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Case Report

Rhabdomyolysis following electrical injury without acute kidney injury

Alper Alp[1*], Burcu Arslan[2], Dilek Gibyeli Genek[1], Bülent Huddam[1]

[1]Department of Nephrology, Faculty of Medicine, Mugla Sıtkı Koçman University, Mugla, Turkey
[2]Department of Internal Medicine, Faculty of Medicine, Mugla Sıtkı Koçman University, Mugla, Turkey

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***Corresponding author:**
Alper Alp,
Email: alperalp@mu.edu.tr

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Abstract

Objective: Rhabdomyolysis is an important etiology for developing acute kidney injury (AKI). Among the many varying reasons for rhabdomyolysis, electrical injury seems to be a lesser-known factor. The clinical presentation of rhabdomyolysis is usually in the form of severe and widespread pain, tenderness, weakness in the muscles and dark urine. It is characterized by the disruption of cell integrity in myocytes as a result of widespread damage to skeletal muscles and the passage of intracellular components into the circulation.

Case Presentation: Here we presented a case report of a young man who had rhabdomyolysis induced by electrical injury which is relatively less common among the other etiological factors with preserved renal functions. He had electrical injury related wounds on extremities. Urgent intravenous fluid therapy was initiated as soon as his admission to the emergency department (ED), without delay.

Conclusion: AKI is very common due to the nephrotoxic effect of myoglobinuria and the prerenal status. It is rare that AKI does not develop in patients with a severe increase in creatinine kinase. It is a very important point to start effective fluid therapy in a short time.

Keywords: Creatine kinase, Accidents, Occupational, Rhabdomyolysis, Fluid therapy

Introduction

Rhabdomyolysis is considered a systemic and serious clinical condition because of both acute kidney injury (AKI) it can cause and other serious complications (1). There are many traumatic and non-traumatic causes in the etiology. Crush syndrome, burns, and lightning strike are common among traumatic ones. Among the non-traumatic causes, sepsis, toxins, drugs, hyperthermia, status epilepticus, and electrolyte disorders are the leading causes. One of the rare causes that can be seen traumatically is the condition caused by electrical injury (2). Various life-threatening metabolic and inflammatory processes may develop secondary to necrosis in skeletal muscle cells. The most important points are effective fluid replacement, monitoring of renal functions and electrolytes, control of septic status, and close observation of compartment syndrome that may occur (3).

Case Presentation

A 26-year-old male patient was brought to the emergency department (ED) shortly after the incident (<1 hour) due to electrical damage as an occupational accident. It was learned that the incident occurred as a result of the patient coming into contact with the electricity lines (high voltage) connected to the city network while working with the solar energy water heating system on the roof. There was no history of additional trauma other than electrical damage. There was an unclear history of hypertension in his medical history. He was not using chronic medication. He was conscious and cooperative when he was admitted to ED. Arterial blood pressure: 160/100 mm Hg, pulse: 77/min, RR 18, and fever was 36.8° C. There were wounds on both hands, dorsal of the right foot, lateral and medial of the right leg due to electrical injuries (Figure 1). Laboratory values of the patient on admission and during follow-up are shown in Table 1. Electrocardiography was in sinus rhythm and was unremarkable. Foley catheter was inserted and dark colored urine was observed. Immediately, intravenous hydration with 0.9% NaCl was initiated after the patient was admitted to ED. Empirical antibiotherapy and daily wound care were performed. Amlodipine 5 mg 1 × 1 for hypertension, cefazolin 1 g 3 × 1 for skin infection and acetaminophen for pain control were administered. Urine output was around 4000-5000 cc per day. The patient's daily creatine kinase (CK) values were monitored as shown in Figure 2. Compartment syndrome did not develop. In the follow-up, the patient whose CK values progressively decreased and urine

output was sufficient did not develop renal dysfunction and/or hyperkalaemia. He was discharged on the 8th day of his hospitalization.

