ORIGINAL RESEARCH PAPER

General Medicine

A STUDY ON THE PREDICTORS OF MORBIDITY AND MORTALITY IN ORGANOPHOSPHORUS POISONING

KEY WORDS:

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ABSTRACT

Background: Poisoning from organophosphorus (OP) pesticides can occur as a result of occupational, accidental, or purposive exposure. Cholinergic syndromes, central nervous system (CNS) disorders, and cardiovascular disorders are some of the clinical manifestations. The most common causes of death are cardiovascular and respiratory failure. Aim: To assess various parameters that can predict the outcome of patients suffering from OP poisoning. **Materials and Methods:** A prospective study was conducted at Government general hospital vijayawada Over a one-year period of time. The clinical history of OP compound exposure and low blood levels of pseudo cholinesterase were used to make the diagnosis of OP poisoning. **Results:** The current study enrolled 133 patients, 98.5% of whom were suicidal and only 1.5% were exposed accidentally. The majority of cases involved young males, with an F/M ratio of 1:3.2. Younger people and patients who required prolonged ventilator support had a higher mortality rate. The death rate was proportional to the amount of poison consumed. Lag time, organ failure (Acute Renal Failure), and plasma pseudocholinesterase levels are all factors to consider. Acute complications were frequently observed and treated. There was no clear link found between liver dysfunction, electrolyte imbalance, and clinical outcome. **Conclusion:** According to this case study, mortality is directly proportional to the lag time, amount of OP substances consumed, and clinical severity. Acute renal failure, severity, pseudocholinesterase levels, and duration of ventilatory support are all factors to consider. This study emphasises the importance of prompt diagnosis and the initiation of early and effective treatment, which may result in fewer complications and lower mortality rates.

INTRODUCTION

Organophosphate toxicity is caused by excessive cholinergic stimulation via acetylcholinesterase inhibition. Serum cholinesterase levels are reduced after organophosphorus (OP) poisoning, as reported by prior studies. The measurement of cholinesterase activity is used to confirm organophosphate poisoning. Although both RBC and better (CNS). As a result, acetylcholinesterase is a more useful marker for organophosphate poisoning[2]. The rapid accumulation of acetylcholine in CNS and peripheral synaptic junctions causes a cholinergic crisis with a variety of muscarinic, nicotinic, and central effects[3]. The gastric mucosa is permeable to organophosphates and is a common route of absorption in suicidal patients. The liver is the organ where organophosphate compounds are activated and detoxified, but they are primarily eliminated through the kidneys[3].

Muscarinic and nicotinic clinical manifestations include bradycardia, hypotension (Muscarinic), tachycardia (nicotinic), increased salivation/ lacrimation, excessive sweating, nausea, vomiting, diarrhoea, pain abdomen, faecal and urinary incontinence. Anxiety, restlessness, convulsion, miosis, insomnia, coma, cheyne-stokes breathing, respiratory and cardiovascular failure are all CNS manifestations [5]. Intermediate syndrome, also known as type II paralysis, typically occurs 24-96 hours after an acute cholinergic crisis. The prevalence of Intermediate syndrome ranges from 8 to 50%[6]. Chronic organophosphate poisoning can result in organophosphate-induced delayed neuropathy, which is most common in agricultural workers[7].

To minimise OP compound absorption, the initial management of acute OP poisoning includes cardiopulmonary stabilisation, decontamination (removal of clothing as a possible source of continued exposure in occupational intoxication), irrigation of skin and eyes, as well as gastric lavage and activated charcoal[8]. Atropine, a central and peripheral muscarinic receptor antagonist, and pralidoxime chloride, which reactivates inhibited acetyl cholinesterase, are the mainstays of treatment [9]. In recent years, new adjunct therapy and low-cost medications like sodium bicarbonate, magnesium sulphate, and antioxidants have been considered for the treatment of OP poisoning[10]. Death usually occurs as a result of cardiovascular and respiratory failure, respiratory muscle paralysis, and obstruction caused by bronchospasm and bronchial secretions[11].

MATERIALS AND METHODS

From July 2021 to June 2022, we enrolled 133 patients in a prospective study at Government general hospital, vijayawada. The study was approved by the institute's ethical committee. This study included all OP poisoning patients. We did, however, exclude patients whose OP poisoning was suspected. A detailed history of the poisoning was obtained from all of the patients' relatives. The patients were subjected to a thorough clinical examination. The diagnosis of OP poisoning was based on clinical characteristics, a history of exposure to a known OP compound, and low serum pseudocholinesterase levels. Patients were treated with respiratory support, atropine, and pralidoxime according to the standard protocol for organophosphate poisoning. Before being discharged from the hospital, all cases that recovered received psychiatric consultation.

