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A Study on Association of Blood Sugar with Pancreatic Enzymes in Opc Poisoning

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ABSTRACT

Introduction: Patients with organophosphorous compound (OPC) poisoning presents with cholinergic signs largely because of overstimulation of muscarinic and nicotinic receptors. The effect of this on the pancreas has not yet been fully studied
Aim: Aim of this study is to know the incidence of hyperglycemia in OPC poisoning and to find the association of hyperglycemia and elevated pancreatic enzyme level.
Methods: Cross sectional study was conducted in Tirunelveli medical college, in 100 patients who consumed OPC, excluding alcoholic, diabetic, patients who underwent pancreatic procedures. In this study serum amylase is compared with variables such as age, sex, vitals, compound, amount, blood sugar, WBC, PCV, lipase.
Results: Among 100 patients studied, serum amylase was elevated in 6 patients. Serum lipase was elevated in 6 patients. Blood sugar was elevated in 10 patients. Among patients with hyperamylasemia only 2% have elevated blood sugar. So, correlations between blood sugar and serum amylase still have to be studied. Hyperglycemia in OPC is due to initial hypoglycemia or due to pancreatic insufficiency. In our study their relation is not statistically significant.
Conclusion: Though rare, pancreatitis can be considered as a complication of organophosphorus compound poisoning. There is no statistically significant evidence of hyperglycemia as a complication in our study

KEYWORDS: OPC, pancreatitis, amylase, lipase, blood sugar

Introduction:
OPC is most popular and widely used insecticide in India. It is consumed as eithersuicidal or unintentional as in case of occupational exposure. These compounds are availableas dusts, granules, or liquids. Toxicity profile varies for different compounds. Also the toxo kinetics varies depends on the route of administration such as transdermal, transconjunctival, inhalational through mucosa of respiratory tract, gastrointestinal tract[1]. Commonly consumed organophosphorus compound in this part of state are monocrotophos, profenophos, and dimethoate. Each of these compounds have different toxicity index so that their lethal dosevaries[2]. Each of these compound known to produce complications other than usual features suggestive of cholinergic action on muscarinic and nicotinic receptors. Usually thesepatients admitted in intensive medical care unit with neurological and respiratory symptoms[3]. Organophosphate poisoning also causes predictable biochemical effects as the autonomic nervous system, which is modified by organophosphate, plays an important role in the regulation of insulin secretion[4]. Hyperamylasemia is frequently seen in organophosphate poisoning due to cholinergic stimulation of pancreas. It is hyperlipasemia which leads to the suspicion of acute pancreatitis[5]. Pancreatitis is a known; but a rare complication, often goes unrecognized. The clinical signs of such an event could be masked by the overwhelming clinical manifestations of the organophosphate poisoning[4]. The occurrence of severe circulatory collapse present in many of the patients who consume a significantly large dose of organophosphate could be the culprit for acute pancreatitis, which is often mild and self limiting. Local effect of the organophosphate may also be a possibility. So, high suspicious eye is essential to identify these patients presenting with unexplained hemodynamic instability. Metabolic complications such as hyperglycemia, diabetic Ketoacidosis have to bekept in mind.Acute pancreatitis often correlates with hyperglycemia in patients[6]. There are certain scales to assess the need for mechanical ventilation, dose ofatropine called Peradeniya scale which is based on features such as, miosis, bradycardiarespiration, fasciculation and level of consciousness. In this study we correlate theassociation of blood sugar with pancreatic enzyme levels.

Aim:
To study the incidence of hyperglycemia in OPC poisoning and to study the prevalence of pancreatitis in OPC poisoning and to find the association of hyperglycemia and elevated pancreatic enzyme level

Materials and Method:
Cross sectional study was done in Department of Medicine, Tirunelveli Medical College Hospital. Institutional Ethics committee approval and Informed consent from the patients were obtained. Patients admitted in the IMCU with consumption of organophosphorus compound were screened. Patients with diabetes mellitus, chronic pancreatitis, history of cholelithiasis, history of pancreatic procedures, alcoholics, dyslipidemia and drug induced pancreatitis were excluded from the study. Patient's complete demographic details, history of consumption of OPC poisoning, and clinicalfeatures suggestive of OPC was collected and identification of container was done. Basic investigations such as total leucocyte count, hematocrit and random blood sugar, were done. Urine analysis was done. Pancreatic enzymes such as amylase and lipase were taken into account. Radio diagnostic studies of abdomen were done.

Results
100% patients were selected for this study; consumption of OPC is more in middle aged people 31 to 40 years. OPC poisoning is more common among male 78%.

