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Prognostic Value of Serum Cholinesterase in Organophosphate Poisoning

Medical Science

KEYWORDS : Acetyl cholinesterase, Organophosphorus compounds, Pseudocholinesterase.

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ABSTRACT

Background:Organophosphorous (OP) poisoning is one of the most common poisonings seen in India. OP compounds act through inhibition of enzyme acetylcholinesterase and estimation of pseudocholinesterase (PCE) activity strengthens the diagnosis in clinically uncertain cases of OP poisoning. OBJECTIVE: To determine whether serum cholinesterase level has a prognostic value in organophosphorus poisoning. DESIGN: Cohort (prospective) prognosis study. SETTING: Tertiary hospital in Mumbai .Materials and Methods: A prospective study conducted in Department of Medicine over period of 1 year. Diagnosis of OP poisoning was based on clinical history of exposure to OP compound and low serum pseudocholinesterase levels. MEASUREMENTS: Serum cholinesterase level was measured in all patients at the time of hospital admission. Patients of suspected OP poisoning of age >18 years admitted to emergency unit at a tertiary healthcare centre were enrolled. Serum PCE level was estimated at the time of admission in all patients and severity of OP poisoning was assessed according to PCE level. Results: The age of the patients varied from 20 to 70 yrs with 65% of them between 21-30 age groups. Majority of cases were young male, with F/M ratio 1:3.S. PCE levels were reduced in all the cases and the mean level was 3,154.16 ± 2,562.40 IU/L. Mortality rates were higher in younger people and in patients associated with lower pseudocholinesterase level (P<0.001). Conclusion: This study highlights the importance of rapid diagnosis, and initiation of early and effective treatment, which may result in less number of complications and also decrease the mortality rates.

INTRODUCTION

It is estimated by World Health Organization (WHO) that around 0.3 million people die every year due to various poisonings.[1]OP poisoning is one of the most common poisoning seen in India. OP acts by inhibiting the enzyme cholinesterase, results in accumulation of acetylcholine at synapses and myoneural junction leading to cholinergic overactivity[4].

Mortality ranges from 4-30% in Indian studies. Respiratory Failure is most common complication of OP poisoning leading to death. Early recognition and prompt ventilator support may improve survival .The commonest types of insecticide/pesticides substance used for poisoning are organophosphates, chlorinated hydrocarbons, aluminium phosphide, carbamates, and pyrethroids. The anticholinesterase effects can be evidenced biochemically by suppressed levels of serum pseudocholinesterase (PCE) and red cell cholinesterases. 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. The mainstay of treatment involves atropine — A central and peripheral muscarinic receptor antagonist and pralidoxime chloride, which reactivates inhibited acetyl cholinesterase.

In the present study, we made an effort to evaluate the prognostic significance of estimating serum PCE level on outcome in patients of OP poisoning.

MATERIALS AND METHODS

A prospective study conducted in Department of Medicine over period of 2 year(2006-8). Diagnosis of OP poisoning was based on clinical history of exposure to OP compound and low serum pseudocholinesterase levels. Thirty consecutive patients admitted for acute organophosphate poisoning were enrolled . All patients were evaluated clinically by history and examination. Treatment was commenced as soon as the patients were attended. Serum PCE level was estimated at the time of admission in all the patients by spectrophotometry method. The laboratory reference range for PCE used in the present study was 7,000-19,000 IU/L. 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 PCE levels, the severity of poisoning was defined as per Kumar *et al*.

Latent: PCE level >50% of normal or >3,500 IU/L

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

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

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

Psychiatric counselling was done in all the patients. For clinical outcome, the total duration of hospital stay or death were considered. Complete recovery or death was used as the end point

Statistical analysis

Data were presented either as mean ± standard deviation (SD) or as percentage and median. 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

The age of the patients varied from 20 to 70 yrs with 65% of them between 21-30 age groups. Majority of cases were young male, with F/M ratio 1:3._PCE levels were reduced in all the cases and the mean level was 3,154.16 ± 2,562.40 IU/L[Table 2]. Mortality rates were higher in younger people and in patients associated with lower pseudocholinesterase level (P<0.001). Majority of patients (75%) reached to

medical facility within 6 h of exposure. The amount of OP compound consumed ranges from 10 ml to maximum 200 ml with mean 77.5ml. The mortality rate was directly proportional to the amount of poison consumed [$P < 0.00003$ statistically significant].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 1]. However, the most consistent feature was miosis (93.2%). There was no mortality in patients with serum PCE levels above 4,000 IU/L.

DISCUSSION

The present study found that the level of PCE on the day of presentation was reduced in all cases of OP poisoning.. Although the average dose of atropine used in study was higher in patients presenting with serum PCE levels <700 IU/L. 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 ($P < 0.05$)..Hypokalemia, hyperglycemia, acute renal failure, transient elevation of liver enzymes can occur in OP poisoning. In our study, hypokalemia and transient elevation of liver enzymes were found in 15.03% and 13.5 % of cases respectively Acute renal failure was reported following exposure to OP poisoning in 15.03% of patients had transient reversible acute renal failure

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%.%. Acute complications seen in this study were episodic convulsions in (13.5%) patients, severe bradycardia (24.8%), hypotension (11.3%) patients. 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, serum cholinesterase at presentation appeared useful to assess the severity of poisoning, particularly in terms of need for ventilator and prolonged duration of hospital stay. Still, we would like to conclude that the serum PCE is reduced in OP poisoning and serum PCE level should be estimated routinely in all cases of OP poisoning because it has prognostic significance.

Conclusion: A relative relationship between PChE level and clinical manifestations and outcomes was found. These findings can assist health professionals to better evaluate patient's prognosis and improve their treatment plan.

Table 1. Assessment of Severity of Acute Organ phosphorous Compound Poisoning

	SEVERITY		
	Mild	Moderate	Severe
Clinical Manifestations	Fatigue, Headache, Paresthesia, Nausea and Vomiting, Diaphoresis, Salivation, Abdominal pain, Diarrhea, Able to ambulate	Symptoms of mild poisoning + Miosis, General weakness, Dysarthria, Fasciculation, Unable to ambulate	Generalized Fasciculation, Marked miosis (Absent pupillary reaction), Flaccid Paralysis, Pulmonary crepitation, Respiratory distress, Cyanosis, Unconsciousness
Plasma Cholinesterase level	< 10 %	10-20 %	< 50 %

Table 2 Grade of poisoning based on pseudocholinesterase level

Grade of Poisoning	Cholinesterase activity
Normal	>50%
Mild	20-50%
Moderate	10-20%
Severe	<10%

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