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Original research article

A study on levels of serum amylase, serum creatine phosphokinase (CPK) and random blood sugars (RBS) in acute organophosphorus compound poisoning

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Abstract

The time of occurrence of symptoms and signs depend on the route of exposure, poison load and chemical nature and solubility characteristics of the compound. Traditionally, symptoms are categorized as acute (minutes to hours) and delayed or late (days to weeks). The time of onset and mechanism of delayed manifestations such as intermediate syndrome,[87] delayed onset coma and extrapyramidal manifestation are different to that of late manifestations such as organophosphate induced delayed polyneuropathy (OPIDP) that typically occurs after 2-3 weeks and up to 4-week post exposure. Patients of age group>18years and both sex with a history of exposure to OP compound were the study subjects. According to POP score, among 188 study participants, the severity of poisoning was mild in 69 patients, moderate in 82 patients and sever in 37 patients.

Keywords: Serum amylase, serum creatine phosphokinase (CPK) and random blood sugars (RBS), acute organophosphorus compound poisoning

Introduction

Organophosphorus compounds (OPCs) are organic chemicals derived from phosphoric acids and its derivatives. They contain at least one carbon-phosphorus bond. The pentavalent types of phosphorus-containing compounds are primarily used in industrial and environmental applications. The substituents attached to the phosphorus of these esters of phosphoric acids play a vital role in toxicity. Organophosphorus pesticides are thiols, amides, or esters of phosphonic, phosphinic, phosphoric, or thiophosphoric acids with two additional organic side chains of the phenoxy, cyanide, or thiocyanate group. Some of the OPCs belong to the phosphonothioates (S-substituted), and phosphonofluoridate categories comprise of nerve agents, commonly known as chemical warfare agents [1].

The time of occurrence of symptoms and signs depend on the route of exposure, poison load and chemical nature and solubility characteristics of the compound. Traditionally, symptoms are categorized as acute (minutes to hours) and delayed or late (days to weeks). The time of onset and mechanism of delayed manifestations such as intermediate syndrome, delayed onset coma and extrapyramidal manifestation are different to that of late manifestations such as organophosphate induced delayed polyneuropathy (OPIDP) that typically occurs after 2-3 weeks and up to 4-week post exposure. Thus, we propose that symptom onset is categorized as acute (within 24-h), delayed (24-h to 2-week) and late (beyond 2-week) [2, 3].

The acute symptoms and signs are due to muscarinic, nicotinic and central receptor effects. Muscarinic symptoms of salivation and bronchorrhea that dominate initially may cause drowsy patients to drown in their secretions. Acute muscarinic effects on the heart (bradycardia, hypotension) can be life-threatening. Nicotinic effects of muscle weakness contribute to respiratory distress whilst the acute central effects of restlessness, agitation, confusion and sometimes convulsions further compromise airway and breathing

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and increase aspiration risk and hypoxia. Since many of these effects are reversed by atropine, early and appropriate medical attention is vital. In developing countries, where OP poisoning is common, quick access to medical care is more problematic than early recognition [4].
The route of exposure determines the rapidity of symptom onset. Common routes of exposure are inhalational, skin and ingestional. The inhalational route has the fastest onset, generally within a few minutes of exposure. In the terrorist attacks in Japan with the nerve gas agent Sarin, instantaneous death by respiratory arrest was suggested in 4 victims. In farmers, inhalation exposure resulting in rapid symptom onset may occur with a sudden change in the wind direction during insecticide spraying [5].
In skin exposure, the volume of exposure, intactness of the skin and solubility characteristics of the OP determines lag-time. In one report, nausea, abdominal cramping, arm and leg weakness occurred within 30-min of dermal exposure of chlorpyrifos, a lipid soluble OP. Although leg weakness improved, weakness of muscles at the site of skin exposure persisted beyond 2-week. In another report, symptom onset occurred at 3-h following the exposure to water soluble OP, monocrotophos, through a skin laceration. Symptoms of poisoning have also occurred after 4-h and 24-h after application of a home-made shampoo contaminated with an OP. In a rare situation of subcutaneous chlorpyrifos self-injection, delayed cholinergic phase, prolonged coma and severe permanent neurologic injury were observed. Delayed and prolonged effects were attributed to the adipose and muscle tissue acting as reservoirs [6].

Methodology
Study design: Cross-sectional study

Study population: patients attending the department of General Medicine Outpatient department and Inpatient Department

Sample size calculation
Since it is a time bound study, all the patients diagnosed with organophosphorus poisoning attending OPD and inpatient department during study period were included in the study.
A total of 188 study participants were included.

Inclusion Criteria
1. Patients of age group>18years and both sex with a history of exposure to OP compound were the study subjects.
2. Patients who are brought to hospital within 24 hours of poison ingestion.