Complete blood hemogram, urea, creatinine, arterial blood gas values, X-ray chest, and serum pseudo-cholinesterase level were all performed at the outset. A structured proforma

was used to retrieve data from the files. Gender, age, amount of organophosphate consumed, mode of exposure, time lag in starting treatment, duration of ventilator support and hospital stay, acute complications, and patient outcome were all studied variables.

Types of Organophosphorus compounds

In most cases, we could identify the type of OP compound because patient attendants would bring the bottle of OP compound that the patient had consumed to our Emergency department. The following are the various types of OP substances consumed in the current study.

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

The DGKC method was used to estimate the serum pseudocholinesterase level in all patients at the time of admission . In the current study, the laboratory reference range for pseudocholinesterase was Female = 3930-10800/l, Male = 4620-11500 u/l. According to Kumar et al., the severity of poisoning was defined based on serum pseudo cholinesterase levels[12].

Pseudo cholinesterase level 20-50% of normal or >1,401-3,500 IU/L in mild poisoning.

Pseudo cholinesterase level 10-20% of normal or 701-1, 400 IU/L in moderate poisoning. Pseudo cholinesterase level is 10% of normal, or 700 IU/L, in severe poisoning.

STATISTICAL STUDY

Data were presented in either mean ± standard deviation (SD) or percentage form. P< 0.05 was considered significant, and all statistical analyses were carried out using SPSS version 12.0. For categorical data, Fisher's exact test was used.

RESULTS

During the study period, 133 cases of OP poisoning were admitted [Table 1]. Only two patients (1.5%) had dermal/inhaled exposure while spraying pesticides in rice fields, while one thirty one (98.5%) patients ingested the compound [Table 1]. There were 102 (76.7%) males and 31 (23.3%) females. The majority of the cases involved young people, with 80% (40years) being predominantly males [Table 1]. There was a wide range of ages, ranging from 13 to 68 years, with a mean age of 31.5 years [Table 2].

Table 1: Showing number and percentage of patients

		SURVIVED	EXPIRED	TOTAL
GENDER	MALE	73(54.8%)	29(21.8%)	102
	FEMALE	18(13.5%)	13(9.7%)	31
AGE	<40 years	79(59.3%)	27(20.3%)	106
	>40 years	12(9.02%)	15(11.2%)	27
MODE OF POISONING	SUICIDAL	89(66.9%)	42(31.6%)	131
	ACCIDENTAL	2(1.5%)	0 (0)	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 BETWEEN 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

After gastric lavage, 48 patients (36.1%) out of 133 were stable. They were kept under observation for three days before being discharged. As shown in [Table 3], the clinical presentation of acute poisoning varied. Miosis, on the other hand, was the most consistent feature (93.2%). Eighteen (13.5%) of the patients experienced episodic convulsions. 13.5% of patients had transient elevations in liver enzymes [Table 3]. However, there was no significant increase in morbidity or mortality in patients with hypokalemia or abnormal liver function tests [P > 0.05, Table 4]. Patients who developed single or multiple organ failure had a higher mortality rate [P<0.0001, statistically significant Table 4].

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 consciouness/altered sensorium	45	33.8
Severe bradycardia at the time of presentation	33	24.8
Lacrimation/salivaton	115	86.4
Urinary/fecal incontinence	78	58.6
Miosis	124	93.2
Bronchospasm	104	78.2
Hypotension	15	11.3
Seizures	18	13.5
LABARATORY PICTURE		
Deranged RFT - S.CREATININE >1.4mg/dl	21	15.03
Deranged LFT	18	13.5
Hypokalaemia	20	15
COMPLICATIONS		
Single organ failure/respiratory failure	45	33.8
Multi organ failure	39	29.3
REQUIRED VENTILATORY SUPPORT	53	39.8

Fifty-three patients required ventilatory support, with only 11 surviving [Table 5]. Patients were ventilated for a minimum of one day and a maximum of 22 days, with a mean of 6.85± 4.32 days [Table 2]. Patients who required ventilator support for more than 7 days died at a higher rate [P< 0.05 statistically significant, Table 5]. The amount of OP compound consumed ranges from 10 to 200 ml, with a mean of 77.5 ml [Table 2]. The rate of death was proportional to the amount of poison consumed [P< 0.00003 statistically significant, Table 5].

