Original Article

Predictors of Morbidity and Mortality in Organophosphorus Poisoning: A Case Study in Rural Hospital in Karnataka, India

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Abstract

Background: Organophosphorus (OP) pesticides poisoning can result from occupational, accidental or intentional exposure. Clinical manifestations include cholinergic syndromes, central nervous (CNS) system and cardiovascular disorders. Death is usually due to cardiovascular and respiratory failure. **Aim:** To evaluate various parameters that can predict outcome of patients in OP poisoning. **Materials and Methods:** A prospective study conducted in Department of Medicine, Adichunchingiri Institute Of medical Sciences and Research Centre, Karnataka, over period of 1 year. Diagnosis of OP poisoning was based on clinical history of exposure to OP compound and low serum pseudocholinesterase levels. **Results:** In the present study 133 patients were enrolled, out of which 98.5% were suicidal cases and only 1.5% had accidental exposure. Majority of cases were young male, with F/M ratio 1:3.2. Mortality rates were higher in younger people and in patients who required prolonged ventilator support. The mortality rate was directly proportional to amount of poison consumed, lag time, organ failure (Acute Renal Failure) and plasma pseudocholinesterase levels. Acute complications were frequently noted and were related to morbidity and mortality. No strict relationship was found between liver dysfunction, electrolyte disturbance and clinical outcome. **Conclusion:** This case study concluded that mortality is directly proportionate to the lag time, amount of OP substances consumed, clinical severity, pseudocholinesterase levels, Acute renal failure and duration of ventilatory support. This study highlights the importance of rapid diagnosis, and initiation of early and effective treatment, which may result in less number complications and also decreases the mortality rates.

Keywords: Acetylcholinesterase, Atropine, Organophosphorus compounds, Pseudocholinesterase, Respiratory failure

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Introduction

Organophosphate compounds are widely used as pesticides in agricultural parts of the world.[1] Toxicity of organophosphates is the result of excessive cholinergic stimulation through inhibition of acetylcholinesterase.

Serum cholinesterase level is depressed after organophosphorus (OP) poisoning, as reported by previous various studies. Confirmation of organophosphate poisoning is based on the measurement of cholinesterase activity. Although red blood cell (RBC) and plasma pseudocholinesterase levels can both be used, RBC cholinesterase correlates better with central nervous system (CNS). Acetylcholinesterase is, therefore, a more useful marker for organophosphate poisoning.[2] The rapid accumulation of acetylcholine in the synaptic junctions of CNS and peripheral tissues results in a cholinergic crisis, characterized by range of muscarinic, nicotinic and central effects.[2]

Gastric mucosa is permeable to organophosphates, and is a classical way of absorption in suicidal cases. Liver is the organ where activation and detoxification of organophosphate compound takes place, but they are eliminated primarily through kidneys.[3]

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Clinical manifestations are broadly classified into muscarinic and nicotinic which include; bradycardia, hypotension (Muscarinic), tachycardia (nicotinic), increased salivation/lacrimation, excessive sweating, nausea, vomiting, diarrhea, pain abdomen, fecal and urinary incontinence.[4] CNS manifestations include anxiety, restlessness, convulsion, miosis, insomnia, coma, cheyne-stokes breathing, respiratory and cardiovascular failure.[5]

Intermediate syndrome or type II paralysis usually occurs after 24-96 hours after acute cholinergic crisis. Incidence of Intermediate syndrome varies from 8-50%.[6] Chronic OP poisoning can cause organophosphate-induced delayed neuropathy and is seen mostly in agricultural workers.[7]

The initial management of acute OP poisoning includes cardio respiratory stabilization, decontamination (removal of clothes for possible source of continued exposure in occupational intoxication), irrigation of skin and eyes as well as gastric lavage and activated charcoal to minimize absorption of the OP compound.[8] The mainstay of treatment involves atropine — A central and peripheral muscarinic receptor antagonist and pralidoxime chloride, which reactivates inhibited acetyl cholinesterase.[9] In recent years new adjunct therapy and cheap medications such as sodium bicarbonate, magnesium sulfate as well as antioxidants have been considered for the management of OP poisoning.[10] Death is usually occurs due to cardiovascular and respiratory failure, paralysis of respiratory muscles and obstruction caused by bronchospasm and bronchial secretions.[11]