Discussion

It was first documented by Bywaters and Beall in the 1940s that there was a relationship between traumatic muscle injury and acute renal failure (4). The increase in the frequency of rhabdomyolysis cases in recent years has been observed significantly (5). Electrical damage constitutes a significant part of occupational accidents and it is stated that electrical burns constitute 27% of all burn patients in developing countries (6). High-voltage injuries (≥1000 volts) are known to have higher mortality and complications, and the associated AKI risk is further increased in this patient group. It is postulated that, in the course of rhabdomyolysis, AKI can be seen as high as 81.4%, rhabdomyolysis constitutes 7%-10% of all AKI cases, and mortality can reach 59% in rhabdomyolysis related AKI in intensive care unit (7). Due to its multisystemic potential adverse effects, it may cause mortality in the short term and serious morbidity in the long term. The damage it causes can be due to thermal or non-thermal effects. Some sources state that African Americans, male gender, morbid obesity, and being over 60 are risk factors for rhabdomyolysis. The clinical presentation of rhabdomyolysis is usually in the form of severe and widespread pain, tenderness, weakness in the muscles and dark urine. There may be a wide spectrum of symptoms/findings depending on etiological factors and complications such as electrolyte disturbances. It is characterized by the disruption of cell integrity in myocytes as a result of widespread damage to skeletal muscles and the passage of intracellular components into the circulation. Related to this, abnormalities such as hyperkalaemia, hyperphosphatemia, hypocalcemia, hyperuricemia, elevated liver function tests-aldolase-LDH, metabolic acidosis, lactic acidosis, and low fractional sodium excretion (<1%) can be seen in the laboratory. AKI is very common due to the nephrotoxic effect of myoglobinuria and the prerenal status. In this mechanism, the development of tubular obstruction and the effect of free oxygen radicals play an important role. Monitoring of CK and myoglobin has prognostic importance in the follow-up of rhabdomyolysis patients. Detection and monitoring of myoglobin may not always be possible due to its short half-life. It has been shown that the peak CK level can be used to predict AKI (8). CK is typically at least 5 times higher than the normal upper limit, usually >5000 U/L. However, there is no absolute cut-off value defined. The muscle-type creatine kinase (MM-CK) isoform rises significantly (about 95%), forming less of the creatine kinase myocardial band (CK-MB) isoform. It starts to increase within 2-12 hours from the beginning of the muscle damage, and reaches a maximum in 24-72 hours.

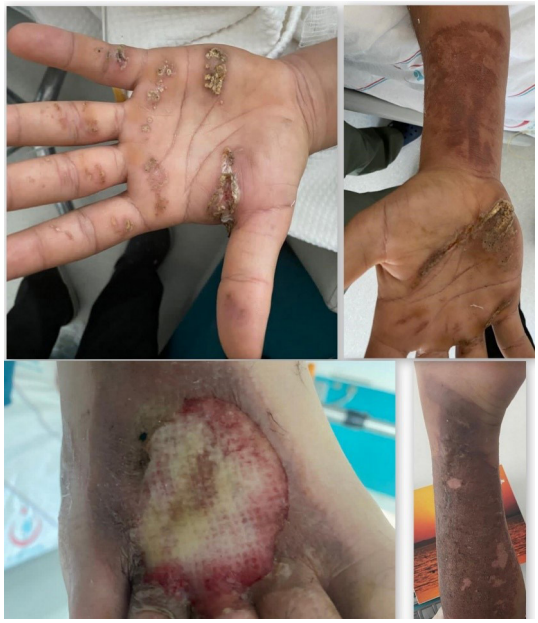

Figure 1. Electrical injury related wounds on different sites of the body

Table 1. Laboratory values on admission and during follow up

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 18
Creatinine (mg/dL)	1,1	0,94	0,91	1,02	0,96	1,03	0,86	1,1	0,96
Na (mmol/L)	135	141	138	143	141	140	138	137	140
Potassium (mmol/L)	3,65	3,7	3,9	3,98	4,4	4,4	4,3	4,4	4,5
Calcium (mg/dL)	8,94	8,73	8,79	n/a	n/a	9,3	9,6	n/a	9,6
Uric acid (mg/dL)	8,4	5,3	4,8	n/a	n/a	n/a	n/a	5,7	5,8
Albumin (gr/dL)	4,8	3,6	n/a	n/a	n/a	n/a	n/a	n/a	4,5
AST (U/L)	63	938	811	711	455	262	136	91	n/a
ALT (U/L)	39	232	260	248	193	171	145	124	49
LDH (U/L)	261	1334	963	814	424	353	301	296	179
Hemoglobin (g/dL)	15,7	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
HCO3	21	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
International normalized ratio	1.05	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a

AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase

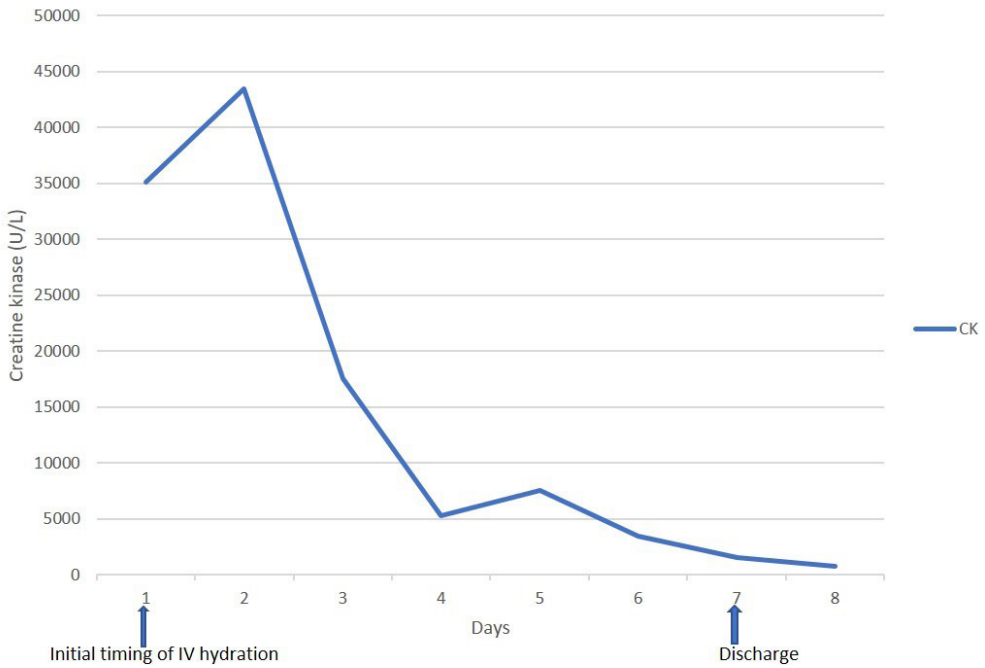

Figure 2. The course of serum creatinine kinase levels

A decrease is seen in 3-5 days. CK half-life is about 1.5 days and decreases at a relatively constant rate by about 40-50% of the previous day. Gradual reduction in CK levels indicates clinically favorable results. Local complications should be considered in the absence of the expected daily decrease or increase in CK levels. Despite CK values of > 40000 U/L in our patient, renal functions were not impaired at all. It is stated that the risk of developing AKI in rhabdomyolysis cases is proportional to the increase in CK levels. Yang et al. stated that when the CK level is over 10 000 U/L, the risk of developing stage II-III AKI (AKIN) is increased (7). Stewart et al. showed that the absolute CK level over 3925 U/L, increased the probability of stage II-III AKIN and death by more than 50% including the indication for renal replacement therapy. Interestingly in this study; electrical burn was associated with less AKI in the model (odds ratio (OR) = 0.43; 95% confidence interval (CI): 0.25–0.75; $P = 0.0026$) (9). In the literature, cases that were diagnosed histopathologically with AKI due to rhabdomyolysis, but did not show an increase in CK levels, as well as cases that did not develop AKI despite very high CK levels, were described rarely (10,11). It is important to keep in mind that these atypical presentations may appear in clinical practice. Although the maximum CK level was over 43 000 U/L in our case, AKI or hyperkalaemia did not develop. It is rare that AKI does not develop in patients with a severe increase in CK. Being young and initiating dynamic fluid replacement therapy as early as possible may be considered as an effective treatment. Apart from this, it is unclear whether some additional risk factors contribute to the development of AKI. Hyperkalemia is a serious complication in the course of rhabdomyolysis. It may even be necessary to perform hemodialysis several times a day due to hyperkalemia. Our patient had hypokalemia at the first presentation and hyperpotasemic course was not observed during the follow-up. Navarrete reported the frequency of hyperkalaemia as 1.2% in patients due to electrical damage and showed that there was no significant relationship between the degree of rhabdomyolysis and hyperkalaemia (12). Although hyperkalaemia is an urgent finding, it may not be present in some patients. Dark urine is indicative of pigment-related acute tubular necrosis. It can be seen in about half of rhabdomyolysis patients. The absence of dark colored urine does not rule out the diagnosis. In our patient, urine, which was dark in color for a certain period of time on the first day, became clear over time and urine microscopy was not performed. In addition to isotonic sodium chloride, mannitol infusion, alkaline therapy and hemodialysis may be used in the treatment. Our patient did not need other treatment options, since there was no acidosis at presentation and the urine output response was good with isotonic sodium chloride.

Conclusion

Electrical injury is relatively less common among the etiological factors of rhabdomyolysis. However, it is very important to start effective fluid therapy in a short time. Such an approach may reduce the risk of AKI in this patient group.

Authors' Contribution

Conceptualization: Alper Alp.
Methodology: Alper Alp, Bülent Huddam.
Validation: Bülent Huddam.

Formal Analysis: Burcu Arslan, Dilek Gibyeli Genek.
Investigation: Alper Alp, Burcu Arslan.
Resources: Alper Alp, Burcu Arslan.
Data Curation: Burcu Arslan.
Writing—Original Draft Preparation: Alper Alp, Dilek Gibyeli Genek.
Writing—Review and Editing: Alper Alp, Bülent Huddam.
Visualization: Dilek Gibyeli Genek, Supervision: Bülent Huddam.
Project Administration: Bülent Huddam.
Funding Acquisition: None.

Competing Interests
None.

Ethical Approval
This case report does not require ethical approval as it is not a human or animal research. The participant had given informed written consent to report the individual patient data.

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