

Serum Creatine Phosphokinase as Prognostic Indicator in the Management of Electrical Burn

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ABSTRACT

Objective: To determine the mean difference of serum creatine phosphokinase according to the extent of tissue damage and prognosis of the patients suffering from electrical burn injuries.

Study Design: A case series study.

Place and Duration of Study: Department of Burns, Liaquat University Hospital, Hyderabad, from September 2005 to August 2006.

Patients and Methods: During the study period, 38 patients with electrical burn injuries were enrolled. Victims of electrical burns with evidence of myocardial injury were excluded. Following admission, serum creatine phosphokinase was measured serially on 10 consecutive occasions. The data was later analyzed statistically using SPSS-10.0.

Results: Of the 38 patients, the mean age of the victims was 28 years, with males dominating the study population (82%). A statistically significant association was found between the level of serum creatine phosphokinase and likelihood of death ($p=0.000$). It was also found that serial monitoring of this enzyme can be used as prognostic indicator in the management of electrical burns injury.

Conclusion: The level of creatine phosphokinase increased with the degree of tissue injuries in patients with electrical burns. This prognostic value is of great importance in the local setup, where sophisticated investigations to detect extent of injuries are not available.

Key words: *Electrical burns. Creatine phosphokinase (CPK). Prognosis.*

INTRODUCTION

In electric shock injuries, the true extent of injury is unlikely to be strongly correlated with the extent of cutaneous burns. To estimate the extent of underlying tissues damage, various diagnostic tests are available in developed countries, like Magnetic Resonance (MR) imaging, MR spectroscopy, Positron Emission Tomography (PET); and Single Photoelectron Emission Computed Tomography (SPECT).¹ The estimation of serum CPK has frequently been used to diagnose the tissue injuries and recent studies suggest that a strong relation exists between elevated serum CPK and the extent of tissue trauma after electric burn.^{2,3} The elevated levels of serum CPK is a marker of increased permeability of muscle membranes⁴ either to functional or mechanical damage to the muscle cell membranes. Three different mechanisms can disrupt the myocytes with rise in serum CPK.⁵⁻⁷ These include direct injury to the cell membranes that is crushing, tearing, burning, muscle cell hypoxia, vascular occlusion, tissue compression, and severe electrolytes disturbances

resulting from any cause. The serum CPK level peaks dramatically in first 12 to 36 hours but decreases steadily over the next several days.⁸

Creatine Phosphokinase (CPK) is an enzyme, which catalyses the reversible phosphorylation of creatine by Adenosine Triphosphate (ATP), found primarily in muscle tissue. Of its three dimmers, BB, MB and MM are composed of the two sub-units B (for brain) and M (for muscle). The primary isoenzyme in the skeletal muscle is CK-MM, while significant amounts of CK-MB are reported to be contained in cardiac muscle cells. The isoenzyme CK-MB of creatine kinase, when increasing to more than 10% of the whole enzyme activity, is characteristic for myocardial damage.⁹ However, the sensitivity is very low, if compared to troponin values.^{10,11} Moreover, the incidence of myocardial damage in association with the electric injuries is very low.^{12,13} Electrical injury is a relatively infrequent but potentially devastating form of multi-system injury with high morbidity and mortality.

The severity of the injury depends on the intensity of the electrical current (determined by the voltage of the source and resistance of the victim), the pathway it follows through the victim's body, and the duration of contact with the source of the current.¹⁴ Immediate death may occur from current-induced ventricular fibrillation, asystole, respiratory arrest secondary to paralysis of the central respiratory control system, or paralysis of the respiratory muscles. Presence of severe

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burns (common in high-voltage electrical injury), myocardial necrosis, the level of central nervous system injury, and the secondary multiple system organ failure determine the subsequent morbidity and long-term prognosis.

The different tissues of body resist the impact of electricity in different manner. Skeletal muscle and peripheral nerve tissue are especially susceptible to injury. This extensive damage to the skeletal muscles is in turn responsible for so many possible aftermaths of electrical burns including fasciotomies, amputation, acute renal failure and even multi-organ failure.

This study was designed to determine the likely relation between the level of serum creatine phosphokinase and extent of muscle damage and its significance as prognostic indicator in electrical injuries.

PATIENTS AND METHODS

This analytical study was carried out at Burns Emergency Unit of Liaquat University Hospital, Hyderabad, from September 2005 to August 2006. During this period, 38 patients with significant electric burn injury were admitted. Informed consent was obtained. Soon after admission and history taking, primary survey was done. The Body Surface Area (BSA) was mapped out according to the Lund and Browder chart. Blood sample was drawn for routine serum investigations and serum CPK. Electrocardiography was conducted and resuscitation with Ringer solution was started intravenously. The amount of the fluid was titrated to get urine output of 1-1.5 ml/kg/hour. Intravenous injections of antibiotics and antacids were started for prophylaxis of infection and stress ulceration, respectively. Oral alkalinizing agent was started to reduce possible precipitation of myoglobin in renal tubules.¹⁵ Narcotic analgesia was used to control pain. After resuscitation, secondary survey was done to exclude any associated musculoskeletal injuries, fracture, head injury and neurological deficits and findings were recorded on a specific proforma. The need for urgent escharotomy/ fasciotomy/decompression was evaluated and performed immediately when indicated. Serum CPK was measured serially, using its total (un-fractionated) amount with a commercial laboratory analyzer system for 10 times on different occasions, first at the time of admission, then daily for 4 days, and later on alternate days. The different surgical procedures performed include fasciotomies, partial and/or full thickness skin grafts, minor amputation of digit, major amputation of more distal portion of extremity, total limb amputation and flaps.

