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Traumatic rhabdomyolysis: Electrolyte changes

Murat Bicakcioglu

İnönü University Faculty of Medicine, Department of Anesthesiology and Reanimation, Division of Intensive Care, Malatya, Türkiye

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Abstract

Severe electrolyte disturbances occur in traumatic rhabdomyolysis (crush syndrome) developing after disasters such as earthquakes. The purpose of this study is to examine the long-term course of these electrolyte abnormalities. Patients followed up in intensive care for at least 10 days due to post-earthquake crush syndrome were included in the study. Their demographic data and sodium, potassium, calcium, and phosphorus levels over the course of 10 days were retrieved retrospectively and recorded. Fifteen out of 23 patients with crush syndrome were enrolled in the study. The dominant electrolyte abnormalities in the first three days of hospitalization were normonatremia, hyperpotassemia, hypocalcemia, and hyperphosphatemia. No change in normonatremia was observed at the end of the 10th day, but hyperpotassemia gave way to normo- and hypokalemia, and hyperphosphatemia to normophosphatemia. Although hypocalcemia was less prevalent and not as deep as in the first five days, it was still the dominant calcium disorder at the end of the 10th day. Crush syndrome can be linked to severe electrolyte abnormalities. These electrolytes may be hypo or hyper in form at the onset of crush syndrome but can reverse in the opposite direction, or an existing abnormality may become more profound during follow-up. Knowing the direction assumed by electrolyte abnormalities, and on which days, will therefore be more beneficial for patient management.

Keywords: Trauma, earthquake, rhabdomyolysis, crush syndrome, electrolyte

Introduction

Rhabdomyolysis developing in association with trauma is one of the most feared complications in natural disasters such as earthquakes [1]. Traumatic rhabdomyolysis is a syndrome characterized by muscle necrosis and the release into circulation of muscle cell contents, such as creatine phosphatase, myoglobin, and various electrolytes. Acute kidney injury (AKI) can develop following rhabdomyolysis. AKI can occur not solely through rhabdomyolysis, but also by means of sepsis, dehydration, and nephrotoxic agents. Mortality in rhabdomyolysis is generally associated with hyperkalemia in the acute period but with kidney failure and sepsis in subsequent periods [2]. Various electrolyte disturbances are observed post-rhabdomyolysis, such as hyperkalemia, hyperpotassemia, and hypokalemia. Electrolyte abnormalities are also frequently seen in earthquake victims [3]. Very few studies have examined electrolyte changes in earthquake victims with crush syndrome.

The purpose of this study was to determine electrolyte irregularities caused by crush syndrome in victims of the Kahramanmaraş earthquakes.

Material and Methods

Patients injured in the two 7.7 and 7.6 magnitude Turkish earthquakes on 2 February 2023, and followed-up for at least 10 days due to crush syndrome in the İnönü University Medical faculty intensive care unit (ICU) were included in the study. Approval for the study was granted by the Inonu University Health Sciences Non-Interventional Clinical Research Ethical Committee (2023/4837). The patients' demographic data, APACHE II and SOFA scores, heart rate, systolic, diastolic, and mean blood pressures, blood urea nitrogen (BUN), creatinine, creatine kinase (CK), myoglobin, lactate dehydrogenase (LDH),

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Corresponding Author: Murat Bicakcioglu, İnönü University Faculty of Medicine, Department of Anesthesiology and Reanimation, Division of Intensive Care, Malatya, Türkiye
Email: muratisin@msn.com

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and fluid intake and urine levels were retrieved retrospectively and recorded. In addition, the patients' sodium (Na), potassium (K), calcium (Ca), and phosphorus (P) values and associated electrolyte disorders over a 10-day period were retrieved retrospectively and recorded.

Crush syndrome was diagnosed on the basis of history, muscle mass being affected, compression (generally for 4-6 hours but potentially even for period less than one hour) and suppression of local circulation [4].