Table 4: showing outcome of patients with organ failure , liver injury and elecrtolyte imbalance

		SURVIVED	EXPIRED	TOTAL NO.OF PATIENTS	P VALUE
HYPOKALEMIA	<2.5	8	2	10	P=1.0
	2.5 - 3.0	6	1	7	
	3.0 - 3.5	3	0	3	

ELEVATE LIVER ENZYMES	36 - 100	11	0	11	P=0.115
	100 - 200	2	1	3	
	> 200	5	2	7	
SINGLE ORGAN FAILURE		32	13	45	P=0.001

Table 5 :showing outcome in patients with acute renal failure , severity of poisioning , lagtime and duration of mechanical ventilation

	MINIMUM	MAXIMUM	MEDIAN/ MEAN	SD
AGE	13 YEARS	68 years	28/31.5	12.98
TIME BETWEEN 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

The lag time for treatment initiation ranged from 1.02 hours to 9.57 hours, with a mean of 4.65± 2.4 [Table 2]. Patients with lag times greater than 6.5 hours had a higher mortality rate [P< 0.05, Table 5]. Twenty-one (15.7%) of the 133 patients had abnormal renal function tests (defined as serum creatinine >1.4).

Renal function derangement was reversible in the majority of cases, and renal function tests improved within a week. Patients with serum creatinine levels greater than 3.5 mg/dl, on the other hand, had a higher risk of death [P <0.05, Table 5]. One of three patients with irreversible renal failure had a serum creatinine level of 10.2 mg/dl on cases of acute renal failure died within a week.

The overall mortality rate was 33.3% (42 patients out of 126), with seven cases (5.2%) discharged against medical advice [Table 1]. On follow-up, delayed complications such as mild sensory loss in the lower limbs or limb weakness were uncommon in our patients.

DISCUSSION

Poisoning from OP pesticides is common in developing countries[11] . India has the highest incidence[13] . Organophosphate poisoning, both suicidal and non-suicidal, is a major problem in rural India, with a rapidly increasing incidence rate[14] .The female to male ratio in our study is 1:3.2. Males were more likely than females to be poisoned in the current study (76.6% vs. 23.3%). Safdar et al[15], and Aziza et al[16] observed a similar trend.

However, Ather et al female to male ratio is 1:1, and Tall et al female to male ratio is 1:1.8, which is quite different from the current study[17,18] . The ages ranged from 13 to 68 years, with a mean of 31.5 years. Hayden et al[19] on the other hand, reported an age range of 13-47 years with a mean age of 23 years.

The incidence of OP poisoning was highest in patients under the age of 40 in the current study. The majority of the cases (80%) were young people, predominantly males between the ages of 13 and 40, which is comparable to other studies such as Khan MN et al[20] , in which the majority of patients were between the ages of 15 and 35. This age group is described as being the most ambitious and vulnerable to various emotional conflicts that may arise during this stage of life. Our findings were consistent with previous research, which found that people aged 21 to 39 years had the highest rate of OP poisoning[21] .In our study, the rate of suicidal poisoning was 98.6%, owing to the fact that it is inexpensive, widely available, and widely used as a pesticide in agricultural farming throughout India. This was consistent with other studies[16,22] , which found a range of 68-96% deliberate self-poisoning. According to Aziza et al [16] ,76.92% of cases were suicidal, while 23.07% were accidental. The current study discovered that the majority of cases of OP poisoning had lower serum pseudo cholinesterase levels on the day of presentation. It was also discovered that moderate to severe cases with pseudo cholinesterase 1400 U/L required more intubation and oxygen. Furthermore, cases with pseudo cholinesterase 700 U/L had the highest mortality (P< 0.05).

Mehta et al[23] observed lower pseudo cholinesterase activity in more than 70% of cases at presentation, which is consistent with our findings. Apart from clinical indicators, Goswamy et al[24] concluded that low serum cholinesterase levels had the greatest predictive value for ventilation in OP poisoning. However, Nouira et al[25] found no statistically significant difference in mean serum cholinesterase levels between mechanically ventilated patients and those who did not require ventilator support.

In OP poisoning, hypokalemia , hyperglycemia, acute renal failure, and transient elevations of liver enzymes can occur[26] Hypokalemia and transient elevations of liver enzymes were found in 15.03% and 13.5% of cases, respectively, in our study. According to Wang WZ et al[27] liver injury was observed in 9.8% and 5.17% of cases and controls, respectively, in OP poisoning, and mortality was higher in cases than controls (22.5% vs 6.32%). However, we did not find a clear relationship between serum potassium and liver enzyme derangement and the severity of OP poisoning and clinical outcome (P > 0.05).

