Original Article

Indian Journal of Medical & Health Sciences
Volume 6 Number 1, January - June 2019
DOI: https://dx.doi.org/10.21088/ijmhs.2347.9981.6119.5

Epidemiology, Clinical Profile and Outcome of Organophosphorous Poisoning in North-Western Rajasthan

Shyam Lal Meena[1], Bal Kishan Gupta[2], Anil Kumar Ranva[3], Rakesh Kumar Sonkaria[4], Naveen Swami[5], Ranveer Singh[6], Jigyasa Gupta[7]

Abstract

Introduction: Organophosphate pesticide poisoning (OPP) is continued to be a major global health problem especially in developing countries. This study was planned to evaluate epidemiological, clinical profile and outcome of OPP admitted in tertiary care hospital North West zone of Rajasthan, India.

Materials and Methods: A total of 96 patients suffering from OP poisoning were evaluated for epidemiology, clinical profile and outcome. Diagnosis of OP poisoning was done by history, clinical examination and serum butyrylcholinesterase level assay. All patients were evaluated as per Performa and follow up till discharge. Severity of OPP was assessed by POP score and GCS score.

Results: Out of 96 cases 76 were males 20 females, age ranging 14-55 years, mean age 26 ± 8, 85.4% were below age 30 years. Most of the patients were farmer, illiterate and belonging to rural area. Most common cause of poisoning was suicidal intention and most common route of poisoning was ingestion. Chlorpyrifos was the most common salt used followed by Monocrotophos, Phorate 10%, Dimethoate 30% and Quinalphos 25%. 8.3% had mild poisoning, 65.6% had moderately severe while 26% had severe OPP as evaluated by POP score. 19 patients required ventilator support, 10 cases developed ARDS and 6 had IMS. Mortality rate was 9.4%. Monocrotophos poison, amount of poison, long duration of poisoning, Hypokalemia, high bicarbonate level, high POP score and low GCS score were associated with poor outcome

Conclusions: Thorough clinical evaluation is important for diagnosis and assessment of severity of OPP. POP score and GCS score help in assessing severity of OPP and its outcome. OPP is associated with life-threatening complications. Early detection and immediate treatment in intensive care units can increase the chances of survival rate in such cases. Banning of the most toxic pesticides may reduce the number of significant unintentional and intentional poisoning episodes.

Keywords: Organophosphate; Poisoning; POP score.

Introduction

Organophosphate pesticide poisoning (OPP) is a major challenging public-health problem in developing countries [1-3]. Organophosphorus (OP) compounds are principally used as pesticides, and their exposure is highly prevalent in developing countries. Toxic effects of OP compound are associated with significant morbidity and mortality making it a major global health problem. The easy accessibility and socio–cultural factors play an important role in the choice of OP compound as a self-poison. The incidence is higher in young, economically active group with a case fatality ratio of 4-30% [4-6]. Deaths from unintentional OP poisoning are less common than those from intentional poisoning and seem to be more common in region where highly toxic OP pesticides (WHO class I toxicity) are available [7-9].

Poisoning is a common method of suicide in India. In many Indian reports, the rates of poisoning as a suicidal method range from 20.6% (10.3% OP) to 56.3% (43.8% OP) [10]. Various studies in India have reported that poisoning rates in the suicide attempters who attend hospital varies from around 40% to over 80% and OP compounds

Author's Affiliation: [1]Assistant Professor, [2]Senior Professor, In-charge Medical ICU, [3-6]Senior Resident, [7]Junior Resident, Department of Medicine, S.P. Medical College & PBM Hospital, Bikaner, Rajasthan 334001, India.

Corresponding Author: Bal Kishan Gupta, Senior Professor, In-charge Medical ICU, Department of Medicine, S.P. Medical College & PBM Hospital, Bikaner, Rajasthan 334001, India.

E-mail: bkgbkn@rediffmail.com

Received on 10.05.2019
Accepted on 08.06.2019

available as pesticides were amongst the most common poisons used [11-14].

