Clinical profile of organophosphate poisoning at a tertiary-care center

Nehal M Shah, Shashikumar H Mundhra

Department of Medicine, GMERS Medical College, Gandhinagar, Gujarat, India. Correspondence to: Nehal M Shah,E-mail: dr.nehalshah@yahoo.co.in

Received October 15, 2015. Accepted November 30, 2015

Abstract

Background: Nowadays, organophosphate (OP) compounds are widely used in agricultural field as an insecticide. Toxicity with these compounds is owing to inhibition of acetylcholinesterase enzyme. Patients are presented with muscarinic and nicotinic side effects. Red blood cells and plasma acetylcholinesterase enzyme (AChE) levels are helpful in diagnosis of the patients. It may cause life-threatening acute and chronic complications. All poisoned patients are required intensive care and treatment with injections atropine and PAM.

Objective: To study the clinical aspect of OP poisoning in detail with hospital stay, clinical course, complication, and recovery and mortality in OP poisoning.

Materials and Methods: This study was done at PDU Medical College, Rajkot, Gujarat, India, comprising 50 cases of OP poisoning. After taking written consent, personal history of the patients was taken. Then, a detailed history regarding poison and clinical symptoms was taken. Then, general and systematic examinations of patients were carried out, and blood samples were sent for investigations. We followed up the patients till discharge or expired.

Result: In our study, maximum incidence of OP poisoning was in between 20 and 40 years age group (60%), and male to female ratio was 2:1. Clinical signs such as bradycardia and tachycardia were present in 20% cases. Miosis was present in 70% cases, and it is more dangerous. Low serum AchE level was found in 68% cases, with mortality in 44.62% among them. Type-I paralysis (52%) and acute respiratory failure (32%) were found as life-threatening complications. In our study, 64% patients survived.

Conclusion: Detailed history and thorough clinical examination of patients are helpful in diagnosing the patients of OP poisoning. Life-threatening complications occurred in these patients. Early detection and immediate treatment in intensive care units with injections atropine and PAM can increase the chances of survival rate of patients.

KEY WORDS: Clinical profile, OP poisoning, management

Introduction

As we are entering in new millennium, production and usage of chemicals, drugs, and insecticides are increasing day-by-day. India is too developing fast and not left behind

Access this article online						
Website: http://www.ijmsph.com	Quick Response Code:					
DOI: 10.5455/ijmsph.2016.15102015267						

much in industry and agriculture. Since the last four decades, organophosphorous compounds have become one of the most commonly used insecticides.^[1] More insecticides have already attained new heights in the already existing common problems.

Thousands of tons of acetylcholinesterase enzymes (AchEs)-inhibiting carbamates and organophosphate (OP) pesticides are used throughout the world for agricultural application as insecticides. The toxicity is attributed to their ability to inhibit AchE that inhibits the activity of neurotransmitter agent acetylcholine (Ach).^[1] AChE activity can be measured in the serum and red blood cells.

In India, OP compounds are easily accessible; therefore, it is the most common mode of poisoning fatalities as a source of both intentional and unintentional poisoning.^[2]

International Journal of Medical Science and Public Health Online 2016. © 2016 Nehal M Shah. This is an Open Access article distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/), allowing third parties to copy and redistribute the material in any medium or format and to remix, transform, and build upon the material for any purpose, even commercially, provided the original work is properly cited and states its license.

Shah and Mundhra: Clinical profile of organophosphate poisoning

ding upon severity of compounds. Muscarinic symptoms such as nausea, vomiting, diarrhea, sweating, salivation, urination, stool incontinence, lacrimation, miosis, and bradycardia and nicotinic signs such as mascular weakness, fasciculation, paralysis, convulsion, and coma are found.^[3]

OP compounds lead to acute and chronic complications. Acute complications include acute respiratory failure, acute respiratory distress syndrome (ARDS), types I and II paralysis, intermediate syndrome (IMS), sudden cardiac death, aspiration pneumonitis, and resecretions. Chronic complications include anxiety, depression, polyneuropathy, paralysis, and coma.^[4]

Poisoning with these compounds is very serious and requires treatment in intensive care unit as they present with life-threatening complications and may result in mortality. It also affects respiration, which may endanger the individual's life.

Mortality rates depend upon amount and type of compound, condition of patient on arrival at hospital, delay in diagnosis and treatment, and respiratory management. There was always correlation with type of compounds, prehospitalization period, and the type of management, and they are useful for preventing the mortality rate in developing countries such as India.^[5]

Treatment includes early resuscitation with oxygen, airway protection, intravenous fluids, muscarinic antagonist such as atropine, and acetyl cholinesterase activator such as PAM.^[4] Gastric lavage could have a role but should only be undertaken once the patient is stable. Patients must be carefully observed after stabilisation for changes in atropine needs, worsening respiratory function because of IMS, and recurrent cholinergic features occurring with fat-soluble organophosphorus compounds.^[6]

So, in a light of increased use of OP compounds and its various applications, it is worth to study in detail the various clinical and biochemical aspects of OP poisoning.

