Clinical Investigation

Survival pattern in patients with acute organophosphate poisoning on mechanical ventilation: A retrospective intensive care unit-based study in a tertiary care teaching hospital

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ABSTRACT

Background and Aims: Organophosphorus (OP) compound poisoning is one of the most common poisonings in India. The aim of the study was to study the outcomes and predictors of mortality in patients with acute OP poisoning requiring mechanical ventilation. **Methods:** A retrospective study was conducted in the intensive care unit and 117 patients were included. Diagnosis was performed from the history taken either from the patient or from the patient's relatives. Demographic data, month of the year, mode of poisoning, common age group, duration of mechanical ventilation, time of starting pralidoxime (PAM), and mortality were recorded. Chi square test, Pearson correlation test, and multivariate binary logistic regression analysis was used. Data are presented as mean ± SD. **Results:** 91.86% (79/86) of cases were suicidal and remaining cases were accidental. Duration of mechanical ventilation varied from less than 48 hours to more than 7 days. Mortality rate was 33.3%, 7.2%, and 100% in those who required mechanical ventilation for more than 7 days, 5 to 7 days, and 2 to 4 days, respectively. Lag time was less than 6 hrs in 13 patients and all of them survived. 17.1% and 28.1% patients died in whom PAM was started 6 to 12 hrs and 13 to 24 hrs after poisoning, respectively. There was statistically significant positive correlation between lag time of starting of PAM with duration of mechanical ventilation and total dose of PAM ($P < 0.0001$). None of the predictors age, lag time, severity of poisoning, and duration of ventilation were independent predictors of death. Overall mortality rate was 18.6%. **Conclusion:** Mortality from OP compound poisoning is directly proportionate to the severity of poisoning, delay in starting PAM, and duration of mechanical ventilation. Death is not dependent on a single factor, rather contributory to these factors working simultaneously.

Key words: Atropine, intensive care unit management, organophosphorus poisoning, pralidoxime

INTRODUCTION

Globally, organophosphorus (OP) pesticide poisoning is a serious occupational hazard accounting for more than 80% of pesticide-related hospitalisation.[1] India being an agriculture-based country, OP pesticide remains the main agent for crop protection and pest control. It is therefore likely to have adverse effects on farmers who are accidentally over exposed while handling these pesticides. However, because of low cost and easy availability, it has also become an agent of choice for self poisoning.[2]

Pesticide self-poisoning is responsible in killing approximately 300,000 people worldwide every year and mostly from rural background. In developing countries the mortality can be as high as 70%.[3,4] High mortality could be probably due to lack of hospital services in the vicinity, inadequate transport facility, increased patient to care givers ratio, and finally non-availability of definite antidote.[3,5]

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OP insecticides inhibit both cholinesterase and pseudocholinesterase activities, as they are irreversible cholinesterase inhibitors. The inhibition of cholinesterase activity leads to accumulation of acetylcholine at synapses, causing overstimulation and disruption of neurotransmission in both central and peripheral nervous systems.[6]

The fatal issue is often related to a delay in diagnosis or an improper management. Management of severe poisoning is difficult, requiring intensive care and use of atropine and oxime cholinesterase reactivators. Key to survival lies in early diagnosis followed by rapid decontamination and definitive therapy which purely lies under the expert domain of emergency medicine.

In this article, we report the retrospective analysis of patients with severe OP insecticide poisoning in a tertiary care hospital in Northern India.

METHODS

A retrospective study was conducted on patients with OP poisoning admitted to our intensive care unit (ICU) between October 2005 and September 2011. Ethical clearance was obtained from the hospital administration for the disclosure of the records which was only for academic purpose. However, the confidentiality of the patients was maintained by not mentioning the name, registration number, and the date of birth of the patient.

All the OP poisoning victims attending to the Accident and Emergency (A and E) and subsequently requiring ICU management were screened from the stored data, by one of the researchers, and recorded on an excel sheet.

As per the case file of the patients, the diagnosis was made on the basis of history of exposure or contact and characteristic clinical picture. The diagnosis was not supported by measuring plasma or red blood cell anticholinesterase levels since these are not measured in our hospital. Toxicology lab screening was also not available at our institution.