Materials and Methods

This study was carried out in the Department of medicine Sardar Patel Medical College & Associate Group of Hospitals Bikaner on 96 consecutive cases of OP poisoning admitted in medical wards & ICU during the period from July 2017 to June 2018. Diagnosis of OP poisoning was made by history of poisoning including container of the poison brought by patient's relative, clinical examination and measurement of serum butyrylcholinesterase activity [15].

Inclusion Criteria:

(1) Patients suffering from OP poisoning.

(2) Patients given consent to participate in the study.

Ex-Clusion Criteria:

(1) Patient admitted with other type of poisoning (other than OP) or mul-tiple poisoning.

(2) Patient suffering from any co-morbid conditions like diabetes mellitus, Ischemic heart disease, malignancy or other chronic conditions.

(3) Patients who are not giving consent for the study.

All patients were evaluated thoroughly by clinical history and physical examination as per Performa. Laboratory investigations were done in all patients at the time of admission including Complete blood count, Renal function test, Liver function test, Butyrylcholinesterase level, Acid Base Gas analysis, Serum electrolytes (Na+, K+, Ca++). Other necessary special investigations like X-ray Chest PA view, Ultrasonography, ECG, CT scan, MRI scan etc were done as per requirement. Mea-surement of serum butyrylcholinesterase was done by spectrophotometry from Lal PathLabs (Bio. Ref. Interval 4.62-11.50 kU/L). All patients were treated [16] and followed up during hospital stay as per protocol. Severity and outcome of OP poisoning was also assessed at the time of admission by Peradeniya organophosphorus poisoning (POP) scale [17] and by GCS score [18].

Results

Out of total 96 cases of OP poisoning (age ranging 14-55 years, mean age 26 ± 8, 85.4% were below age 30 years) 70 were males (72.9%, age ranging 14-55, mean age 28 ± 8) and 26 females (27.1%, age ranging 17-35, mean age 22 ± 5). Most of the patients were farmer and illiterate belonging to ru-ral area. Most common cause of poisoning was suicidal intention and most common route of poi-soning was ingestion. Epidemiologic profile is shown in Table 1.

Table 2 shows clinical evaluation at the time of hospitalization. Nausea/vomiting were present in majority (94.8%) of the cases. Excessive secretion was present in 67 cases. Altered Sensorium was present in 11 cases, pain abdomen in 7, diarrhea in 10, seizures in 1 and respiratory distress was present in 14 cases. Frothing was seen in 5 cases. Most of the cases (65.6%) had moderately severe OPP while 26% had severe OPP as evaluated by POP score. 10 cases had ARDS out of which 6 had POP score 11, one had 10 and three had PPOP score 9; GCS score was 3 in three cases, 4 in 1 case, 6 in 2, 7 in 1 and 9 in three cases. 6 cases developed IMS, their POP score was 11 in 1, 10 in 2, 9 in 1, 8 in 1 and 7 in 1 while GCS score was 4 in 2, 6 in 1, 7 in 1 and 8 in 2 cases.

Table 3 shows distribution of cases according to type of poison used. Chlorpyrifos was the most common salt used (32 patients and out of them 2 expired) followed by Monocrotophos (15 patients, 3 expired), Phorate 10% (14 patients, 2 expired), Dimethoate 30% was used by (12 patients, 1expired), Quinalphos 25% (12 patients, 1 expired) while Acephate, Malathione, Oxydemeton, and trizophos 40% each was used by 1 patient and all of them were recovered.

Table 4 shows statistical analysis of different parameters in relation to outcome. Overall mortality rate in our study was 9.4%. Hypokalemia and high bicarbonate levels were found to be significantly associated with poor outcome. Amount of poison consumed and dose of atropine required were also associated with outcome. Mortality rate was 14.3% in suicidal poisoning while it was 2.5% in acci-dental poisoning. 9 patients (13.4%) out of 67 cases because of ingestion poisoning died while all 29 cases due to inhalation poisoning survived. Mortality was 42% in patients who required ventilator support and it was 33.3% who developed IMS and 70% in patients with ARDS. Markedly reduced activity of Butyrylcholinesterase enzyme was also associated with high mortality, 21.2% (7 patients) died out of 33 patients (34.4%) who had enzyme activity <1. 8 patients had POP score 0-3 (mild poisoning) and 63 had 4-7 (moderate poisoning) all survived

Table 1: The epidemiologic profile of the cases.

