receptors are found in numerous tissue like nerves, muscle, and gray matter of the brain. Pseudocholinesterase, also known as plasma acetylcholinesterase is principally synthesized in liver. Is pri-

marily seen in plasma and modest amount is present in CNS white matter, pancreas, and heart. It is a hepatic protein that is decreased by significant proportions in liver dysfunction, malnutrition, pregnancy, infectious conditions, neoplastic disease and narcotic abuse. A decrease in plasma cholinesterase levels lead to a net decrease in cholinesterase activity in both central and peripheral nervous systems.

The serine hydroxyl group of organophosphates are the imperative site of acetylcholine. Thus binding irreversibly, causing decommission of the esterase, leading to accumulation of acetylcholine at neuromuscular end-plate.

The aggregation of acetylcholine at these neuromuscular junctions leads to unrelenting depolarization of skeletal muscle, thus leading to fasciculation's and weakness of the certain group. The neurotransmitter in central nervous system are disrupted. Thus, strong nucleophile such as pralidoxime (2-PAM) within 24 hours can reverse the interaction, leading to acetylcholinesterase damage. The rise in RBC cholinesterase levels is very sluggish; around 0.5-1% daily.

Few organophosphate esters produce a certain type of Delayed neurotoxicity known as axonopathic. Numerous of these agents marketed in developing countries have been associated with delayed onset of neuropathy resulting from cumulative doses or enormous amount of single dose. Organophosphorus ester-induced delayed neuropathy (OPIDN) takes at least 10 days to develop following a single acute exposure.

Organophosphates poisoning is a prevailing health trouble in India. This finding is consistent with various other studies, in both accidental and occupational poisonings.⁷ This upsurge in numbers is not accounted by occupational hazard in agriculture industry, but is reasonably a reflection of facile availability of OP compounds to the public and their pullulating popularity as suicidal agents. Intentional poisoning with these agents was reported to be around 10-36.2% in developed countries, 40-60% in African countries and as high as 65-79.2% in developing countries.⁸ The study revealed that the 20-30 age group was significantly more prone to suicide than other age groups.

Linden and Burns⁹ reported that the main route of exposure to OP compound was ingestion (74%). In our study, 40 patients (67%) had ingested the OP. Fifteen patients required intubation in the ED because of respiratory failure, which is the most common complication encountered in the literature.¹⁰ The mean stay in hospital recorded in the report of the Emerson et al.¹¹ study of 69 patients between 1987 and 1996 in Western

Australia was 7 days (1-25 days).¹² In our study, the mean stay in hospital was 6.7 days (1-25 days), which is in keeping with other reports.¹³

In the present study, majority of the patients belonged to the young age group. Males show a clear preponderance over female. This finding is in discordance with studies conducted in Turkey (M: F=1:1.47),¹⁴ Nepal (M: F=1:2).¹⁵ However, in the study conducted in Chennai by Shivaprasad et al., (2001),¹⁶ male patients (74%) outnumbered female (26%).

In the present study, poisoning with suicidal intent was more common (82%) than the accidental one (P-value< 0.0001). This is in congruence with studies conducted in Nepal,¹⁵ Turkey,¹⁴ Gulbarga¹⁷ where poisoning with suicidal intent accounts for 95.24%, 75.9%, and 97.25% of total cases of OP poisoning respectively. The mean interval between poison consumption and admission to the hospital was 4.4 hours. In studies conducted at Chennai,¹⁶ maximum patients (89.69%) presented within 6 hours.

CONCLUSION

As Agrochemicals like organophosphates are widely used in rural areas of developing countries like INDIA. Straightforward and unaccounted availability of these highly hazardous compounds is the foremost reason for such high incidence. An uptight law making for trading, marketing and stocking of these chemicals is crucial in reducing mortality and perhaps will conceivably reduce the incidence of poisoning in developing countries. In the long term, endorsing an alternative to agrochemicals might be the utmost essential strategy of preventing OP poisoning.

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