Ulus Travma Acil Cerrahi Derg 2010;16 (3):225-228
Klinik Çalışma

The myopathic effects of electrical injury

Elektrik yaralanmasının miyopati etkileri

Dursun AYGÜN,[1] Hayriye GÖNÜLLÜ[2]

BACKGROUND
In this study, we investigated the effect of voltage levels on muscle damage in patients with electrical injuries.

METHODS
This retrospective study included 36 patients with electrical injury (high voltage, 21; low voltage, 15). Initial serum creatine kinase (CK), aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were obtained from each patient on admission.

RESULTS
Although CK levels were observed to be higher in the high-voltage injured patients than in those exposed to a low voltage, the difference was not statistically significant. The serum AST and ALT levels were elevated in both groups, but there was no significant difference between the groups.

CONCLUSION
Skeletal muscle damage can be caused by both high-voltage and low-voltage electrical injury, and in these patients, the degree of muscle damage may be consistent with the elevated initial serum levels of muscle enzymes, especially of CK. However, further researches are necessary to determine if there is strong evidence of a direct correlation between voltage level and the degree of muscle damage.

Key Words: Alanine aminotransferase; electrical injury; high-voltage; low-voltage; myopathy; serum creatine kinase; serum aspartate aminotransferase.

AMAÇ
Bu çalışmada, elektrik çarpmasıyla yaralanan hastalarda voltaj seviyesinin kas harabiyeti üzerine etkisi araştırıldı.

GEREÇ VE YÖNTEM
Bu geriye dönük olarak yapılan çalışmaya elektrik çarpmasıyla yaralanan 36 hasta alındı (21 yüksek voltaj ve 15 düşük voltaj). Başvuruda her bir hastanın serum kreatin kinaz (CK), aspartat aminotransferaz (AST) ve alanin aminotransferaz (ALT) değerleri belirlendi.

BULGULAR
Her nekadar biz çalışmamızda yüksek voltaja maruz kalan hastalardaki serum CK seviyelerini düşük voltaja maruz kalan hastalardakilerden daha yüksek bulduysak da, bu iki grup arasındaki fark anlamlı değildi. Serum AST ve ALT seviyeleri her iki grupta da yüksek idi. Fakat gruplar arasında hiçbir anlamlı fark yoktu.

SONUÇ
İskelet kas harabiyetinin yüksek ve düşük voltaj elektrik yaralanması ile meydana gelebildiği ve bu hastalarda kas harabiyeti derecesinin kas enzimlerinin özellikle CK'nın, yükselmiş başlangıç serum seviyeleri ile uyumlu olabildiği sonucuna varıldı. Bununla birlikte voltaj seviyesinin kas harabiyetinin derecesi ile direkt korelâsyonunun güçlü bir delilinin olup olmadığını belirlemek için daha ileri araştırmalar gereklidir.

Anahtar Sözcükler: Alanin aminotransferaz; elektrik yaralanması; yüksek-voltaj; düşük-voltaj; kas harabiyeti; serum kreatin kinaz; serum aspartat aminotransferaz.

Electrical injury is the passage of electrical current through the body. Although it is relatively uncommon, it still carries high risk of morbidity and mortality.[1] Electrical injuries have been classified generally as low voltage and high voltage.[2,3] It is known that low-voltage electrical injury in humans normally does not result in significant tissue damage; however, low-voltage electrical injuries have been reported to sometimes cause significant morbidity and mortality. In high-voltage electrical injury, tissue damage is usually more severe.[4-6] Additionally, high voltage results in greater current flow, which may be high enough to

[1]Department of Neurology, Ondokuz Mayıs University, Faculty of Medicine, Samsun; [2] Department of Emergency Medicine, İzmir Tepecik Training and Research Hospital, İzmir, Turkey.

[1]Ondokuz Mayıs Universitesi, Tıp Fakültesi, Nöroloji Anabilim Dalı, Samsun; [2]İzmir Tepecik Eğitim ve Araştırma Hastanesi, Acil Servis, İzmir.