Materials and Methods

We enrolled 133 patients in a prospective study which was conducted in Department of Medicine, Adichunchingiri medical college, Bangalore from July 2013 to June 2014. Institute ethical committee approved the study. All patients of OP poisoning were included in this study. However, we excluded those patients in whom OP poisoning was doubtful. Detailed history was taken from all the patients' relatives about the circumstances of poisoning. Detailed clinical examination of the patients was done. Diagnosis of OP poisoning was based on clinical features, history of exposure to a known OP compound and was supported by low serum pseudocholinesterase levels. Patients were treated as per the standard protocol of organophosphate poisoning with respiratory support, atropine and pralidoxime. All patients were dealt up to recovery or death from poisoning. Psychiatric consultation was done in all the cases that recovered, before discharging them from the hospital.

Baseline investigations included complete blood hemogram, urea, creatinine, arterial blood gas values, X-ray chest and serum pseudo-cholinesterase level. Data was retrieved from the files on a structured proforma. Studied variables included gender, age, amount of organophosphate consumed, mode of exposure, time lag in starting treatment, duration of ventilator support and hospital stay, acute complications and outcome of patients.

Type of organophosphorus compound consumed

We could identify the type OP compound in most of the cases, patient attenders would bring the bottle of OP compound, that patient would have consumed, to our Emergency department. The various types of OP substances consumed in the present study are as follows.

Diethomate ($N = 33$), chloropyrifos ($N = 15$), Quinalophos ($N = 12$), Acetamide ($N = 1$), dichlorrofos ($N = 5$), Dicofol ($N = 1$), Emamectin, endosulfan ($N = 1$), Ethiophos ($N = 2$), Glycophosate ($N = 1$), carbosulfan ($N = 1$), methylparathion ($N = 3$), Monocrotophos ($N = 5$), Phorate ($N = 1$), profens ($N = 3$), Profenofos ($N = 2$), Profenofos/cypromethrin ($N = 1$), Unknown OP compound ($N = 45$), Dermal exposure ($N = 2$).

Methodology

Serum pseudocholinesterase level was estimated at the time of admission in all the patients by DGKC method (LiquiChek). The laboratory reference range of pseudocholinesterase used in the present study was Female = 3930-10800/l, male 4620-11500 u/l. Based on the serum pseudocholinesterase levels, the severity of poisoning was defined as per Kumar *et al*.[12]

Mild poisoning: Pseudocholinesterase level 20-50% of normal or >1,401-3,500 IU/L.

Moderate poisoning: Pseudocholinesterase level 10-20% of normal or 701-1, 400 IU/L.

Severe poisoning: Pseudocholinesterase level is <10% of normal or < 700 IU/L.

Statistical study

Data were presented either as mean ± standard deviation (SD) or as percentage. Probability values of $P < 0.05$ were considered significant, and all statistical analyses were performed using SPSS version 12.0. Fisher's exact test was used for categorical data.

Results

In present study, 133 cases of OP poisoning were admitted during the study period [Table 1]. One thirty

one (98.5%) patients ingested the compound and only two patients (1.5%) had dermal/inhaled exposure while spraying pesticides in rice fields [Table 1]. One hundred two (76.7%) were males and 31 (23.3%) female. Most of the cases were young people 80% (< 40years) predominantly males [Table 1]. There was wide variation in age ranging from a minimum of 13-68 years with mean age of 31.5 years [Table 2].