Table 1 Age distribution of Study Patients

Age group	Number of Patients
< 30	32
31 to 40	41
41 to 50	19
> 51	8
Total	100

Table 2 Random blood sugar level in study patients

RBS	Number of Patients
<200 mg/dl	90
>200mg/dl	10

About 10% of our study subjects developed hyperglycemia as evidenced by a persistent RBS level > 200 mg% associated with glycosuria. (Table 2)

Table 3 Pancreatic enzyme S. Amylase level in study patients

S.Amylase	Number of Patients	P Value
< 190 U/L	94	<0.0001
> 190 U/L	6	

The incidence of hyperamylasemia is equally distributed between various age groups in our study population and was found only in those patients who consumed more than 5 grams of poison.Elevation of pancreatic enzymes is associated with mild decrease in systolic and diastolic blood pressure. Patients who had an elevated pancreatic enzyme level, also had a higher mean pulse rate and respiratory rate compared to others. Patients with hyperamylasemia had a mean oxygen saturation of 90.83 in comparison to others who had a mean value of 95.22. when 5 out of 6 patients with elevated pancreatic enzymes needed ventilator support, 22 out of 94 patients in the remaining were connected to mechanical ventilators. 52 % of the patients who consumed organophosphate had leucocytosis; though it didn't correlate with pancreatic enzymes

Table 4 Cross tabulation of S. Lipase with S. Amylase

S.Lipase	Amylase < 190 U/L	Amylase > 190 U/L	P value
< 55 U/L	94	0	0.04
> 55 U/L	0	6	

Table 5 Cross tabulation of USG abdomen findings with S. Amylase level

USG Abdomen	Amylase < 190 U/L	Amylase > 190 U/L	P value
normal	92	0	0.04
biliary sludge	2	0	
edematous pancreas	0	3	
fluid around pancreas	0	3	
total	94	6	

Six percent of the patients showed high serum amylase level (table 3). The association of elevated serum lipase in the same subjects (table 4),dehydration and hemoconcentration as evidenced by raised hematocrit, along with ultra sonogram finding of edematous pancreas and fluid around pancreas(table 5) points to acute pancreatitis in those patients.

Table 6 Cross tabulation of RBS with S. Amylase

RBS	Amylase < 190 U/L	Amylase > 190 U/L	P value
< 200 mg/dl	86	4	0.878
> 200 mg/dl	8	2	

The incidence of hyperglycemia didn't correlate well with hyperamylasemia or other evidence of pancreatitis. (Table 6)

Discussion

In our study, males have high ratio in consuming OP poison compared to female. This study is to emphasise the importance of diagnosing pancreatitis in unexplained hypotension or shock in OPC excluding other causes also whether the blood sugar elevation correlates with elevation of amylase, suggesting pancreatitis is also the cause for hyperglycemia other than stress hyperglycemia caused by release of stress hormones such as catecholamines which is induced by hyperinsulinemia that occurs during cholinergic phase. Paradoxically, studies show that hypoglycaemia can occur that cause seizure leading to diagnostic confusion[7]. As per Dressel and colleagues experiment OPC induced pancreatitis is secondary to functional duct obstruction and stimulated exocrine secretion[8]. Hyperglycemia is found in only 7% and glycosuria in 14% in our study hyperglycemia is seen in 10% along with glycosuria. Ketones are found in urine in only 2%, of which 1% is due to starvation ketosis. Remaining 1% is due to hyperglycemic ketoacidosis that present as DKA. Among the 10% of hyperglycemias, only 2% have evidence of pancreatic Inflammation. Pancreatic enzymes are elevated in 6% of the study population of which only 2% has hyperglycemia which is caused by pancreatic endocrine insufficiency[9]. In our study all the 6% of study population has evidence of dehydration and Hemo concentration favoured by rise hematocrit which has to be followed up serially. As per our Study, type of compound, amount of compound, does not predicted by amylase level. Systolic and diastolic BP does not correlates with amylase level in significant manner, except mild decrease in systolic or diastolic BP, tachycardia in OP poison correlates well with amylase level. In our study population with amylase > 190 have pulse rate of mean around 95/min also respiratory rate if increased is associated with statistically significant. Increase in amylase level in our study among 6 patients with hyperamylasemia, 4 patients has increased respiratory rate further decreased oxygen saturation have good association with amylase level thus the need for ventilatory support. In our study out of 6 patients having amylase >190 IU/L 4 patients have decreased SPO2 in range of 85 to 90%. 5 out of 6 patients needs ventilator support that have evidence of pancreatic Inflammation leucocyte count is increased in OP poison irrespective of rise in serum amylase finally serum lipase increases parallelly with amylase suggesting that other cause of hyperamylasemia is ruled out. A total of 47 patients was studied in a teaching hospital in Yuzuncuyoi university of which 4 patients have elevated amylase and lipase level around 300U/L and 60U/L of patients in which amylase levels between 100-300 U/L Only 2 patients have elevated lipase level, as assessed by calorimetric method.

Conclusion

Acute Pancreatitis can occur as a complication of organophosphorous compound poisoning. Hence, a high degree of suspicion should be kept in mind in all patients especially when the patient is dehydrated with raised haematocrit value without any other obvious reasons. The need to keep the suspected patients for prolonged nil per oral therapy may thus be emphasised. However, hyperglycemia as a complication is still inconclusive as it can also happen in any stress induced states or the patient may even be an already diabetic person who was undiagnosed. A further study with a larger study group is needed to infer hyperglycemia as an associated complication.

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