Correspondence *(İletişim):* Dursun Aygün, M.D. Ondokuz Mayıs Üniversitesi, Tıp Fakültesi Nöroloji Anabilim Dalı, 55139 Samsun, Turkey.
Tel: +90 - 362 - 312 19 19 / 3243 Fax *(Faks)*: +90 - 362 - 457 60 41 e-mail *(e-posta):* daygun@omu.edu.tr

cause massive destruction of the tissues.[2] It has been reported that electrical injury causes skeletal muscle damage (myopathy), and the extent of muscle damage can be assessed with serum measurements of creatine kinase (CK).[2,4,7] It has been reported that serum CK and aspartate aminotransferase (AST) levels increase significantly in groups with skeletal muscle damage compared to controls.[8-11] High serum CK activity usually reflects the magnitude of acute muscle necrosis and is the most sensitive indicator of muscle injury.[8] It is also the best measure of the course of muscle injury.[8] Kopp et al.[7] investigated the possible correlation between the extent of muscle cell damage and initial CK levels in a retrospective analysis of 42 high-voltage injuries. The authors demonstrated that the degree of muscle tissue damage was consistent with the initial increased serum levels of CK, and they suggested that the serum course of CK can be used as a prognostic factor.[7] Wang et al.[12] indicated that CK levels are sensitive tests for evaluating the depth of burn in patients with electrical burns. To our knowledge, electrical injury-induced myopathies are extremely rare, and we did not find any study in the literature comparing high- and low-voltage electrical injuries.

In this study, we investigated the relationship between the degree of muscle damage, by measuring the initial levels of the muscle enzymes, and voltage level in electrical injury patients.

MATERIALS AND METHODS

This retrospective study was approved by the Local Ethics Committee of our University.

All adult patients (aged 18 years or older) who presented to the emergency department with electrical injury were included in this study.

In the present study, we investigated the association between voltage level and the degree of muscle damage by retrospectively analyzing the serum CK, alanine aminotransferase (ALT), and AST findings in 36 patients suffering electrical injury between December 2001 and May 2006.

According to current publications in the literature, the cases were divided into two groups as low- (<1000 V; Group 1) and high-voltage (>1000 V; Group 2) injuries.[2,3] Measurement of total CK serum levels was performed using a Cobas Integra CK kit for serum (Cobas Integra® 800) with a linear range from 35 to 195 U/L. Similarly, serum AST (N: 8-46 U/L) and ALT (N: 7-46 U/L) were also measured using the Cobas Integra kits The groups were compared with respect to serum CK, AST and ALT levels.

The results were analyzed using computer software (Statistical Package for Social Sciences [SPSS], version 10.0). The Mann-Whitney U test was used for the statistical analysis of data. A value of $p<0.05$ was considered statistically significant.

RESULTS

In total, data of 36 patients (33 M, 91.7%; 3 F, 8.3%; Table 1) were analyzed. Of the 36 patients, 15 (41.7%) had suffered from a low-voltage injury, while 21 (58.3%) had sustained a high-voltage injury.

Patients were aged between 17-65 years, with an average of 34.05±15 years. The mean age was 33.07±15 years in Group 1 and 34.76±15 years in Group 2, and the difference between the groups was not statistically significant ($p>0.05$; Table 1).

The mean initial serum CK level was 1213.73±2072 U/L in Group 1 and 2882.42±4041 U/L in Group 2 (Table 1; Fig. 1), and there was no significant difference between the groups ($p>0.05$) (Table 1, Fig. 1).

The mean serum AST was 120.77±133 U/L in Group 1 and 119.00±104 U/L in Group 2 ($p>0.05$), and these values for ALT were 73.78±79 U/L and 72.85±71 U/L, respectively (Table 1, Fig. 1); there were no significant differences between the groups for either parameter ($p>0.05$).

The mean Glasgow Coma Scale (GCS) score was 13.20±3.8 in Group 1 compared to 13.90±3.3 in Group 2 ($p>0.05$). The patient characteristics are summarized in Table 1.