Forty eight patients (36.1%) out of 133 were stable after gastric lavage. They were kept under observation for the next 3 days and finally discharged. The clinical presentation of acute poisoning was variable as shown in [Table 3]. However, the most consistent feature was miosis (93.2%). Eighteen (13.5%) patients developed episodic convulsions. Transient elevation in liver enzymes were noticed in 13.5% patients [Table 3]. However, no significant increase in morbidity/mortality was seen in patients with hypokalemia or deranged liver function test [$P > 0.05$, Table 4]. Patient who developed single /multi-organ failure had increased mortality [$P < 0.0001$, statistically highly significant Table 4].

Fifty three patients required ventilatory support, out of which only 11 patients survived [Table 5]. Patients were on ventilator support for minimum 1 day to maximum 22 days with a mean 6.85 ± 4.32 days [Table 2]. Mortality was higher in patients who required ventilator support >7 days [$P < 0.05$ statistically significant, Table 5].

The amount of OP compound consumed ranges from 10 ml to maximum 200 ml with mean 77.5 [Table 2]. The mortality rate was directly proportional to the amount of poison consumed [$P < 0.00003$ statistically significant, Table 5].

The lag time for initiation of treatment was minimum 1.02 hours to maximum 9.57 hours with mean 4.65 ± 2.4 [Table 2]. Mortality rate was higher in patients with lag time > 6.5 hours [$P < 0.05$, Table 5].

Twenty-one out of 133 (15.7%) developed derangement in renal function tests (defined as serum creatinine >1.4). In most of cases, derangement of the renal function was reversible and renal function tests improved within a week. However, increased mortality was seen in patients with serum creatinine >3.5 mg/dl [$P < 0.05$, Table 5]. Out of three cases that had irreversible renal failure, one case had serum creatinine of 10.2 mg/dl on day of admission with severe metabolic acidosis and he died on the same day due to cardiac arrest. Rest of the two cases with Acute renal failure died within a week's time.

The overall mortality rate was 33.3% (42 out of 126 patients) and seven cases (5.2%) were discharged against medical advice [Table 1]. Delayed complications like mild sensory loss of lower limbs or weakness of limbs were uncommon in our patients on follow-up.

Discussion

OP compounds were synthesized by von Hoffman. OP pesticide poisoning is common in developing worlds.[11] The highest incidence is seen in India.[13] Suicidal and non-suicidal organophosphate poisoning is a major problem in rural areas of India, with rapidly increasing incidence rate.[14]

In our study the female to male ratio is 1:3.2. In the present study, the incidence of poisoning was higher in males than in females (76.6% Vs. 23.3%). Similar trend was also observed by Safdar *et al.*,[15] and Aziza *et al*.[16] However, the

Table 1: Showing number and percentage of patients

Total no. of patients *n* = 133, (7 patients discharged against medical advise)		Gender		Age		Mode of poisoning	
		Male (*N* = 102) (76.7%)	Female (*N* = 31) (23.3%)	<40 years (%)	>40 years (%)	Suicidal (%)	Accidental (%)
Survived	84 (63.15%)	73 (54.8)	18 (13.5)	79 (59.3)	12 (9.02)	89 (66.9)	2 (1.5)
Expired	42 (31.6%)	29 (21.8)	13 (9.7)	27 (20.3)	15 (11.2)	42 (31.6)	0 (0)
Total	126	102	31	106	27	131	2

Table 2: Showing mean, median and standard deviation of variables

	Minimum	Maximum	Median/Mean	SD
Age	13 years	68 years	28/31.5	12.98
Time bet consumption and hospitalisation (lag time)	1.02 hours	9.57	4.05/4.65	2.433
Cholinesterase level	330	1890	700/905	450.23
Hospital stay	1 day	28 days	7.89/ 11.195	7.81
Amount of poison consumed	10 ml	200 ml	50/77.5	54.86
No of days on ventilator support	1 day	22 days	6.857	4.32

female to male ratio given by Ather *et al.*, is 1:1 and Tall *et al.*, is 1:1.8 which is quite different from present study.[17,18]