In A and E, treatment was started as per the protocol for managing OP poisoning patients in our hospital. Clothes were removed and body was washed with soap water. Nasogastric tube was passed to decompress the stomach. Initial management of all patients with pralidoxime (PAM) and atropine were done as per the recommended dosage schedule. A starting loading dose of 3-10 mg of atropine was administered depending upon the severity. Once atropinised maintenance dose of 1-3 mg was given every hourly. The target end point of atropinisation was (1) Chest clear on auscultation with no wheeze (2) heart rate >80/min (3) pupil no longer pin point (4) dry axilla, and (5) systolic blood pressure >80 mmHg.

PAM was administered with a bolus dose of 2 g over a period of 4 hrs followed by 1-g IV infusion every 6-8 hours. Patients with Glasgow Coma Scale (GCS) <8, hypoxia, convulsions and unstable haemodynamics were intubated and mechanically ventilated. All the patients were then shifted to the ICU for further management.

In ICU the patients were managed as per the ICU protocols in our hospital. Administration of PAM and atropine was continued till the target end point was reached. Atropine was subsequently replaced by glycopyrolate, the dose of which was progressively decreased. PAM in the dose of 1 g IV infusion over a period of 6-8 hrs was continued until fasciculations disappeared or skeletal muscle weakness was relieved. Sedation and analgesia was obtained administering midazolam and fentanyl as intravenous bolus followed by infusion.

Patients requiring ventilatory support were initially put on assist pressure control mode and subsequently weaned off by synchronised intermittent mandatory ventilation (SIMV), pressure support (PS) ventilation. Positive end expiratory pressure (PEEP) was added as per the lung characteristics. Patients were extubated using rapid shallow breathing index (RSBI) or spontaneous breathing trial (SBT) as the extubation criteria.

Continuous monitoring and daily investigations were done as per the requirement for the patient management. Severity of poisoning was graded using modified Dreisbach's classification [Appendix 1].[7]

The various parameters recorded against each patient on the excel sheet were: demographic data,

Appendix 1: Dreisbach's classification showing severity of poisoning

Grade	Symptoms
Mild	Nausea, vomiting, diarrhoea, sweating
Moderate	Lacrimation, salivation, miosis, fasciculation
Severe	Incontinence, apnoeic spells, ARDS, areflexia seizures, coma

ARDS – Acute respiratory distress syndrome

month of the year, mode of poisoning, reason for ingestion/poisoning, time from ingestion of OP to administration of PAM (Lag time), number of patients sustaining cardiac arrest in A and E, duration of mechanical ventilation and mortality.

The Chi-square test was used to analyse the non-parametric data. Pearson correlation test was used to prove the significance between the lag time of starting of PAM and duration of mechanical ventilation. Multivariate binary logistic regression analysis was done with death as the dependent variable and age, lag time to PAM therapy, severity of poisoning and duration of ventilation as the covariates. Data are presented as mean ± standard deviation. $P < 0.05$ was considered statistically significant.

RESULTS

During the study period, 117 patients reported to the A and E with history of acute OP poisoning, of whom 8 patients were brought dead to the A and E, 16 patients were referred to other hospitals due to non-availability of beds and ventilators in the ICU and 93 patients were admitted to the ICU. However, records of seven patients were incomplete and hence were excluded from the study.

Out of 86 patients, 52 (60.46%) were males of whom 11 (21.2%) patients died and 34 (39.54%) were females of whom 8 (14.7%) patients died [Table 1].

The age of the patients ranged from 17 years to 53 years. 69/86 (80.2%) of patients aged less than 40 years out of which 10 (14.5%) died. 17/86 (19.8%) of the patients aged ≥40 years, out of which 6 (35.5%) died. The mean (SD) age of the patients was 30.51 ± 10.78 years [Table 1].

The mode of poisoning was suicidal in 79 (91.8%) patients of whom 12 (15.2%) patients expired and 7 (8.2%) was accidental of whom 4 (57.1%) expired [Table 1].

The severity of poisoning was graded according to Dreisbach's classification [Appendix 1]. Accordingly, out of 86 patients, 14 (16.3%) had mild, 30 (34.9%) had moderate and 42 (48.8%) had severe grade of poisoning. All 14 (100%) patients with mild poisoning survived, whereas 24 (80%) patients with moderate poisoning and 32 (76.2%) patients with severe poisoning survived [Table 2].

All (13, 100%) patients who had been administered PAM within 6 hrs of poisoning (Lag time) survived, whereas 34 (82.9%) patients with lag time between 6 and 12 hours and 23 (71.9%) patients with lag time 13-24 hrs survived [Table 2].