Objective

To study the clinical aspect of OP poisoning in detail with hospital stay, clinical course, complication, and recovery, mortality in OP poisoning, to know various biochemical alterations in OP poisoning, and to assess clinical recovery in correlation with biochemical changes in level of serum cholinesterase.

Materials and Methods

Selection Criteria

This study comprised 50 cases of acute OP compound poisoning, which were admitted in Pandit Din Dayal Upadhyay Medical College and Hospital, Rajkot, Gujarat, India. We included all patients of OP poisoning, those who came with history and clinical features of OP poisoning, irrespective of their vital status during our study period.

After explaining the procedure to patients in their vernacular language and obtaining consent from them, this study comprised detailed history, clinical examination, and laboratory investigation of each of the cases including serum cholinesterase on admission and at the time of discharge. The diagnosis of OP compound was based on definite history of OP poisoning, examination of container, typical clinical features, clinical examination findings, characteristic color of stomach wash or vomits, atropine tolerance, and decrease in cholinesterase activity in plasma or blood.

Method of Study

Details regarding age, sex, name, address, marital status, occupation, socioeconomic class, psychological problems, family history, major illness, etc. were taken into consideration.

Detailed history regarding poison such as name of poison, quantity, route, intention, mean time delay, type, and site was noted in each case.

General and systemic examinations with detailed clinical examination of respiratory system, cardiovascular system, alimentary system, and central nervous system were done in all cases.

Laboratory investigations including complete blood count, renal function test, serum cholinesterase levels on admission/ on discharge, electrolytes, electrocardiogram, chest X-Ray (PA) were carried out in each case.

Result

Fifty cases of only OP compound poisoning were studied in detail during the period between January 1, 2004 and June 30, 2005 at PDU Medical College and Hospital, Rajkot. The following were the observations:

Table 1: Combined age- and sex-wise distribution	ution
--	-------

	-		
Age (years)	Male	Female	Total, N (%)
13–20	2	10	12 (24)
21–30	18	4	22 (44)
31–40	6	2	8 (16)
41–50	5	-	5 (10)
≥51	3	-	3 (06)

Table 2: Symptomatology-wise distribution

Table 2. Cymptomatology wise distribution					
Symptoms	No. of cases (%)				
Muscarinic					
Nausea	50 (100)				
Vomiting	50 (100)				
Perspiration	35 (70)				
Stool/urine incontinence	35 (70)				
Salivation	30 (60)				
Altered sensorium	20 (40)				
Convulsion	-				
Respiratory difficulty	18 (36)				
Nicotinic					
Twitching/flickering	33 (66)				
H/o OP poisoning	42 (84)				

Table 3: Clinical signs-wise distribution

Pulse	No. of cases (%)	Mortality (%)
Bradycardia (<60/min)	10 (20)	03 (30)
Tachycardia (>100/min)	10 (20)	1 (10)
Norma (60–100/min)	30 (60)	11 (36.64)
Miosis-present	35 (70)	12 (34.29)
Miosis-absent	15 (30)	4 (26.67)

 Table 4: Grading of severity of OP poisoning in relation to mortality, according to serum cholinesterase level

Grading depends on AchE level on admission	No. of cases (%)	Mortality (%)
Mild (20%–50%) (>800 U/L)	7 (14)	1 (14.28)
Moderate (10%–20%) (400–800 U/L)	9 (18)	2 (22.22)
Severe (<10%) (<400 U/L)	34 (68)	15 (44.62)

Table 5: Complication-wise distribution in relation to mortality

Complication	No. of cases (%)	Mortality (%)
Type I paralysis(acute cholinergic crisis)	26 (52)	3 (11.54)
Acute respiratory failure	16 (32)	10 (62.5)
Aspiration pneumonitis	12 (24)	4 (33.33)
Sudden cardiac arrest	8 (16)	6 (75)
ARDS	6 (12)	6 (100)
Type II paralysis (IMS)	5 (10)	4 (80)
Resecretion	2 (4)	1 (50)
GBS	-	_

	Table 6: 0	Outcome	and	hospital	stav-wise	distribution
--	------------	---------	-----	----------	-----------	--------------

Outcome	Total	Duration of hospital stay (in days)					
		First	Second	Third	Fourth	≥ Fifth	
Survived (discharge)	32	-	_	2 (6.25)	2 (6.25)	28 (87.5)	
Expired	16	10 (62.5)	3 (18.75)	2 (12.5)	1 (6.25)	_	
Absconded or DAMA	2	-	_	2 (100)	-	-	

Maximum incidence of OP poisoning was in between 20 and 40 years age group (60%). The youngest age was 13 years, and the oldest was 60 years. Male to female ratio was 2:1. Below 20 years of age, 20% cases were female subjects and 4% were male subjects, and older than 20 years, 84% cases were male subjects when compared with 12% female subjects [Table 1].