The age ranged from 13-68 years with mean age was 31.5 years. However, Hayden *et al.*,[19] showed age range from 13-47 years with a mean age of 23 years. In the present study, the incidence of OP poisoning, was highest in patients aged less than 40 years. Majority of the cases (80%) were young people, predominantly males from the age group 13-40 years, this is comparable to other studies as done by Khan MN *et al.*,[20] in which maximum number of patients were between 15-35 years of age. The people in this age group are described to be the most ambitious and more vulnerable to various emotional conflicts that may occur during this phase of life. Our observation was similar to the previous studies that showed the highest incidence of OP poisoning in people aged between 21-39 years.[21]

Table 3: Showing clinical manifestations and complications in patients

Variable	No. of patients N = 133	Percentage (%)
Presenting symptoms		
Anxiety/Restlessness	110	82.7
Loss of consciousness/Altered Sensorium	45	33.8
Severe bradycardia at time of Admission	33	24.8
Lacrimation/Salivation	115	86.4
Urinary/Faecal incontinence	78	58.6
Miosis	124	93.2
Bronchospam	104	78.2
Hypotension	15	11.3
Fits	18	13.5
Deranged renal function test serum creatinine >1.4 mg/dl	21	15.03
Deranged liver function test	18	13.5
Hypokalemia	20	15
Complications		
Single organ failure/Respiratory failure	45/133	33.8
Multi – Organ failure	39/133	29.3
Required ventilator support	53/133	39.8

In our study, the incidence of suicidal poisoning is 98.6%, probably because it is cheap, easily available and used as a major pesticide in agricultural farming throughout India. This was in agreement with other studies[16,22] which showed deliberate self-poisoning varying from 68-96%. In the study by Aziza *et al.*,[16] 76.92% cases were suicidal and 23.07% were accidental.

The present study found that the level of serum pseudocholinesterase levels on the day of presentation was reduced in majority of cases of OP poisoning. Also it was observed that the need for intubation and oxygen requirement were more in moderate to severe cases with pseudocholinesterase <1400 U/L. In addition mortality was highest in cases with pseudocholinesterase <700 U/L ($P < 0.05$). This showed a significant association of rate of mortality with the serum pseudocholinesterase levels. Similar

Table 4: Showing outcome of patients with organ failure, liver injury and electrolyte imbalances

	Hypokalemia (N = 20)			Elevated liver enzymes (N = 18)			Organ failure (N = 73)
	<2.5	2.5-3	3-3.5	36-100	100-200	>200	Single organ failure
Survived	8	6	3	11	2	5	32
Expired	2	1	0	0	1	2	13
Total no of patients	10	7	3	11	3	7	45
P value	*P* = 1.0			*P* = 0.115			*P* = 0.0001

Table 5: Showing outcome in patients with acute renal failure, severity of poisoning, lag time and duration of mechanical ventilation

	Acute renal failure S. Creatinine (MG/DL) (N = 21)			Lag time (Hours) (N = 133)			No of days on ventilator (N = 53)			Pseudo cholinesterase levels (U/L) (N = 133)			Amount of poison consumed (ML) (N = 133)		
	<1.4	1.5-3.5	>3.5	<3.5	3-6.5	>6.5	<2	2-7	>7	<700	700-1400	>1400	<50	50–100	>100
Survived	112	13	5	49	31	11	6	4	1	38	30	23	31	30	30
Expired	0	0	3	11	14	17	7	16	19	16	12	14	2	15	25
Total no of patients	112	13	8	60	45	28	13	20	20	54	42	37	33	45	55
P Value Fisher exact	*P* = 0.001			*P* = 0.002187			*P* = 0.015			*P* = 0.0012			*P* = 0.00003		

to our finding, Mehta *et al.*,[23] observed lower activity of pseudocholinesterase in more than 70% of cases at presentation. Goswamy *et al.*[24], concluded that apart from clinical indicators, low serum cholinesterase levels were of greatest predictive value for ventilation in OP poisoning. However, Nouira *et al.*[25] did not find any statistically significant difference in mean serum cholinesterase levels in those mechanically ventilated and those do not need ventilator support.