All the patients required endotracheal intubation and mechanical ventilation and the mean ± SD duration of ventilation was 4.83 ± 3.41 days, range 1-17 days [Table 2]. The recorded time interval between OP poisoning and starting of PAM (lag time) was significantly higher in those who died. Majority (69/86, 80.23%) of the patients required mechanical ventilation for a period of 2 to 7 days. Mortality was 33.3% (3/9) in patients who required mechanical ventilation for more than 7 days, 7.2% (5/69) in those who received mechanical ventilation for 2 to 7 days and 100% (8/8) in those who received mechanical ventilation for <2 days [Table 2].

Highest incidence of poisoning was recorded (30/86, 34.88%) during summer (March–May) followed by rainy season (June–Aug) and least during spring (Sept–Nov).

Table 1: Demographic data

	Total no. of patients	Gender		Age (years)		Cause of poisoning	
		Male	Female	<40	≥40	Suicidal	Accidental
Survived (%)	70 (81.4)	41 (78.8)	29 (85.3)	59 (85.5)	11 (64.7)	67 (84.8)	3 (42.9)
Expired (%)	16 (18.6)	11 (21.2)	5 (14.7)	10 (14.5)	6 (35.3)	12 (15.2)	4 (57.1)
Total	86	52	34	69	17	79	7

Table 2: Severity of poisoning, lag time and duration of mechanical ventilation. data are *n* (%)

	Severity			Lag time (hours)			Duration of MV (days)		
	Mild	Moderate	Severe	<6	6-12	13-24	<2	2-7	>7
Survived (%)	14 (100)	24 (80)	32 (76.2)	13 (100)	34 (82.9)	23 (71.9)	0	64 (92.8)	6 (66.7)
Expired (%)	0	6 (20)	10 (23.8)	0	7 (17.1)	9 (28.1)	8 (100)	5 (7.2)	3 (33.3)
Total	14 (16.3%)	30 (34.9%)	42 (48.8%)	13	41	32	8	69	9

MV – Mechanical ventilation

Duration of mechanical ventilation had a positive and a linear correlation with the lag time for starting PAM ($P < 0.0001$, r = 0.76, y = 6.0078x + 25.93). The duration of mechanical ventilation increased with the increase in lag time. It was statistically significant [Table 3]. Total dose of PAM also had a positive, statistically significant correlation with the lag time ($P < 0.0001$, r = 0.88, y = 1.8204x + 3.3612) [Table 4]. Cardiac arrest was recorded in 14 patients out of whom 9 patients were successfully resuscitated and sent to the ICU. Return of Spontaneous Circulation (ROSC) was achieved in all the patients. However, 6 patients finally expired. 9/86 (10.47%) patients developed VAP, 1 patient suffered from pulmonary oedema and 6/86 (6.98%) developed ARF. The total mortality was 16 out of 86 patients (18.60%). Among these 16 patients, 6 patients had moderate and 10 patients had severe poisoning.

Multivariate binary logistic regression analysis was done with death as the dependent variable and age, lag time to PAM therapy, severity of poisoning and duration of ventilation as the covariates. Though the odds of death due to OP poisoning given presence of age, lag time, severity of poisoning and duration of mechanical ventilation were statistically insignificant, but the odds ratio (OR) more than 1 in all these cases show exposure associated with higher odds of outcome [Table 5].

DISCUSSION

This retrospective analysis revealed that OP poisoning was predominantly suicidal, affecting males, aged less than 40 years. Most of the patients received PAM within 6-12 hours of poisoning and required mechanical ventilation for 2-7 days. There was a positive and linear correlation between lag time and mechanical ventilation. The incidence of poisoning was highest during summers and the mortality was 29.1%.

In the present study, the incidence of poisoning was highest during the summer months (34.88%). This could be probably because during the summer months the agricultural income would be nil and hence there would be an enhanced risk in self-poisoning as one is unable to meet all the needs of one's dependents.