Parameter	No of cases (%)	p-value
Age Group		
≤20	25 (26)	
21-30	57 (59.4)	0.009*
31-40	8 (8.3)	
>40	6 (6.3)	
Sex		
Male	70 (72.9)	
Female	26 (27.1)	>0.05
Residence		
Rural	94 (97.9)	
Urban	2 (2.1)	<0.001*
Socioeconomic status		
High	1 (1)	
Middle	3 (3.1)	<0.001*
Low	92 (95.8)	
Educational status		
Illiterate	42 (43.8)	
<Graduate	51 (53.1)	0.01*
≥Graduate	3 (3.1)	
Occupation		
Farmer	79 (82.3)	
Labourer	4 (4.2)	
House Wife	3 (3.1)	0.01*
Driver	1 (01)	
Student	9 (9.4)	
Cause of poisoning		
Accidental	40 (41.7)	
Suicidal	56 (58.3)	0.075
Route of poisoning		
Ingestion	67 (69.8%)	
Inhalation	29 (30.2%)	0.03*

Table 2: Clinical evaluations at the time of presentation

Parameter	Number of cases	%
Presenting symptoms		
Duration of Poisoning		
<3 hrs	36	37.5
3-12 hrs	48	50
>12 hrs	12	12.5
Excessive Secretion	67	69.8
Nausea/ Vomiting	91	94.8
Fasciculations	72	75
Altered Sensorium	11	11.5
Pain Abdomen	7	7.3
Diarrhea	10	10.4
Seizures	1	1
Respiratory Distress	14	14.6
Physical examination		
Miosis	96	100
Pallor	5	5.2
Frothing	5	5.2
Secretion	76	79.2
Crepts in lungs	46	47.9
ARDS	10	10.4
IMS	6	6.25
GCS Score		
14 – 15	14	14.6
9 – 13	66	68.8
3 – 8	16	16.7
POP Score (Severity of Poisoning)		
0 – 3 (Mild)	8	8.3
4 – 7 (Moderate)	63	65.6
8 – 11 (Severe)	25	26.0

Table 3: Distribution of cases according to type of poison used

Type of Poison	Total (%)	Outcome	
		Expired	Recovered
Chlorpyrifos 20%	32 (33.33%)	2 (6.25)	30 (93.75)
Monocrotophos 36%	18 (18.75%)	3 (16.67)	15 (83.33)
Phorate 10%	16 (16.67%)	2 (12.5)	14 (87.5)
Quinalphos 25%	13 (13.54%)	1 (7.69)	12 (92.31)
Dimethoate 30%	13 (13.54%)	1 (7.69)	12 (92.31)
Acephate 75%	1 (1.04%)	0	1 (100)
Malathion 50%	1 (1.04%)	0	1 (100)
Oxydemeton 25%	1 (1.04%)	0	1 (100)
Triazophos 40%	1 (1.04%)	0	1 (100)
Total	96 (100)	9 (9.38)	87 (90.62)