We observed that muscarinic symptoms were more common than nicotinic symptoms. Among muscarinic symptoms, nausea and vomiting were present in all cases. Other symptoms in decreasing order were perspiration and incontinence of stool and urine (70%), salivation (60%), altered sensorium (40%), and respiratory difficulty (36%). We found history of OP poison in 84% cases, while rest of cases were diagnosed by clinical examination and laboratory results [Table 2].

We found that bradycardia and tachycardia each was present in 20% cases, while other 60% cases showed normal pulse rate. Mortality was higher in normal pulse rate (36.64%), when compared with bradycardia (30%) and tachycardia (10%). Miosis was present in 70% cases with mortality in 34.29% cases among them, while miosis was absent in 30% cases [Table 3].

We divided all patients according to AchE level into three categories. They were considered mild when > 800 IU/L or 20%–50% AchE was found in serum, which was found in 14% cases; among them, mortality was 14.28%. They were considered moderate when AchE was either 400–800 IU/L or 10%–20% in serum, as seen in 18% cases; about 22.22% patients expired among moderate group. Severe category included <400 IU/L or <10% level, which was found in 68% cases, and 44.62% cases expired among severe group [Table 4].

In our study, we found that various types of complications such as type-I paralysis (52%), followed by acute respiratory failure (32%), aspiration pneumonitis (24%), sudden cardiac death (16%), ARDS (12%), and type –II paralysis (IMS, 10%). We observed that mortality was higher in ARDS (100%)

patients, followed by type-II paralysis (80%), sudden cardiac death (75%), and acute respiratory failure (62.5%) [Table 5].

We found that 64% patients survived, 32% expired, and 4% were discharged against medical advice. Higher incidence of death was observed on the first day (62.5%). About 87.5% survived patients were staying for more than 5 days in hospital [Table 6].

Discussion

A total of 50 cases of acute OP poisoning was studied in this study from January 1, 2004 to June 30, 2005 at PDU Medical College and Hospital, Rajkot, Gujarat, India.

Maximum incidence of OP poisoning was in between 20 and 40 years age group, which is comparable to the study by Emerson et al.,^[7] which shows 95% cases in 30–50 years age group and the lowest incidence in elderly persons. The same finding was also observed in the study by Kora et al.^[8] We see that maximum incidence of OP poisoning are found in female subjects in younger age group when compared with male subjects. In our study, male subjects were involved more than female subjects. Whereas the finding by Banerjee et al.^[9] showed that female subjects are more commonly involved.

This study shows that muscarinic effects predominates and appears first and then nicotinic effects. Study by Emerson et al.^[7] shows that muscarinic symptoms were found in 92% cases, while this study showed in 100% cases. This study is also correlated with the findings by Mishra et al.^[10] that nausea and vomiting is present in 88% cases. Miosis is a good clinical sign to diagnose OP poisoning, which is also comparable with CHA study that showed 64%. We found that miosis was found in 70% cases in our study and in 91% cases in the study done by Banerjee et al.;^[9] so, it is also comparable with our study.

Nicotinic symptoms appear late, and that indicates progression of disease process. Fasciculation is a bad prognostic sign, and, in our study, it was found in 60% cases which was comparable to the study by Chugh et al.,^[11] which showed in 40% cases. Mortality is three times higher among patients who had fasciculation at the time of admission. History of OP poisoning is a major conformational data in OP poisoning, which helps in further management of OP poisoning.

Mortality was high among patients who showed normal heart rate rather than who showed bradycardia or tachycardia at time of admission.

It is concluded that maximum mortality was noted in those patients who had low serum AChE level on admission (e.g., <400 IU/mL), which is concurrent with the study by Goswamy et al.^[12] It is also concluded that high atropine tolerance and early administration of high doses of PAM will increase the chances of survival in OP poisoning, which is also supported by the findings of Gohel et al.^[13] and de Silva et al.^[14] Early stoppage of atropine is hazardous in course of OP poisoning. So, it is better to continue atropine till patient is out of danger. Adequate atropinization carried less mortality (16%) when compared with under atropinization (66%) and over atropinization (37%). Acute life-threatening conditions in OP poisoning are ARDS, acute respiratory failure, sudden cardiac death, resecretions, and type II paralysis (IMS). This study is also comparable with the study by Faiz et al.,^[15] which showed that morbidity and mortality are higher in patients with acute complications.