OP compounds are used worldwide in agriculture as well as in household gardens.[8] This easy availability of the compounds has resulted in a gradual increase in accidental and suicidal poisoning, mainly in developing countries.[9,10] OP poisoning due to self poisoning or suicidal poisoning accounts for at least 40-60% of all cases in some African countries.[8,10] In India, there is a lack of definitive data about the appropriate incidence of OP poisoning. According to Kumar *et al*.,[11] the effective number of cases is approximately up to 76,000 annually, much higher than the figure of National Crime Records Bureau. In our study, the incidence of suicidal poisoning is 91.86%, probably because it is cheap, easily available over the counter and used as a major pesticide in agricultural farming throughout India. This was in agreement with other studies[12-16] which showed deliberate self-poisoning varying from 68% to 96%. In the study by Aziza *et al*.,[13] 76.92% cases were suicidal and 23.07% were accidental. This incidence

Table 3: Correlation between lag period of starting of PAM and duration of mechanical ventilation

	Lag time	Duration of mechanical ventilation
Lag time		
Pearson correlation coefficient (*r*)	1	0.76
Significance (2 tailed)	-	0.0001*
N	86	86
Duration of mechanical ventilation		
Pearson correlation coefficient (*r*)	0.76	1
Significance (2 tailed)	0.0001*	-
N	86	86

* – Correlation is significant at the 0.01 level (2 tailed), *N*: Total number of organophosphorus poisoning victims, PAM – Pralidoxime

Table 4: Correlation between lag period of starting of PAM and total dose of PAM

	Lag time	Total dose of PAM
Lag time		
Pearson correlation coefficient (*r*)	1	0.88
Significance (2 tailed)	-	0.0001*
N	86	86
Total dose of PAM		
Pearson correlation coefficient (*r*)	0.88	1
Significance (2 tailed)	0.0001*	-
N	86	86

* – Correlation is significant at the 0.01 level (2 tailed), *N* – Total number of organophosphorus poisoning victims, PAM – Pralidoxime

Table 5: Multivariate binary logistic regression analysis with death as the dependent variable and age, lag time to PAM therapy, severity of poisoning and duration of ventilation as the covariates

Death	OR*	95% CI†	*P* value
Age	1.16	0.91-1.48	0.21
Lag time	1.07	0.86-1.33	0.55
Severity of poisoning	1.18	0.93-1.53	0.26
Duration of ventilation	1.05	0.80-1.38	0.73

* – Odd's ratio, † – Confidence interval

of suicidal poisoning is much higher than the recorded range of 10.3% to 43.8%.[17]

Diagnosis of OP poisoning depends mainly on history, characteristic clinical presentation and is supplemented by decreased levels of serum and erythrocyte cholinesterase levels. In this study, the cholinesterase levels were not assessed due to non-availability of the facility. However, as a principle, treatment of OP poisoning should be started immediately and must not await the results for serum cholinesterase levels.

Treatment of OP poisoning, apart from measures to decontaminate, is primarily aimed at reversing the effects of the compound by administration of atropine. Atropine is highly effective in antagonising the actions of organophosphates at muscarinic receptor sites and is administered to adults in doses of 2 mg every 5 to 10 minutes. PAM, compound, which regenerates and reactivates acetylcholinesterase from the OP-cholinesterase complex, is used as an antidote to treat OP poisoning. Although it works at nicotinic, muscarinic, and central nervous system receptors, its main therapeutic effect is predicted to be the recovery of neuromuscular transmission at nicotinic synapses. It should be administered as soon as possible to prevent ageing and promote adequate reactivation of red cell acetylcholinesterase. However, a beneficial response has been reported even after 24 hours of exposure.[11] Our observation was in accordance with this concept. All the patients with a lag time less than 6 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 doses of PAM and increased duration of mechanical ventilation. The high mortality [14/32 (43.75%)] in those who received PAM after 12 hours of poisoning was probably due to ageing of the receptors.[18]

Stomach decompression was carried out in all patients with nasogastric tubes; Li *et al*.,[19] doubt the efficacy of this method in their study. They state that despite widespread use of multiple gastric lavages for OP pesticide poisoning across Asia, there is currently no high-quality evidence to support its clinical effectiveness and there is a need for studies to identify in which patients and for what duration gastric lavage is able to remove significant quantities of poison. Large clinical trials will be required to address the effectiveness and safety of gastric lavage (either single or multiple) in acute OP pesticide poisoning.

In the present study, the incidence of poisoning was higher in males than in females. Similarly, in the study by Srinivas *et al*.,[12] the males outnumbered females (57% vs. 43%) with all types of pesticides including OP compounds. Similar trend was also observed by Safdar *et al*.,[20] and Aziza *et al*.[13] However, in some studies[14-16,21] OP poisoning was more common in females than males.