The data were analyzed using SPSS-10.0. The continuous variables like CPK were presented by mean \pm SD, while categorical variables like final outcome and complications were presented by frequencies and

percentages. Repeated measure ANOVA was applied to compare serum CPK between alive and dead cases on subsequent follow-up, findings and LSD-test was applied for post-Hoc comparison. P-value \leq 0.05 was considered statistically significant result.

RESULTS

A total number of 38 patients were analyzed, out of whom, 31 (82%) were males and 7 (18%) were females. The age of patients ranged from 7 to 62 years with mean age of 28 years. The mean body surface area burnt was 23% and ranged from 0.75 to 70% as shown in Figure 1. Thirty cases (79%) suffered from contact and flash burns, whereas 7 cases (18.4%) suffered from flash burns and 1 (2.6%) from arc injury. Nineteen cases (50%) were the victims of high voltage injury, while 14 cases (36%) were sufferers of low voltage, and 5 cases (14%) had household electrical injury.

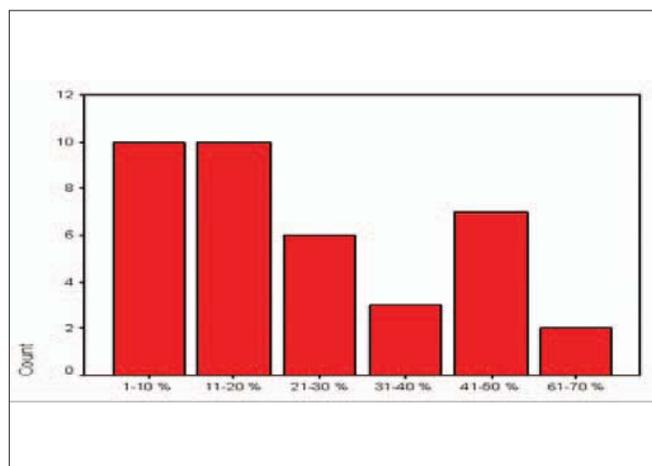


Figure 1: Body surface area involved in electrical burns.

Eighty different surgical procedures were performed on 38 cases. The commonest procedure performed was fasciotomies in 26 cases (32.5%), skin grafting in 16 cases (20%), partial limb amputations in 10 cases (12.5%), total limb amputations in 09 cases (11.25%), multiple procedures in 03 cases (3.75%) and revision surgery in 07 cases (8.75%). Twelve patients died. Septicemia was the cause of death in 6 cases (50%), renal failure in 03 cases (25%), and multiple organ failure in 3 cases (25%).

The probability of performing more major surgical procedures was found to increase in direct relation to the level of serum CPK at the time of admission ($p=0.000$); greater the serum CPK, greater was the need for invasive surgical procedure to save the life of the patient.

This was also same with reference to development of different complications ($p=0.000$), like wound infection, invasive sepsis and acute tubular necrosis. Mean serum

CPK level for all 38 patients, at the time of admission, was 38878 ± 13195.21 U/L. Statistically significant difference ($p=0.000$) was observed for those patients who expired during the period of hospital stay, who showed not only initial high levels of serum CPK (mean 48518.07 U/L) but its decline after indicated surgical procedure was not as dramatic as for those who survived (mean CPK 28098.26 U/L), as shown in Figure 2 and Table I.

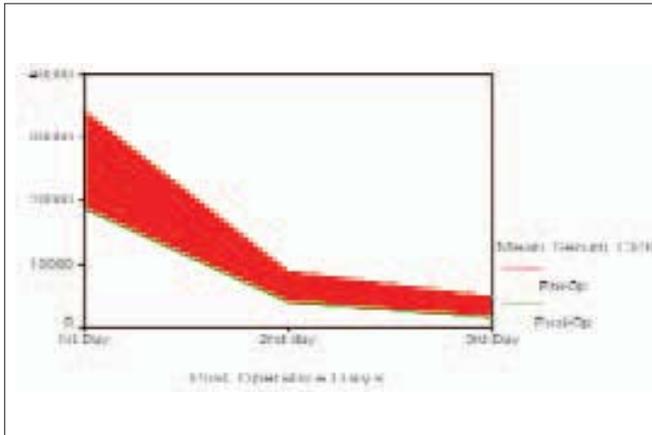


Figure 2: Mean serum CPK before and after emergency surgery.