This study shows that maximum complication and mortality were noted during the first 24 h (e.g., 20% mortality), which is also comparable with the study by Munidasa et al.^[16] They suggested that maximum mortality was observed in the first 72 h. Davies et al.^[17] also supported that poor survival was noted in those patients who came late to hospital, showing high GCS score on admission, and maximum cases of survival had hospital stay of more than 5 days. So, a week is sufficient to observe clinical course of OP poisoning.

In our study, nearly, about two-thirds of patients survived, which is comparable with the study by Gohel et al.^[13] in which 70% patients recovered and 27% expired. For those patients who have passed the acute phase, chances of survival are better in them.

Conclusion

As agricultural industries are growing, OP poisons are widely used as insecticides. For diagnosis, we require detailed history and clinical examination, with the support of laboratory investigations such as AchE level. Maximum incidence of poisoning is found in younger age group. Patients are presented with muscarinic and nicotinic signs and symptoms. Among complications, ARDS is more dangerous. Chances of survival are high among patients who reached hospital earlier and received immediate treatment in intensive care unit. Injections Atropine and PAM are very helpful to treat the patients.

References

- Fukuto TR. Mechanism of action of organophosphorus and carbamate insecticides. Environ Health Perspect 1990;87:245–54.
- Corriols M, Marin J, Berroteran J, Lozano LM, Lundberg I, Thorn A. The Nicaraguan Pesticide Poisoning Register: constant underreporting. Int J Health Serv 2008;38(4):773–87.
- MD Guidelines. Toxic Effects, Organophosphate and Carbamate Pesticides. Available at: http://www.mdguidelines.com/toxic-effectsorganophosphate-and-carbamate-pesticides
- Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet 2008; 371(9612):597–607.
- Thunga G, Sam KG, Khera K, Pandey S, Sagar SV. Evaluation of incidence, clinical characteristics and management in organophosphate poisoning patients in a tertiary care hospital. J Toxicol Environ Health Sci 2014;2(5):73–6.
- Eddleston M, Singh S, Buckley N. Organophosphorus poisoning (acute). BMJ Clin Evid 2005;(13):1744–55.
- Emerson GM, Gray NM, Jelinek GA, Mountain D, Mead HJ. Organophosphate poisoning in Perth, Western Australia, 1987– 1996. J Emerg Med 1998;17(2):273–7.

- Kora SA, Doddamani GB, Halagali GR, Vijayamahantesh SN, Umakanth B. Sociodemographic profile of the organophosphorus poisoning cases in Southern India. J Clin Diagn Res 2011;5(5):953–6.
- Banerjee I, Tripathi S, Roy AS. Clinico-epidemiological characteristics of patients presenting with organophosphorus poisoning. N Am J Med Sci 2012;4(3):147–50.
- Mishra A, Shukla SK, Yadav MK, Gupta AK. Epidemiological study of medicolegal organophosphorus poisoning in central region of Nepal. J Forensic Res 2012;3:167.
- Chugh SN, Aggarwal N, Dabla S, Chhabra B. Comparative evaluation of "atropine alone" and "atropine with pralidoxime (PAM)" in the management of organophosphorus poisoning. J Indian Acad Clin Med 2005;6(1):33–7.
- Goswamy, Chaudhuri A, Mahashur AA. Study of respiratory failure in organophosphate and carbamate poisoning. Heart Lung 1994;23(6):466–72.
- Gohel DR, Panjwani SJ, Jacob C. Oximes in organophosphorous compound poisoning. J Assoc Phys India 1997;45:95–162.
- de Silva HJ, Wijewickrema R, Senanayake N. Does pralidoxime affect outcome of management in acute organophopsphate poisoning. Lancet 1992;339(8802):1136–8.

- Faiz MS, Mughal S, Memon AQ. Acute and late complications of organophosphate poisoning. J Coll Physicians Surg Pak 2011; 21(5):288–90.
- Munidasa UA, Gawarammana IB, Kularatne SA, Kumarasiri PV, Goonasekera CD. Survival pattern in patients with acute organophosphate poisoning receiving intensive care. J Toxicol Clin Toxicol 2004;42(4):343–7.
- Davies JOJ, Eddleston M, Buckley NA. Predicting outcome in acute organophosphorus poisoning with a poison severity score or the Glasgow coma scale. QJM 2008;101(5):371–9.

How to cite this article: Shah NM, Mundhra SH. Clinical profile of organophosphate poisoning at a tertiary-care center. Int J Med Sci Public Health 2016;5:1621-1625

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