In the present study, the incidence was highest in patients aged less than 40 years. The people in this age group are described to be most ambitious, productive and responsible. Therefore, these are the people most vulnerable to various emotional conflicts that can 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 and 39 years.[22,23]

The duration of mechanical ventilation in our patients was 4.83 ± 3.41 days. These patients are prone to develop respiratory failure for many reasons, and multiple mechanisms including aspiration of gastric contents, excessive secretions, thoracic weakness, decreased respiratory drive, pneumonia and sepsis-complicating ARDS.[24,25] Acute respiratory failure (less than 24 h) is seen in 33% of patients with acute poisoning.[26] Intermediate syndrome is a complication which develops 1-4 days after acute OP poisoning, and is characterised by respiratory insufficiency, proximal muscle weakness and cranial nerve palsies. Most patients with intermediate syndrome develop respiratory failure, which requires mechanical ventilation.[27,28] With the support of mechanical ventilation, it usually resolves within 4-18 days with high survival ratio. Reported frequency of intermediate syndrome varies from 8% to 49%.[28-30]

The mortality following OP poisoning varies between 4% and 30%.[31] In a study by Safdar *et al*.,[20] 4% of patients who received mechanical ventilator support ultimately expired. In another study, mortality was 50% in patients requiring mechanical ventilation.[14] In contrast to these observations, Aziza *et al*.[13] reported 8% mortality in patients who received mechanical ventilation. In the present retrospective analysis, the overall mortality was 29.06% which was within the range of the previous studies. Interestingly, all the patients (100%) who were on ventilator for less than 2 days, expired. These patients probably had increased lag time or reported late to the A and E resulting in severe degree of poisoning leading to

severe respiratory failure. The mortality was also high in patients requiring mechanical ventilation for more than 7 days (33%). These patients probably developed lung complications due to prolonged mechanical ventilation. The high mortality in patients ventilated for 2-4 days is most probably due to the severity of poisoning. Nowadays high concentrations of OPCs are sold to save storage space. Along with these, delay in presentation from remote villages further deteriorates the clinical condition of the patient. In these cases patients, though on ventilator, keep worsening and die eventually.[18]

The average dose of PAM required per patient was 23.58 grams. The approximate daily dose of PAM is 12 g[17] to achieve an effective therapeutic blood level of 4 ng/ml.[18] Joshi *et al*.[32] stated in their study about the positive outcome associated with the use of higher dose of PAM. There is no single study to our knowledge describing the average dose of PAM per patient. A recent meta-analysis, prepared by South Asian Cochrane Network and Centre and Toxicology Special Interest Group, Department of Medicine. Christian Medical College, Vellore, failed to support the use of the WHO recommended regimen of PAM (30 mg/kg pralidoxime chloride bolus followed by 8 mg/kg/hr infusion). The financial impact of such therapy, particularly in rural India has been raised as an issue in absence of definite beneficial effect of oximes in OP poisoning. They concluded that further RCTs are required to examine other strategies and regimens.[33]

There was no statistically significant association with death and age, lag time, severity of poisoning and duration of ventilation independently. This signifies that death due to OP poisoning is not dependent on a single predictor as age, lag time, etc., Death in OP poisoning is rather due to overlapping of all the factors. Higher lag time, more severe poisoning, greater duration of mechanical ventilation-all simultaneously contribute to death.[34]

CONCLUSION

Easy availability and low cost has made OP insecticide as an agent of choice for self-poisoning. It is of great concern since it affects the most productive age group of the society. The mortality is very high since the victims are predominantly from rural India where poisoning is very severe due to delay in the access to medical management. Mortality is directly proportionate to the severity of poisoning, delay in starting PAM and duration of mechanical ventilation. Though each predictor (age, lag time, severity of poisoning and duration of ventilation) is associated with mortality, death due to OP poisoning results from overlapping contribution of these factors. No single factor is independently responsible for mortality in these patients.

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Announcement

Conference Calendar Details

Name of the conference: 62nd Annual National Conference of the Indian Society of Anaesthesiologists, ISACON 2014
Date: 26th to 29th December 2014
Venue: Velammal Medical College "Velammal Village", Madurai – Tuticorin, Ring Road, Annupanadi, Madurai – 625009, Tamil Nadu, India
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