Table I: Mean CPK with respect to final outcome.

Variables/ mean CPK on	Alive (n=26)	Dead (n=12)	p-value
Admission	28098.26 ± 10758.64	48518.07 ± 5428.44	0.00
2 nd day	18311.53 ± 9844.67	43041.84 ± 6021.37	0.00
3 rd day	17730.08 ± 8072.42	38230.60 ± 4540.97	0.00
4 th day	12036.55 ± 5918	30830.80 ± 4368.10	0.00
5 th day	8232.26 ± 12209.37	17045.58 ± 7088.86	0.00
7 th day	2163.80 ± 1092.43	9712.71 ± 4240.31	0.00
9 th day	779.91 ± 302.29	4110.19 ± 1536.28	0.00
11 th day	237.63 ± 100.87	1374.41 ± 495.62	0.00
13 th day	85.25 ± 36.89	303.49 ± 331.52	0.00
15 th day	45.00 ± 00	87.79 ± 336.26	0.00

Key: mean \pm standard deviation. A substantial fall in serum CPK level taken from baseline to day-15 follow-up both inter group and intra groups was observed ($p<0.001$).

A substantial fall in serum CPK level taken from baseline to day-15 follow-up was observed ($p<0.001$).

CPK levels directly correlated with the tissue damage and it became more evident when the level dropped to a significant level after performing different procedures, as shown in Figure 2.

The percentage of burnt area is significantly related to the level of CPK at the time of admission ($p= 0.000$), but the likelihood of performing different procedures in relation to body surface area affected was also not statistically appreciable.

It was found that likelihood of mortality and morbidity increased with higher levels of CPK at the time of admission and remained at relatively higher level even

after emergency surgical procedure like fasciotomy, decompression and debridement. Patients, who survived (26/38), were those who responded well to the emergency surgical procedure and their serum CPK showed remarkable reduction.

DISCUSSION

Creatine phosphokinase level is the most sensitive indicator of rhabdomyolysis due to any cause when compared to myoglobin.¹⁶ The half-life of CPK is 1.5 days and so it remains elevated longer than serum myoglobin levels.¹⁷ The estimation of serum and urine myoglobin is useful only in the very early phase of the disease.¹⁸ It has short plasma half life of 2-3 hours and can only be detected in urine when plasma concentration exceeds 1.5 mg/dl.¹⁹ The serum level may return to normal in 6-8 hours due to its rapid filtration through the kidneys and metabolism to bilirubin.²⁰ When a part of body becomes a circuit element, power dissipation within that part produces heat and a rise in temperature. This heat is distributed to deeper tissues, and the low-resistance soft tissues, interposed in between highly-resistant skin and bone, sustain the greatest damage. Amongst the soft tissues, skeletal muscles suffer the largest rise in temperature and destruction. Joule heating has traditionally been used to describe the mechanism of tissue damage due to electrical burns.²¹ Recently, another tissue damaging mechanism known as electroporation is described, where permeabilization of cell membranes and direct electroconformational denaturation of macromolecules such as proteins mediate significant skeletal muscle necrosis with insignificant external wounds but with progressive internal damage.²²⁻²⁴ Thus, the external damage often contradicts the severity of deeper wounds. After initial resuscitation, the aim of the management is prevention of complications like renal failure, life-threatening vascular compromise and compartment syndrome.

As a result of massive soft tissue damage, the level of CPK rises in serum tremendously and this serum marker of electrical injury was used as a prognostic indicator in this study. The initial value of CPK provided some clue of the damage sustained by the deeper tissues even when there were no marked clinical manifestations at the time of presentation. This observation corresponds with the findings presented by Ahrenholz *et al.*²⁵ and Kopp *et al.*² Thus, greater the levels of CPK at admission, higher are the chances of complications. A well-timed fasciotomy/decompression can be limb saving.^{26,27} However, amputation can save life. The major bulk of our study population comprised of males. There was no appreciable relation between body surface area burn, with the initial level of CPK and the probability to perform different therapeutic procedures.

These findings are supported by international literature.²⁸ It was found that the likelihood of mortality and morbidity increased with higher levels of CPK at the time of admission and it remains at relatively higher levels even after emergency surgical procedure like fasciotomy, decompression and debridement. This reflects ongoing muscle damage probably at sites remote from the site of surgical exploration. Patients, who survived, were those who responded well to the emergency surgical procedure and their serum CPK showed remarkable reduction.

Since the wick, Doppler catheter, or manometer are not yet available in our setup to monitor intra-compartmental pressure, an initial high level or persistent rise of this enzyme in the serum can provide a base for more timely active management.

CONCLUSION

The serum levels CPK in patients with electric burn injury is a sensitive marker for ongoing muscle damage, which may not be visible at initial assessment. In setting where facilities for diagnostic imaging are not available to determine the muscle necrosis, or intra-compartmental pressure monitoring devices are not available or when the patient is unstable, serial monitoring of this enzyme can help in managing the complications at an early stage and predicting the outcome.

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