Table 4: Statistical analyses of different parameters in relation to outcome

Parameters	Outcome				p
	Expired		Recovered		
	Mean ± SD	Range	Mean ± SD	Range	
Age	30.56 ± 11.16	18-50	25.48 ± 7.66	14-55	0.074
Duration of poisoning	11.33 ± 10.75	6-24	5.51 ± 7.50	3-12	0.01*
Hospital Stay	7.00 ± 4.64	1-16	7.38 ± 3.24	1-19	0.750
Amount of Poisoning	292.89 ± 139.80	130-510	138.04 ± 71.40	20-456	0.034*
Amount of Atropine Used	66.67 ± 53.85	30-200	40.52 ± 29.87	10-200	<0.001*
Hemoglobin	12.73 ± 8.32	10.90-14.10	11.85 ± 2.41	4.10-18.60	0.278
MCV	88.41 ± 8.32	79.70-101.0	83.80 ± 8.46	62.00-105.20	0.123
TLC (Thousands)	14.96 ± 6.61	8-26	12.74 ± 10.59	1.2-10.2	0.541
Platelet Count (Lacs)	2.30 ± 1.11	1.26-4.57	1.90 ± 0.66	0.76-3.45	0.113
RDW	15.29 ± 0.97	14.10-16.70	15.16 ± 1.97	11.90-22.80	0.848
Alkaline Phosphatase	166.62 ± 101.81	87-398	132.57 ± 106.0	9.40-713.00	0.386
Blood Urea	41.12 ± 8.10	27-52	35.79 ± 19.72	80-165	0.452
Serum Creatinine	1.19 ± 0.34	0.80-1.90	1.60 ± 4.36	0.34-40.00	0.792
Sodium	139.71 ± 3.30	136-145	139.55 ± 17.84	14-191	0.981
Potassium	3.87 ± 0.44	2.50-4.70	4.34 ± 1.20	3.60-7.00	0.025*
pH	7.80 ± 9.96	2-25	6.33 ± 7.79	2-40	0.715
HCO_3	89.10 ± 94.93	20.80-197.5	38.76 ± 11.11	25.60-75.00	0.002*
POP Score	10.78 ± 0.19	10-11	5.89 ± 3.43	3-10	<0.0001*
GCS Score	4.33 ± 4.0	3-9	10.92 ± 4.63	6-15	<0.0001*
Butyrylcholinesterase activity	1.22 ± 0.025	<1-2.5	2.06 ± 0.81	<1-3.85	0.0003*
Mechanical Ventilator required	19 cases (19.8%) required mechanical ventilator support, 8 died				
IMS	6 cases (6.25%) developed IMS out of which 2 died				
ARDS (10, 10.4%)	10 cases (10.4%) developed ARDS out of which 7 died				

Table 5: Profile of Expired Cases

Variables	Expired Cases Serial Number								
	8	34	38	51	53	55	59	66	67
Age (years)	27	45	50	28	22	22	18	25	38
Sex	Male	Male	Male	Male	Male	Male	Female	Male	Male
On Ventilator	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Complication	ARDS	ARDS	IMS	ARDS	IMS	ARDS	ARDS	ARDS	ARDS
Duration of poisoning (hrs)	6	12	6	24	6	12	6	24	6
Amount of Poison	50	40	50	30	600	40	50	40	100
Butyrylcholinesterase activity	2.5	1.44	<1	<1	<1	<1	<1	<1	<1
Duration of Hospital stay (days)	3	5	8	16	4	1	7	12	7
Amount of Atropine Used	130	170	200	510	496	220	280	390	240
POP Score	11	11	11	10	11	11	10	11	11
GCS Score	3	3	4	9	3	3	4	6	4

while 25 had 8-11 score out of which 9 died (36%). 14 patients had GCS score 14-15 and 66 had 9-13 all survived while 16 had 3-8 score out of which 9 died (56.3%). Details of clinical profile of each expired patient are shown in table 5.

Discussion

Organophosphate pesticide poisoning has become a significant cause of death especially in the developing nations and is the most frequently used method of suicide [19]. Globally, there is a scarcity of information on the magnitude of both intentional and unintentional poisoning, as well as on the relative importance of different pesticides. Detail and accurate community-based data on the pesticides responsible for fatal self-harm are not available for most of rural Asia [20]. Recent WHO estimates suggest that more than 3.1 million cases of acute pesticide poisoning occur worldwide annually; the majority of them were being caused by OP compounds used for agricultural purposes in developing countries because of easy availability and cost [4].

Our study shows most of the cases belong to young age group (<30 years age), farmer, less literacy, low socioeconomic status and rural habitat similar observations has been made by previous workers [13,21,22]. Most common salt of OP poisoning in our study was Chlorpyrifos followed by Monocrotophos, Phorate 10%, Dimethoate 30% and Quinalphos 25% while Somasundaram et al. [21] reported Demethoate was the most common agent followed by Endosulphen and Betox while Thunga et al. [23] found most common OP compounds exposed were methyl parathion and quinolo-phos. This may be because of local availability of different OP compound. Most of the patient had nausea, vomiting, hyper salivation, excess respiratory secretion (muscarinic effects), sweating and fasciculation (nicotinic effects) which are pathognomic of OP poisoning [24].