Biostatistical data analyses

Qualitative data were summarized as number (percentage) values, and quantitative data as mean±standard deviation and median (minimum-maximum) values. The Mann-Whitney U test, Pearson's chi-square test, Fisher's exact chi-square test, and the Mann Whitney U test were applied as appropriate. p values <0.05 were regarded as statistically significant. All analyses were carried out on IBM SPSS Statistics version 26.0 for Windows software (NY, USA).

Results

Fifteen of the 23 patients with crush syndrome admitted to our ICU because of the earthquake were included in the study due to hospitalization times of 10 days or longer. The patients' mean age was 31.6±14.9 years, and seven (46.6%) were male. Their clinical and laboratory data are shown in Table 1.

Table 1. The patients' clinical and laboratory data

Parametres	Mean (±SD)	Median (Min-Max)
Age (years)	31.6 (±14.9)	26 (14-58)
APACHE II	13.7 (±7.9)	15 (3-27)
SOFA	3.5 (±2.9)	3 (0-10)
Time spent under rubble (hours)	21.4 (±21.8)	13 (2-72)
Height (m)	1.7 (±0.1)	1.65 (1.55-1.84)
Weight (kg)	77.3 (±11.9)	75 (60-110)
BMI (kg/m^2)	27.2 (±3.2)	25.7 (23.4-32.5)
Heart rate (beats/min)	113.7 (±31.6)	110 (60-190)
SAP (mmHg)	126.3 (±22.5)	120 (100-170)
DAP (mmHg)	75.3 (±12.1)	80 (55-90)
MAP (mmHg)	92.3 (±14.1)	93.3 (70-116.7)
BUN (mg/dL)	31.4 (±20.5)	24 (9-87.4)
Creatinine (mg/dL)	2.4 (±1.9)	2 (0.6-8.3)
CK (U/L)	38954.6 (±48027.1)	25543 (1713-185100)
Myoglobin (U/L)	3311.5 (±1327.1)	4015 (520-4105)
LDH (U/L)	2991.3 (±5048.7)	1039 (373-16936)
Fluid intake (mL)	7042 (±3066.4)	5900 (3180-12900)
Urine (mL)	3840.7 (±2622.5)	3650 (200-10000)

BMI: body mass index, SAP: systolic arterial pressure, DAP: diastolic arterial pressure, MAP: mean arterial pressure, BUN: blood urea nitrogen, CK: creatine kinase, LDH: lactate dehydrogenase

Nine (60%) patients received renal replacement therapy.

The patients' Na, K, Ca, and P values and 10-day trends are shown in the Figure 1.

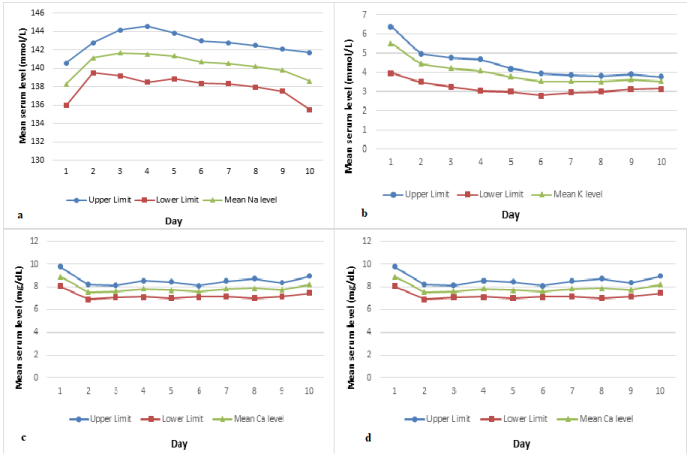

Figure 1. Daily changes in sodium, potassium, calcium, and phosphorus levels **1a.** Sodium **1b**. Potassium **1c.** Calcium **1d.** Phosphorus

Normonatremia was dominant on the first day of presentation (80%, 95% CI: 55.6-94), although sodium values then rose until the fifth day of follow-up, before again decreasing. Normonatremia was dominant over 10-day follow-up (Table 2).