Exclusion Criteria
1. Patients with history of OP poisoning mixed with any other poison or alcohol.
2. History suggestive of Myopathy, Epilepsy, Psychiatric illness, Auto immune disease, Malignancy, Trauma, Sepsis, Renal disease, Myocardial infarction and myocarditis, recent IM injections.
3. History of drug intake possibly causing myopathy like Statins, fibrates, dexamethasone, frusemide, and amphotericin B.
4. Patients with history of chronic alcoholism, lipid disorders, gall stones, parotid gland disease, acute or chronic pancreatitis, pancreatic disorders, abdominal trauma, Endoscopic retrograde Cholangiopancreatography (ERCP).
5. Patients with hepatic or renal disorders.
6. History of intake of drugs likely to produce pancreatitis- azathioprine,6 - mercaptopurine, thiazides, furosemide, pentamidine, valproate and sulphonamides.
7. Patients with age<18years.
8. Patients with history of type2 diabetes mellitus

Results

Table 1: Severity of poisoning according to pop score

Severity according to pop score	No of cases	Percentage
Mild	69	36.70
Moderate	82	43.62
Severe	37	19.68

According to POP score, among 188 study participants, the severity of poisoning was mild in 69 patients, moderate in 82 patients and sever in 37 patients.

Table 2: Serum CPK, RBS, Serum Amylase and serum cholinesterase levels with respect to severity of OP poisoning.

	Mild	Moderate	Severe	P value

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	Mean	SD	Mean	SD	Mean	SD	
Serum CPK	214.44	47.56	452.70	99.94	856.40	197.61	<0.0001
RBS	104.59	18.48	106.31	21.27	91.05	27.74	0.12
Serum amylase	97.46	21.55	162.10	24.50	204.64	40.02	0.0034
Serum cholinterase	3326	741.92	1615	516.18	560.86	287.78	<0.0001

Serum CPK levels
Mild: 214.44±47.56
Moderate: 452.70±99.94
Severe: 856.40±197.61
This difference was statistically significant.

RBS
Mild: 104.59±18.48
Moderate: 106.31±21.27
Severe: 91.05±27.74
However, the difference was not statistically significant.

Serum amylase
Mild: 97.46±21.55
Moderate: 162.10±24.50
Severe: 204.64±40.02

Serum cholinesterase
Mild: 3326±741.9
Moderate: 1615±516.18
Severe: 560.86±287.78
This difference was statistically significant.

Discussion
Organophosphorus pesticide self-poisoning is an important clinical problem in rural regions of the developing world, and kills an estimated 200 000 people every year. Unintentional poisoning kills far fewer people but is a problem in places where highly toxic organophosphorus pesticides are available. Medical management is difficult, with case fatality generally more than 15%.
Improved medical management and provision of antidotes and intensive care beds, together with bans on the most toxic pesticides should reduce the case fatality for self-poisoning and noticeably reduce the number of deaths from self-harm in rural Asia.
Various prognostic tools such as serum creatine phosphokinase (CPK), lactate dehydrogenase (LDH), serum immunoglobulins, and circulating complements for early detection of patients at high risk for developing respiratory failure have been tried. There will be the elevation of serum CPK in OP poisoning due to myonecrosis caused by persistent depolarization at the neuromuscular junction and oxidative cellular damage to muscle membrane.
Increased serum amylase is a well-documented biochemical derangement of OP poisoning which may be due to parasympathetic overstimulation of the pancreas. Organophosphate compounds act by inhibiting acetyl cholinesterase enzyme causing increased concentration of acetylcholine leading to muscarinic and nicotinic receptor overactivity. Although choline esterase inhibition plays a key role in organophosphate poisoning, other metabolic factors are also important, one such contributing factor for the severity of organophosphate compound poisoning is dysglycemia. A variety of glycaemic changes ranging from hypoglycaemia to hyperglycaemia and rarely ketoacidosis in Organophosphate poisoning is reported. Besides neurotoxicity, organophosphate induced endocrine toxicity, immunotoxicity, reproductive toxicity, genotoxicity, disruption of cellular oxidative balance and glucose homeostasis [7].
Hence, we undertook this study to determine the serum amylase, serum creatine phosphokinase (CPK) and Random blood sugars (RBS) in acute organophosphorus compound poisoning and also to correlate serum amylase, serum creatine phosphokinase (CPK) and Random blood sugars (RBS) with cholinesterase level and assess the efficacy as the prognostic indicator**.**
In our study, out of 188 study participants majority of them were in the age group of 21-30 years and most of them were male.
Yesha Chauhan *et al*., [8] in their study mentioned that, the levels of CPK were elevated significantly in patients with respiratory failure. 7 out of 8 patients with raised initial CPK level has respiratory failure and eventually death. Only one patient with mildly elevated CPK level has no respiratory failure.
Dhanalakshmi K *et al*., observed that the median CPK values in latent, mild, moderate and severe cases were 121.5 IU/L,276.5 IU/L, 308IU/L and 467IU/L respectively (p=0.015) [9].
Subathra. C *et al*. in their study observed that CPK values were increased among 60% of study group at 0 hours and 68% at 48 hours. There was a significant increase in serum CPK levels from 0 to 48 hours

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which is of high statistical significance (p =0.001). The correlations of serum CPK with outcomes such as ventilatory function and survival of the patient were statistically significant and positive at both periods of measurement. Therefore, serum CPK measurement is not only crucial at 0 hours, it is equally important to have serial monitoring of serum CPK levels, which ultimately tells the prognosis of the patients [10].

Conclusion

Serum CPK levels

Mild: 214.44±47.56, Moderate: 452.70±99.94, Severe: 856.40±197.61

RBS

Mild: 104.59±18.48, Moderate: 106.31±21.27, Severe: 91.05±27.74

Serum amylase

Mild: 97.46±21.55, Moderate: 162.10±24.50, Severe: 204.64±40.02

Serum cholinesterase

Mild: 3326±741.9, Moderate: 1615±516.18, Severe: 560.86±287.78

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