In our study patients who came within 3 hours of poisoning were survived while longer du-ration of poisoning was associated with high mortality. The duration of presentation to the hospital is an important factor to determine the clinical course and outcome [25]. The delayed presentation of the patients in our study might be due to delayed recognition of the victim after exposure to the poi-son, delay in transport, or difficulty in access to the health-care centers. Delayed presentation after 4 h was also found by the work done by Ahuja et al. where they had mentioned that delayed initiation of resuscitative measures could be the possible contributing factor for the high mortality in their subjects [26].

The mortality with acute poisoning in a well-established centre with advanced life support is 1%–2% [27]. The mortality in our study was 9.4% which may be because of late presentation after poising and high amount of poisoning but it was less than found in earlier reported studies in India which was comparable to the mortality observed in the study by Ahuja et al. (18%) [26] and Joshi and Patel (15.8%) [28]. We found high incidence of ingestion of OP poison for suicide intention which is also associated with high mortality. Agricultural pesticide poisoning is a major contributor to the global burden of suicide [29]. Regulation of the pesticides used can result in a sustained overall reduc-tion in the number of deaths from intentional pesticide poisoning [30]. Studies has shown that POP score 17 and GCS score 18 are important to assess prognosis in OPP similarly we also found high POP score and low GCS score were associated with high mortality, more complications like ARDS, IMS, requirement for ventilator support and prolonged hospital stay [31].

Conclusion

Thorough clinical evaluation by detailed history and physical examination is important for diagnosis and assessment of severity of OPP. POP score and GCS score help in assessing severity of OPP and outcome. Life-threatening complications occurred in these patients. Early detection and immediate treatment in intensive care units with appropriate measure can increase the chances of survival. Banning of the most toxic pesticides may reduce the number of significant unintentional and intentional poisoning episodes.

References

1. Jeyaratnam J. Acute pesticide poisoning: a major global health problem. World Health Stat Q. 1990;43:139–44.
2. Van der Hoek W, Konradsen F, Athukorala K, Wanigadewa T. Pesticide poison-ing: a major health problem in Sri Lanka. Soc Sci Med. 1998;46:495–504.
3. Eddleston M, Phillips MR. Self poisoning with pesticides. BMJ. 2004;328:42–44.
4. Sungur M, Guiven M. Intensive care management of organophosphate insecticide poisoning. Crit Care. 2001;5(4):211-15.