Table 1. The demographic, clinical and laboratory characteristics of the patients

Characteristic	Low voltage	High voltage	p
Patients (n/%)	15 (41.7)	21 (58.3)	—
Mean age (years)	33.07±15	34.76±15	>0.05
F/M	1/14	2/19	—
GCS score (N: 3-15)	13.20±3.8	13.90±3.3	>0.05
CK (N: 35-195 U/L)	1213.73±2072	2882.42±4041	>0.05
AST (N: 8-46 U/L)	120.77±133	119.00±104	>0.05
ALT (N: 7-46 U/L)	73.78±79	72.85±71	>0.05
pH	7.37±0.06	7.38±0.09	>0.05
WBC (N: 4-10x1000/uL)	13480.00±7894	13819.05±5646	>0.05

F: Female; M: Male; GCS: Glasgow coma scale; CK: Creatine kinase; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; WBC: White blood cell.

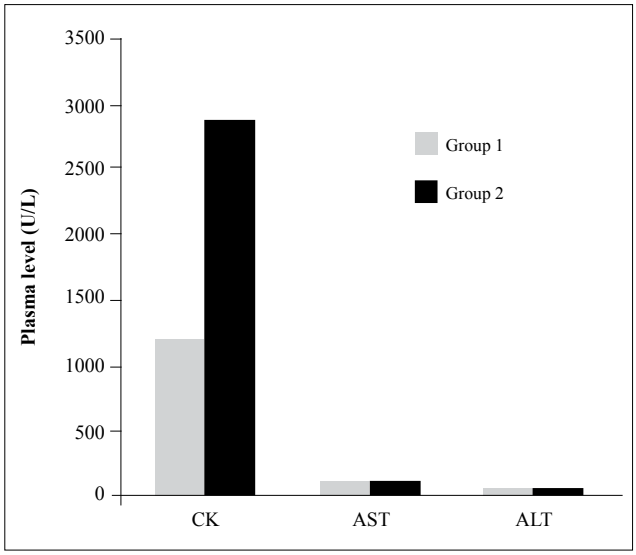

Fig. 1. The levels of the serum CK, ALT, and AST in the patients with low- (Group 1) and high-voltage (Group 2) electrical injury.

CK: Creatine kinase; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase.

DISCUSSION

It has been reported that the pathological effects of electric current in humans are due to thermal heating of the tissues; dysfunction of organ systems, such as the circulatory and respiratory systems; or continuous stimulation of the nerves and striated muscles.[4] The severity of injury also depends on the voltage, exposure duration, resistance of the tissues, and contact surface, in addition to the intensity, type, and pathway of the current.[1,4] For example, while direct current produces a single contraction in the muscles, alternating current leads to tetanic muscle contractions.[2,5] However, electrical injuries are essentially divided into two main subcategories as high and low voltage, using 500 or 1000 V.[2,3] Although both high and low voltage can cause significant morbidity or mortality, high voltage exhibits a greater current flow and hence causes more severe tissue damage.[4,5] Thus, we think that both high- and low-voltage electrical injuries may produce muscle damage (myonecrosis) with myoglobinemia and myoglobinuria. In the present study, the patients in the groups had similar characteristics in the factors related to the severity of electrical injury except for the voltage exposure. We compared the groups to determine the effects of voltage level on the degree of muscle damage. While the patients exposed to high voltage had a higher level of CK than those exposed to low voltages, the difference between the two groups was not statistically significant. These results reveal that both high and low voltage can produce muscle damage; however, high voltage had a more severe effect on muscle damage than low voltages.

