Original Article

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Evaluation of Serum Creatine Phosphokinase as a Possible Marker for Severity in Organophosphorus Poisoning

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Abstract

Background : Acute poisoning of organophosphorous compound has reached epidemic extent in largely element of the country mainly in developing countries like India. Case reports on clinical implication of total serum creatine phosphokinase in acute OP compound intake has been accounted occasionally, however there are no great extent studies with mention to total serum creatine phosphokinase level in OP compound. Hence this is an attempt to study the total serum creatine phosphokinase in assessing the severity of OP compound poison. **Subjects & Methods:** Patients with history of OP compound consumption getting admitted in Department of Medicine, Gujarat Adani Institute of medical Science, Bhuj, Kutch, Gujarat during the period of two years were occupied up for study in view of inclusion and exclusion criteria. Information needed for this study collected through a proforma and pre-test proforma from every patient. **Results:** The levels of CPK were elevated significantly in patients with respiratory failure. In this study 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. **Conclusion:** In this study only 8 out of 100 case shows raised total serum CPK level. Seven out of 8 positive case who developed respiratory failure and death. All 7 patients has marked raise in total serum CPK level. So the initial raise in total serum CPK level correlated well with severity of OP compound poison and prognosis, suggesting its use as a prognostic indicator of OP compound poison.

Keywords: Organophosphorus poisoning, Total serum CPK level, Plasma AchE, Tachycardia, Bradycardia.

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Introduction

Organophosphorous (OP) poisoning may cause the life-threatening events resulting in respiratory failure. OP compound extensively used in India as insecticides and most often for suicidal purpose mainly because of its easy availability. premature detection and appropriate interference of toxicity from these compounds are of immense significance, to critical care providers and patients.[1] Case reports on clinical significance of total serum CPK level in acute OP poison has been accounted from time to time, but there are no important studies with reference to total CPK level in OP compound poison.[2] Hence this is an attempt completed to study clinical significance of creatine kinase on OP compound poison.

OP compound acts by inhibition of AchE at muscarinic and nicotinic receptors. EchE and PchE level decrease in OP poison. But their estimation is costly. Raise in total S.CPK level can be an alternative to cholinesterase level to assess the severity of OP poison. Presences of muscle fibre necrosis in OP poison leads to increase in CPK level. In addition to this there is Rhabdomyolysis in intermediate syndrome leads to increase in CPK level.[3]

Serum CPK level is prominent yet in the deficiency of intermediate syndrome, provided the patient is sternly poisoned, apparently owing to muscle fiber necrosis, as revealed by muscle biopsy. If there is ongoing injury to the muscle owing to development of difficulties, the CPK level continues to be high. while half-life of CPK is about 1.5 days, it normalizes within 5–6 days of a solitary insult to the muscle.[4]

Serum CPK level can be an competent biomarker in case of acute OP poisoning not merely owing to its easy accessibility and not expensive, however also as serial monitoring of its level through the whole course of therapy can forecast the projection. nevertheless, the chief drawback with this marker is its non-specificity.[5] As a result, ruling out

of other causes of lifted CPK in a patient of acute OP poisoning is necessary. Serum creatine phosphokinase can be of scrupulous significance in developing countries, where other expensive biomarkers are complicated to approximate. Hence the objective of the current study was to measure the changes in serum level of total creatine phosphokinase in assessing the severity of OP compound poison.

Subjects and Methods

Patients with history of OP compound consumption getting admitted in Department of Medicine, Gujarat Adani Institute of medical Science, Bhuj, Kutch, Gujarat during the period of two years were taken up for study considering inclusion and exclusion criteria.

Methods of Collection of Data

- Information will be serene through a proforma and pre-test proforma from each patient
- Qualifying patient will be undergoing detailed history, clinical examination and biochemical examination

Type of Study: Prospective observational study.

Sample Size: 200 patients

Inclusion Criteria:

Patient with definite history of OP compound poison and who actually produce the evidence of OP compound sample will be the study subjects.

Exclusion Criteria:

- Patients with OP compound poison consumption along with any other poison like kerosene/alcohol
- Patient with indication of exposure to a entirely different poison other than OP compound
- Patient who are chronic alcoholics
- Patient with history suggestive of myopathy
- History of drug intake like steroids, statins, fibrates, etc
- Any trauma involving muscle

Results

This study conducted in Medical Hospital amid over declared period, showed incidence of OP compound poisoning common in males compared to females. There were total 200 patients included in the study. Of them there were 124 males and 76 females. Majority of patients were in the age group < 30 years with a mean of 33.23 ± 10.12.

There were different type of OP compound that were consumed by the patient admitted in the medical college & hospital. As per the allocation of patients according to the category of OP Compound inspired, mainly frequent compound addicted was Phorate (30%) followed by Quinolphos (24%), the least consumed compound was found to be Chlorpyriphos consumed by 10% of patients.

As per the peradeniya score the patients were separated into 3 different groups. Mainstream of the subjects had a mild (61%) to moderate (30%) severity of organophosphorus poisoning at appearance, with 9% having severe poisoning. The most common clinical finding in patients was bradycardia (33%) followed by tachycardia (15%). Hypertension was seen in (17%) patients and hypotension (2%).

In the current study abnormal ECG was noted in 94 cases. The most common finding in ECG is sinus bradycardia (31%) followed by sinus tachycardia (16%) and normal is (53%). ST elevation is 0% [Table 1].