Potassium values were hyperkalemic on the first day ilk. The values decreased from the second day, reaching normal values. A mild decrease was then observed until the sixth day, after which it followed a fixed course (Table 3).

The predominant change in calcium on the first day was in the form of hypocalcemia (46.7%, 95% CI: 23.7-70.6). The incidence of hypocalcemia then rose until the fifth day (100%, 95% CI 99-100), but then exhibited a decreasing tendency. However, the dominant electrolyte change over 10-day follow-up was hypocalcemia (Table 4).

Hyperphosphatemia was dominant on the first two days of follow-up. Values began declining as of the third day, and normophosphatemia was dominant on the 10th day (Table 5).

Table 2. Sodium and 10-day changes

Day	Hyponatremia		Normonatremia		Hypernatremia	
	Prevalence	95% CI	Prevalence	95% CI	Prevalence	95% CI
1	13.3	2.9-36.3	80.0	55.6-94	6.7	0.7-27.2
2	0.0	0.0 - 0.0	93.3	72.8-99.3	6.7	0.7-27.2
3	13.3	2.9-36.3	73.3	48.3-90.3	13.3	2.9-36.3
4	6.7	0.7-27.2	60.0	35.3-81.2	33.3	14.0-58.4
5	6.7	0.7-27.2	73.3	48.3-90.3	20.0	6.0-44.4
6	6.7	0.7-27.2	80.0	55.6-94.0	13.3	2.9-36.3
7	6.7	0.7-27.2	80.0	55.6-97.1	13.3	2.9-36.3
8	6.7	0.7-27.2	86.7	63.7-97.1	6.7	0.7-27.2
9	13.3	2.9-36.3	73.3	48.3-90.3	13.3	2.9-36.3
10	6.7	0.7-27.2	80.0	55.6-94	13.3	2.9-36.3

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Table 3. Potassium and 10-day changes

Day	Hypokalemia		Normokalaemia		Hypokalemia	
	Prevalence	95%CI	Prevalence	95%CI	Prevalence	95%CI
1	6.7	0.7-27.2	33.3	14.0-58.4	60.0	35.3-81.2
2	6.7	0.7-27.2	80.0	55.6-94.0	13.3	2.9-36.3
3	6.7	0.7-27.2	80.0	55.6-94.0	13.3	2.9-36.3
4	20.0	6.0-44.4	73.3	48.3-90.3	6.7	0.7-27.2
5	6.7	0.7-27.2	80.0	55.6-94.0	13.3	2.9-36.3
6	46.7	23.9-70.6	46.7	23.9-70.6	6.7	0.7-27.2
7	53.3	29.4-76.1	46.7	23.9-70.6	0.0	0.0-0.0
8	46.7	23.9-70.6	53.3	29.4-76.1	0.0	0.0-0.0
9	40.0	18.8-64.7	60.0	35.3-81.2	0.0	0.0-0.0
10	46.7	23.9-70.6	53.3	29.4-76.1	0.0	0.0-0.0

Table 4. Calcium and 10-day change

Day	Hypocalcemia		Normocalcemia		Hypercalcemia	
	Prevalence	95%CI	Prevalence	95%CI	Prevalence	95%CI
1	46.7	23.9-70.6	40.0	18.8-64.7	13.3	2.9-36.3
2	93.3	72.8-99.3	0.0	0.0-0.0	6.7	0.7-27.2
3	86.7	63.7-97.1	13.3	2.9-36.3	0.0	0.0-0.0
4	80.0	55.6-94.0	20.0	6.0-44.4	0.0	0.0-0.0
5	100.0	99.0-100.0	0.0	0.0-0.0	0.0	0.0-0.0
6	93.3	72.8-99.3	6.7	0.7-27.2	0.0	0.0-0.0
7	93.3	72.8-99.3	0.0	0.0-0.0	6.7	0.7
8	73.3	48.3-90.3	20.0	6.0-44.4	6.7	0.7
9	80.0	55.6-94.0	20.0	6.0-44.4	0.0	0.0-0.0
10	66.7	41.6-86.0	26.7	9.7-51.7	6.7	0.7