It has been reported that muscle damage due to electrical injury is associated with contraction of striated muscles.[2,4,5] It is a consequence of electrical nerve stimulation or the direct triggering of striated muscles, and this condition can result in rupture, rhabdomyolysis, edema, and myonecrosis.[2,7] Compartment syndromes may also develop because of muscle swelling and necrosis.[13] It has been reported that in the muscle cells, the primary pathology is membrane permeability or membrane rupture, and this condition results in the loss of cellular enzymes.[7] As a result, the serum levels of the muscle enzymes, including CK, elevate. Therefore, the increased serum concentrations of such enzymes may be indirect markers of muscle damage.[2,7,14,15]

Ahrenholz et al.[16] reported that serum CK and CK myocardial band isoenzyme (CK-MB) levels were obtained from 116 of 125 electrical burn patients, and electric current-induced myocardial infarction (MI) was rare. Furthermore, MI could not be diagnosed by elevated CK-MB measurements alone. However, Kopp et al.[7] studied the CK levels in the serum of high-voltage injury patients and demonstrated that initial serum CK levels were related to the degree of muscle damage. They found markedly elevated CK activity (180903 U/L on average) on admission.[7] We observed a lower average CK level than their patients in our high-voltage injury patients. In contrast to previously published studies, our study included patients exposed to both high and low voltage. In the present study, the serum AST and ALT levels were also elevated in both groups, but findings in Group 1 were comparable to those in Group 2.

A limitation of the present study was the small size of the groups. However, the present results are consistent with previous studies in the literature data.[5]

We conclude that skeletal muscle damage can be caused by both high- and low-voltage electrical injury, and that in these patients, the degree of muscle damage may be consistent with the elevated initial serum levels of muscle enzymes, especially of CK. However, further researches are necessary to determine if there is strong evidence of a direct correlation between voltage level and the degree of muscle damage.

REFERENCES

1. Koumbourlis AC. Electrical injuries. Crit Care Med. 2002;30:S424-30.
2. Spies C, Trohman RG. Narrative review: Electrocution and life-threatening electrical injuries. Ann Intern Med 2006;145 531-7.
3. Yowler CJ. Recent advances in burn care. Curr Opin Anaesthesiol 2001;14:251-5.
4. ten Duis HJ. Acute electrical burns. Semin Neurol 1995;15:381-6.
5. Martinez JA, Nguyen T. Electrical injuries. South Med J

2000;93:1165-8.
6. Cooper MA. Emergent care of lightning and electrical injuries. Semin Neurol 1995;15:268-78.
7. Kopp J, Loos B, Spilker G, Horch RE. Correlation between serum creatinine kinase levels and extent of muscle damage in electrical burns. Burns 2004;30:680-3.
8. Bohlmeyer TJ, Wu AH, Perryman MB. Evaluation of laboratory tests as a guide to diagnosis and therapy of myositis. Rheum Dis Clin North Am. 1994;20:845-56.
9. Preston DC, Shapiro BE, Brooke MH. Proximal, distal, and generalized weakness. In: Bradley WG, Daroff RB, Fenichel GM, Jankovic J, editors. Neurology in clinical practice. Philadelphia, PA: Butterworth & Heinemann; 2004. p. 367-86.
10. De Bleecker J, Van den Neucker K, Colardyn F. Intermediate syndrome in organophosphorus poisoning: a prospective study. Crit Care Med 1993;21:1706-11.
11. Giltay EJ, van Schaardenburg D, Gooren LJ, Kostense PJ, Dijkmans BA. Decreased serum biochemical markers of muscle origin in patients with ankylosing spondylitis. Ann Rheum Dis 1999;58:541-5.
12. Wang XW, Jin RX, Bartle EJ, Davies JW. Creatinine phosphokinase values in electrical and thermal burns. Burns Incl Therm Inj 1987;13:309-12.
13. Aminoff MJ. Effect of physical agents on the nervous system. In: Bradley WG, Daroff RB, Fenichel GM, Jankovic J, editors. Neurology in clinical practice. Philadelphia, PA: Butterworth & Heinemann; 2000. p. 1541-4.
14. Duff K, McCaffrey RJ. Electrical injury and lightning injury: a review of their mechanisms and neuropsychological, psychiatric, and neurological sequelae. Neuropsychol Rev 2001;11:101-16.
15. Fish RM. Electric injury, part I: treatment priorities, subtle diagnostic factors, and burns. J Emerg Med 1999;17:977-83.
16. Ahrenholz DH, Schubert W, Solem LD. Creatine kinase as a prognostic indicator in electrical injury. Surgery 1988;104:741-7.