Acute renal failure was reported in 15.03% of patients after OP poisoning. Out of twenty-one cases, eighteen had transient reversible acute renal failure and three (2.2%) had irreversible renal failure. Patients with acute irreversible renal failure (serum creatinine >3.5 mg/dl) had a higher mortality rate. Arefi Met al[28] discovered that 16.7% of OP poisoning patients had renal failure in their study, which is similar to our findings. Similarly, S panda et al[29] discovered differences in renal function between survivors and non-survivors, implying the importance of renal function in predicting mortality. The transient renal injury could be caused by an organophosphate's direct action, causing tubular cell necrosis, or by a secondary mechanism that followed the cholinergic crisis, causing hypovolemic shock and rhabdomyolysis.

In this study, the most common signs were miosis (93.2%), increased salivation (86.4%), anxiety and restlessness (82.7%), bronchospasm (78.2%), and incontinence (58.6%). Other common clinical features observed in this study are listed in Table 3 with percentages, which are also comparable to other studies[30,31] In the current study, 33.8% of the cases presented with altered sensorium or loss of consciousness, which was followed by deep coma in the majority of cases. According to Sequeira et al[32], the prevalence of deep coma is 21%. Acute complications seen in this study included episodic convulsions (13.5%), severe bradycardia (24.8%), and hypotension (11.3%). There were no serious ventricular arrhythmias observed. Acute complications [33] included bradycardia in 29 (93.5%), mental status change in 10 (32.2%), low oxygen saturation in 21 (67.8%) and 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 rate in patients ventilated for two days and between two and seven days is most likely due to the severity of the poisoning[8]. The mortality rate after OP poisoning ranges between 4 and 30%[34]. According to Safdar et al[15] 4% of patients who received mechanical ventilation eventually died. In another study, 50% of patients requiring mechanical ventilation died[22] In contrast to these findings, Aziza et al [16] reported 8% mortality in mechanically ventilated patients.

In our study, it was also discovered that the majority of patients who died had a delay (maximum 9.57 hours) between the consumption of OP substance and the initiation of treatment, which is supported by a study done by Suleman MI et al[35]. The majority of patients with lag times less than 6.5 hours recovered and survived, whereas patient recovery and survival decreased as lag time increased. Furthermore, patients with longer lag times required more mechanical ventilation[8].

Mortality was higher in those who consumed a large amount of OP substances (50-100 ml) and highest in those who consumed more than 100 ml [P< 0.05, Table 5]. Morbidity and mortality were proportionately higher in the majority of patients who developed single/multi-organ failure and required prolonged ventilatory support in the current study (P < 0.05, Table 4). The overall mortality rate in our study was 33.3%, which was higher than in studies by Numidasa UA et al[36], and Pandyal BP et al [37].However, according to Yamashita et al [34] , the frequency of mortality due to organophosphates ranged between 4% and 30%, and 5.5% in a study by Malik et al[33].Higher mortality rates may be due to late arrival, lack of treatment at the periphery prior to arrival at the hospital, poverty and illiteracy, uncertainty of the mortality rate of OP poisoning, and a lack of intensive care unit (ICU) facilities[36].

CONCLUSION

OP poisoning is common in developing countries and is a major concern because it affects the most productive age group in society. Because of its ease of availability and low cost, OP poisoning has become a common agent for self-poisoning, particularly in rural India.Acute complications are more common in OP poisonings than chronic complications. The lag time in starting treatment and/or the amount of OP substances consumed, clinical severity (single/multi organ failure), and duration of ventilatory support all have a direct relationship with mortality and morbidity. Patients who develop acute complications such as severe bradycardia and severe acute renal failure have a higher mortality rate. Although each predictor (age, lag time, poisoning severity, amount of organophosphate consumed, organ failure, acute kidney injury, and duration of ventilation) is associated with mortality, death from organophosphate poisoning is caused by the overlapping contribution of these factors.For the death of these individuals, no single cause acts alone. Since patients who receive early and effective therapy typically do better, have fewer problems, and have a lower morbidity and death rate, the significance of rapid diagnosis and early and successful treatment should not be underestimated. In addition to reducing the frequency of acute or chronic problems, good supportive and ICU treatment will also lower the death rate in these instances.

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