Hypokalemia, hyperglycemia, acute renal failure, transient elevation of liver enzymes can occur in OP poisoning.[26] In our study, hypokalemia and transient elevation of liver enzymes were found in 15.03% and 13.5 % of cases respectively. Wang WZ *et al.*[27] observed that liver injury was seen in 9.8% and 5.17% of cases and control group, respectively in OP poisoning and mortality was higher in cases than controls (22.5% vs 6.32%). However, we failed to document a strict relationship between derangement of serum potassium and liver enzymes with the severity of OP poisoning and clinical outcome ($P > 0.05$).

Acute renal failure was reported following exposure to OP poisoning in 15.03% of patients. Eighteen patients out of twenty one cases, had transient reversible acute renal failure and three cases (2.2%) had irreversible renal failure. Mortality was higher in patients with acute irreversible renal failure (serum creatinine >3.5 mg/dl). Arefi M *et al.*[28] in their study found 16.7% of OP poisoning had renal failure which is similar to our study. Similarly, S panda *et al.*[29] observed altered renal function between the survivors and non survivors suggesting their importance in predicting mortality. The transient renal injury may be due to both a direct action of the organophosphate, causing tubular cell necrosis or secondary mechanism that followed the cholinergic crisis, causing hypovolemic shock and rhabdomyolysis.

The most frequent signs noted in this study were miosis 93.2%, increased salivation 86.4%, anxiety and restlessness 82.7%, bronchospasm 78.2% and incontinence in 58.6%. Other frequent clinical features noted in this study are mentioned in Table 3 with percentages, also comparable with other studies.[30,31] In the present study there were 33.8% cases of presented with altered sensorium or loss of conscious, in majority of cases it was subsequently followed by deep coma. Sequeira *et al.*[32] showed the frequency of deep coma to be 21%. Acute complications seen in this study were episodic convulsions in (13.5%) patients, severe bradycardia (24.8%), hypotension (11.3%) patients. Serious ventricular arrhythmias were not observed. Acute complications[33] were bradycardia in 29 (93.5%), change in mental status in 10 (32.2%), low oxygen saturation (less than 90%) in 21 (67.8%) and subsequent convulsions in 3 (9.6%).

The duration of mechanical ventilation in our patients was 6.857 ± 4.32 days. In the present study, the mortality was highest in patients requiring mechanical ventilation for more than 7 days ($P < 0.05$), probably due to lung complications from prolonged mechanical ventilation and increased lag time. The high mortality in patients ventilated for <2 days and between 2-7days is most probably due to the severity of poisoning.[8] The mortality following OP poisoning varies from 4-30%.[34] In a study by Safdar *et al.*,[15] 4% of patients who received mechanical ventilator support ultimately expired. In another study, mortality was 50% in patients requiring mechanical ventilation.[22] In contrast to these observations, Aziza *et al.*[16] reported 8% mortality in patients who received mechanical ventilation.

In our study, it became evident also that most of patients who expired, there was a delay (maximum 9.57 hours) between consumption of OP substance and initiation of treatment, which is also supported by study done by Suleman MI *et al.*[35] Majority of the patients with a lag time less than 6.5 hrs recovered and survived, whereas the recovery and survival of patients decreased with the increase in lag time. Moreover, the patients with increased lag time required increased duration of mechanical ventilation.[8]

Mortality was higher in those cases that had consumed large amount of OP substances (50-100 ml) and was highest in cases who consumed >100 ml [$P < 0.05$, Table 5]. In present study, morbidity and mortality was proportionately higher in majority of patients who developed single/mutli-organ failure and required prolonged ventilatory support ($P < 0.05$, Table 4]. In our study, overall mortality rate was 33.3%, which is higher than shown in a study done by Numidasa UA *et al.*,[36] and Pandyal BP *et al.*[37] However, frequency of mortality due to organophosphates given by Yamashita *et al.*,[34] varied between 4% and 30% and 5.5% in a study by Malik *et al.*[33] The reason for higher mortality rates may be due to late arrival, not receiving any treatment at periphery before arrival to the hospital, poverty and illiteracy, unawareness regarding mortality rate of OP poisoning and non availability of intensive care unit (ICU) facilities.[36]