5. Eddlestron M, Szinicz L, Eyer P. Oximes in acute organophosphorus poisoning: a systematic review of clinical trials. QJM. 2002;275–283.
6. Cherian MA, Roshini C, Visalakshi J, Jeyaseelan L, cherian AM. Biochemical and Clinical Profile After Organophosphorus Poisonning – A Placebo – Controlled Trial using Pralidoxime. JAP1. 2005; 53:427-30.
7. WHO. Public health impact of pesticides used in agriculture. World Health Or-ganization; Geneva: 1990.
8. McConnell R, Hruska AJ. An epidemic of pesticide poisoning in Nicaragua: im-plications for prevention in developing countries. Am J Public Health. 1993;83:1559–62.
9. Rosenthal E. The tragedy of Tauccamarca: a human rights perspective on the pes-ticide poisoning deaths of 4 children in the Peruvian Andes. Int J Occup Environ Health. 2003;9:53–58.
10. Nandi DN, Mukherjee SP, Banerjee G, Ghosh A, Boral GC, Chowdhury A, Bose J. Is suicide preventable by restricting the availability of lethal agents? A rural survey of West Bengal. Ind J Psychiatry. 1979;21:251–55.
11. Badrinarayana A. Suicide attempt in Gulbarga. Ind J of Psychiatry. 1977;19:69–70.
12. Karalliedde L, Eddleston M, Murray V. The global picture of organophosphate in-secticide poisoning. In: Karalliedde L, Feldman S, Henry J, Marrs T, edi-tors. Organophosphates and health. edn. Imperial College Press; London: 2001.pp.431–71.
13. Gupta SK, Kumar S, Sheikh MI. Study of organophosphorus poisoning in Surat, India. JIAFM. 2006;28(3):0971-0973.
14. Gunnell D, Eddleston M Suicide by intentional ingestion of pesticides: a continu-ing tragedy in developing countries. Int J Epidemiol. 2003 Dec; 32(6):902-9.
15. Jońca J, Żuk M, Wasąg B, Janaszak- Jasiecka A, Lewandowski K, Wielgomas B, et al. New Insights into Butyrylcholinesterase Activity Assay: Serum Dilu-tion Factor as a Crucial Parameter. PLoS ONE. 2015;10(10):e0139480. doi:10.1371/journal. pone.0139480.
16. Wadia RS. Treatment of organophosphate poisoning. Indian J Crit Care Med; 2003;7:85–87.
17. Pradeep. V. Vernekar 1, Dr Kiran Shivaraj. Peradeniya organophosphorus poisoning scale (POP) as a predictor of respiratory failure and mortality in or-ganophosphorus poisoning. Sch J App Med Sci; 2017;5(5B):1841-44.
18. 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):371–79. doi:10.1093/qjmed/ hcn014.
19. Bertolote JM, Fleischmann A, Eddleston M, Gunnell D. Deaths from pesticide poisoning: a global response. Br J Psychiatry. 2006 Sep;189:201-3.
20. Chintale KN, Patne SV, Chavan SS. Clinical profile of organophosphorus poison-ing patients at rural tertiary health care centre. Int J Adv Med 2016;3:268-74.
21. Somasundaram KV, Pail A, Shukla SK. Epidemiological profile of OP poisoning cases treated at pravara hospital, Loni, India. Ind J Prev Soc Med. 2009;40(3&4):184-88.
22. Dayanand Raddi, Anikethana G V Clinical profile of organophosphorus poisoning in a tertiary care hospital Indian Journal of Basic and Applied Medical Research. 2014 Dec;4(1):14-22.
23. Thunga G, Sam KG, Khera K, Pandey S, Sagar SV. Evaluation of incidence, clin-ical characteristics and management in organophosphorus poisoning patients in a tertiary care hospital. J Toxicol Environ Hlth Sci. 2010;2(5):73-78.
24. Bhagwat K. A review on organophosphorus toxicity in the farmers of solapur dis-trict from India. International Journal of Biological Research. 2014;2(2):69-77.
25. Ramesha KN, Rao KB, Kumar GS. Pattern and outcome of acute poisoning cases in a tertiary care hospital in Karnataka, India. Indian J Crit Care Med. 2009;13:152–5.
26. 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. J Clin Diagn Res. 2015;9:UC01–4. [PMCID: PMC4625313] [PubMed: 26557594].
27. Boukatta B, El Bouazzaoui A, Guemoune R, Houari N, Achour S, Sbai H. An epidemiological study of adult acute poisoning in Fez: Morocco. J Clin Toxicol. 2014;4:1–5.
28. Joshi M, Patel DV. A study on clinical profile of patients with acute poisoning. GCSMC J Med Sci. 2015;4:97–100.
29. Mew EJ, Padmanathan P, Konradsen F, Eddleston M, Chang SS, Phillips MR, Gunnell D. The global burden of fatal self-poisoning with pesticides 2006-15: Systematic review. Journal of Affective Disorders. 2017;219:93–104.
30. Gunnell D, Knipe D, Chang SS, Pearson M, Konradsen F, Lee WJ, Eddleston M. Prevention of suicide with regulations aimed at restricting access to highly ha-zardous pesticides: a systematic review of the international evidence. Lancet. Glob Health. 2017;5:e1026–37.
31. Rajbanshi LK, Arjyal B, Mandal R. Clinical Profile and Outcome of Patients with Acute Poisoning Admitted in Intensive Care Unit of Tertiary Care Center in East-ern Nepal. Indian J Crit Care Med. 2018;22(10):691–96. doi:10.4103/ijccm. IJCCM_207_18.