Table 1: Distribution of patients according to their ECG changes (N = 200)

ECG Changes	No. of patients
Sinus Tahycardia	32
Sinus Bradycardia	62
ST elevation	0

Table 2: Distribution of patients according to their Total serum CPK changes (n=200)

CPK positivity (>300U/L)	No. of patients
At Day 1	16
At Day 3	0
At Discharge	1

Table 3: Distribution of patients according to their outcome (N = 200)

Outcome	No. of patients
Respiratory failure	74
No Respiratory failure	124
Death	44

The CPK changes were assessed at the end of first day and third day. At the end of day 1 the elevated CPK levels more than 300 U/L were found in 16 patients whereas at the end of day 3 there were no patients with elevated CPK levels. As per the outcome of the patients the respiratory failure was found in 76 patients and 124 patients did not have any respiratory failure.

Association of clinical severity (peradeniya score) of patients with raised total serum CPK level at end day 1 shows that CPK levels positivity was seen in 16 patients where as in 184 patients there was CPK negative levels. Maximum patients

had mild symptoms and very few patients had severe patients. Statistical analysis shows that the difference between the groups was found to be statistically significant with p value 0.01. Serum CPK shows significant difference between mild, moderate and severe poison. CPK level in survivors and non-survivors statistically significant (p=0.002).In non-survivors CPK level greatly elevated throughout the course of disease.

Discussion

In this study 200 patients of organophosphorus poisoning who were brought to Hospital with varying degree of severity were included and the total serum creatine phosphokinase in assessing severity of op compound poison is studied.

In this study patient in the age group ranging from 18 to 85 years were included with mean age of 32.39 ± 11.26. The highest number of patients were in the age group < 30 years (44%), followed by 30-39 years (34%), which is similar to that in other studies.60,61 The incidence of poisoning was more in males (62%) when compared to females (38%). Present findings were concurrent well with research performed by Dash et al. study,[6] which demonstrated an incidence of 67% in males and 23% in females.

Phorate (30%) was the mainly widespread compound occupied in the poisoning. It was pursued by quinolphos (24%) and dimethoate (23%). This was dissimilar from the research performed by Karki P et al.[7] Who found the most widespread complex as Methyl parathion (23%) followed by Propoxur (5%), which can be described by the dissimilarity in accessibility of compound in a meticulous geographic location.

Out of the 100 patients who were considered 61% had mild, 30% had moderate and 9% had severe level of poisoning. Bradycardia (33%) was the common clinical sign seen in this study, which can be explained based on increased parasympathetic tone. Bradycardia was seen in 8 out 9 severe poison based on POP scale. Hence bradycardia can be considered as an indicator of severity of poisoning. Blood pressure changes in the form of hypertension (SBP ≥140mmHg and/or DBP ≥90mmHg) and hypotension (SBP ≤90mmHg) were seen in 17% and 2% respectively. Two patients who developed hypotension did not recover and died after a prolonged ICU stay. This clinical feature was in concurrence with the research done by Saadeh AM et al.[8]

The ECG reflects the widespread cardiac toxicity of OP compound. Ludromirsky et al. had illustrated 3 phases of cardiotoxicity after OP poison. Phase concise period of augmented sympathetic tone, Phase 2-prolonged period of parasympathetic activity, Phase 3-QT prolongation followed by Torsades de pointes, VT and the VF. In this study we used ECG only to document presence of sinus bradycardia, Sinus tachycardia and exclusion of ST elevation in the study. In the current study abnormal ECG was noted in 94 cases. The most common finding in ECG is sinus bradycardia (31%) followed by sinus tachycardia (16%). The results were same with other studies. In the study done by Balouch et al,[9] and Sadeesh et al,[8] sinus bradycardia was the most common ECG finding when compared to sinus tachycardia.

In this present study, raised total serum CPK level used as an indicator in assessing the severity of OP compound poison and its prognostic significance were positive (CPK ≥300 IU/L) in 16 out of 200 patients with mean value of 986.75 IU/L on day 1 of admission. This was analogous to the study conducted by Kuntal Bhattacharya et al.[2] 14 out of 16 patients who has significantly raised total serum CPK level died, it itself explain its prognostic significance and its role in assessing the severity of OP compound poison. Among the 16 patients who had found to have raised total serum CPK level, Phorate was the compound in 6 patients, Methyl parathion in 4 patient, Dimethoate in 4 patients and Quinolphos in 2 patient.

However present research demonstrates that serum CPK level is eminent yet in nonexistence of transitional syndrome offer the patient is harshly poisoned, most probably owing to muscle fibre necrosis. Given that the half-life of CPK is concerning 1.5 days, it standardize within 5-6 days of a solitary insult to the muscle. It has been exposed by Senanayeke et al.[10] that the POP score can powerfully forecast the harshness, morbidity and mortality of OP poisoned patient. In addition to present study, we establish that occasionally POP score can be illusory and still a patient with comparatively low POP score can develop difficulties and/or death with elevated total serum CPK level.

Conclusion

Serum CPK level can be a competent biomarker in case of acute OP poisoning not merely owing to its simple accessibility and small cost, but also as sequential monitoring of its level during the whole course of therapy can forecast the diagnosis. Nevertheless, the chief disadvantage with this indicator is its non-specificity. So, keeping out of other causes of raised CPK in a patient of acute OP poisoning is necessary.

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