Conclusion

OP poisonings is common in developing worlds and is of great concern, since it affects the most productive age group of the society. OP poisoning has become an agent of choice for self-poisoning because of its easy

availability and low-cost factor especially in rural India. In OP poisonings, acute complications are seen more frequently than chronic complication. Mortality and morbidity are directly proportionate to the lag time in initiation of treatment and/or amount of OP substances consumed, clinical severity (single/multiorgan failure) and duration of ventilatory support. Mortality is also higher in patients who immediately develop acute complications like severe bradycardia and severe acute renal failure. Although each predictor (age, lag time, severity of poisoning, amount of organophosphate consumed, organ failure, acute kidney injury and duration of ventilation) is associated with mortality, death due to organophosphate poisoning results from overlapping contribution of these factors. No single factor is independently responsible for mortality in these patients. Therefore, the importance of rapid diagnosis, early and effective treatment should not be overlooked because patients who receive early and effective treatment generally do better and have less complications and decreased morbidity and mortality rate. Good supportive and ICU care cannot only reduce the frequency of acute or chronic complications, but will also decrease mortality rate in these cases.

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References

1. Afshari R, Majdzadeh R, Balali-Mood M. Pattern of acute poisonings in Mashhad, Iran 1993-2000. J Toxicol Clin Toxicol 2004;42:965-75.
2. Jeyaratnam J. Acute pesticide poisoning: A major global health problem. World Health Stat Q 1990;43:139-44.
3. Eddleston M. Patterns and problems of deliberate self-poisoning in the developing world. QJM 2000;93:715-31.
4. Lotti M, Moretto A. Cholinergic symptoms and Gulf War syndrome. Nat Med 1995;1:1225-6.
5. Singh G, Khurana D. Neurology of acute organophosphate poisoning. Neurol India 2009;57:119-25.
6. Yang CC, Deng JF. Intermediate syndrome following organophosphate insecticide poisoning. J Chin Med Assoc 2007;70:467-72.
7. Emerick GL, Peccinini RG, de Oliveira GH. Organphosphorus-induced delayed neuropathy: A simple and efficient therapeutic strategy. Toxicol Lett 2010;192:238-44.
8. Wadia RS. Treatment of organophosphate poisoning. Indian J Crit Care Med 2003;17:85-7.
9. Singh S, Chaudhry D, Behera D, Gupta D, Jindal SK. Aggressive atropinisation and continuous pralidoxime (2-PAM) infusion in patients with severe organophosphate poisoning: Experience of a northwest Indian hospital. Hum Exp Toxicol 2001;20:15-8.
10. Pajoumand A, Shadnia S, Rezaie A, Abdi M, Abdollahi M. Benefits of magnesium sulfate in the management of acute human poisoning by organophosphorus insecticides. Hum Exp Toxicol 2004;23:565-9.
11. Yanagisawa N, Morita H, Nakajima T. Sarin experiences in Japan: Acute toxicity and long-term effects. J Neurol Sci 2006;249:76-85.
12. Kumar SV, Fareedullah MD, Sudhakar Y, Venkateswarlu B, Kumar EA. Current review on organophosphorus poisoning. Arch Appl Sci Res 2010;2:199-215.
13. Rodgers ML. OP poisoning. Am J Emerg Med 2006;22: 335-44.
14. Gupta SK, Peshin SS, Srivastava A, Kaleekal T, Pandian TV. An epidemiological pattern of poisoning in India. Pharmacoepidemiol Drug Saf 2002;11:73-4.
15. Safdar A, Saeed A, Muhammad NR. Organophosphorus poisoning: Emergency management in intensive care unit. Prof 2003;10:308-14.
16. Hussain AM, Sultan ST. Organophosphorus insecticide poisoning: Management in surgical intensive care unit. J Coll Physicians Surg Pak 2005;15:100-2.
17. Ather NA, Ara J, Khan EA, Sattar RA, Durrani R. Acute organophosphate insecticide poisoning. J Surg Pak 2008;13:71-4.
18. Tall A, Ly AB, Nabeth P, Sall AA, Sarr FD, Faye J, *et al*. Epidemiological investigation of death cases by pesticide poisoning. J Agric Biotechnol Sustainable Develop 2010;2:51-5.
19. Hayden K, Norton M, Darcey D, Ostbye T, Zandi P, Breitner J, *et al*., Cache County Study Investigators. Occupational exposure to pesticides increases the risk of incident AD: The Cache County study. Neurology 2010;74:1524-30.
20. Khan Ra, Rizvi SL, Ali MA, Hasan SM. Pattern of intoxication in poisoning cases; reported in casualty of Bahawal Victoria Hospital Bahawalpur. Med J 2003;10:236-8.
21. Jamil H. Organophosphorus insecticide poisoning. J Pak Med Assoc 1989;39:27-31.
22. Sungur M, Güven M. Intensive care management of organophosphate insecticide poisoning. Crit Care 2001;5:211-5.
23. Mehta AB, Shah AC, Joshi LG, Kale AK, Vora DD. Clinical features and plasma acetylcholinesterase activity in poisoning with insecticidal organophosphorus compounds. J Assoc Physicians India 1971;19:181-4.
24. Goswamy R, Chaudhuri A, Mahashur AA. Study of respiratory failure in organophosphate and carbamate poisoning. Heart Lung 1994;23:466-72.
25. Nouira S, Abroug F, Elatrous S, Boujdaria R, Bouchoucha S. Prognostic value of serum cholinesterase in organophosphate poisoning. Chest 1994;106:1811-4.
26. van der Hoek W, Konradson F. Analysis of 8000 Hospital admission for acute poisoning in rural areas of Sri Lanka. Clin Toxicol (Phila) 2006;44:225-31.
27. Wang WZ, Li YQ, Zhang JZ, Wang L, Ma GY, Cao SQ. Effect of the pre-hospital systematic treatment on prognosis patients of with severe acute organophosphoruspesticide poisoning. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi 2005;23:371-3.
28. Arefi M, Taghaddosinejad F, Salamaty P, Soroosh D, Ashraf H, Ebrahimi M. Renal failure prevalence in poisoned patients. Nephrourol Mon 2014;6:e11910.
29. Panda S, Nanda R. Laboratory abnormalities in patients

with organophosphorous poisoning Indian Medical Gazette – JANUARY 2014.
30. Tahir MH, Raja JI, Haq IU. Acute organ phosphorus poisoning – An experience. Pak Armed Forces Med J 2006; 56:150-6.
31. Karki P, Ansari JA, Bhandary S, Koirala S. Cardiac and electrocardiographical manifestation of acute organophosphate poisoning. Singapore Med J 2004;45:385-9.
32. Sequeira JF, Andre P, Miranda, Sa J. Intoxication by organophosphates: Prognostic evaluation on 143 patients. Med Intern 1994;1:4-10.
33. Malik GM, Mubarik M, Romshoo GJ. Organphosphorus poisoning in the Kashmir Valley, 1994-1997. N Engl J Med 1998;338:1078.
34. Yamashita M, Yamashita M, Tanaka J, Ando Y. Human mortality in organophosphate poisoning. Vet Hum Toxicol 1997;39:84-5.
35. Suleman MI, Jibran R, Rai M. The analysis of organophosphate poisoning case treated at Bhawal Victoria Hospital, Bahawalpur 2000-2003. Pak J Med Sci 2006;22:244-9.
36. Numidasa 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;42:343-7.
37. Paudyal BP. Poisoning: Pattern and profile of admitted cases in a hospital in central Nepal. JNMA J Nepal Med Assoc 2005;44:92-6.

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