Table 5. Phosphorus and 10-day change

Day	Hypophosphatemia		Normophosphatemia		Hyperphosphatemia	
	Prevalence	95%CI	Prevalence	95%CI	Prevalence	95%CI
1	6.7	0.7-27.2	20.0	6.0-44.4	73.3	48.3-90.3
2	13.3	2.9-36.3	20.0	6.0-44.4	66.7	41.6-86.0
3	40.0	18.8-64.7	40.0	18.8-64.7	20.0	6.0-44.4
4	46.7	23.9-70.6	13.3	2.9-36.3	40.0	18.8-64.7
5	46.7	23.9-70.6	20.0	6.0-44.4	33.3	14.0-58.4
6	53.3	29.4-76.1	13.3	2.9-36.3	33.3	14.0-58.4
7	40.0	18.8-64.7	26.7	9.7-51.7	33.3	14.0-58.4
8	53.3	29.4-76.1	26.7	9.7-51.7	20.0	6.0-44.4
9	40.0	18.8-64.7	33.3	14.0-58.4	26.7	9.7-51.7
10	26.7	9.7-51.7	53.3	29.4-76.1	20.0	6.0-44.4

Discussion

The study findings revealed that hyperpotassemia, hypocalcemia, and hypophosphatemia are the dominant electrolyte disorders in surviving crush syndrome patients. In terms of sodium values, normonatremia was dominant.

In a study of 118 patients following the Bam earthquake, Safari et al. [3] identified hyponatremia, hypocalcemia, and hyperphosphatemia as the dominant electrolyte disorders. In another study involving potassium disorders after the Bam earthquake, Safari et al. [5] reported that hyperpotassemia was the dominant electrolyte change in the first days. He et al. [6] observed hyperkalemia, hypokalemia, hyponatremia, hypocalcemia, hyperphosphatemia, and hypophosphatemia, and reported rates of 34.0% for hyponatremia and 61.1% for hypocalcemia in patients with crush syndrome.

In a study of 228 patients, Zhang et al. reported an incidence of hyponatremia of 50.6%. Those authors concluded that hyponatremia is frequently observed in patients with crush syndrome and is associated with poor prognosis. Hyponatremia in such patients has been attributed to non-osmotic vasopressin release [7]. Patients can also be given large quantities of fluid, which may also lead to the development of hyponatremia. The patients in the present study received up to 12 liters of fluid. However, the incidence of hyponatremia was not as high as that reported in the previous literature. This may perhaps be attributable to only patients who survived and were followed-up for at least 10 days being enrolled in this study.

Hyperpotassemia is the most important problem to cause a risk of mortality in patients with crush syndrome [8]. Emergency hemodialysis occupies an important place in the management of hyperpotassemia in addition to fluid therapy and diuresis. Predictive factors examined when patients with crush syndrome on dialysis include anuria, fluid overload, and serum creatinine, BUN, and bicarbonate levels. In addition, potassium values exceeding 7 mg/dL are by themselves as predictive factor. Patients may require dialysis two or three times a day in the first 15 days. Prophylactic dialysis can also be applied due to hyperkalemia in patients with crush syndrome [9]. Hyperpotassemia generally predominates after injury, after which Hypopotassemia predominates in association with treatment and management. Hypopotassemia generally develops for reasons such as high levels of fluid resuscitation and urine alkalinization [5]. Although only surviving patients were included in the present study, hyperpotassemia was a dominant condition in all, but returned to normal values in the first five days, with a particularly rapid decrease in the first two days. Indeed, we observed no hyperpotassemia after the sixth day. Normopotassemia was present in more than 50% of the patients, the remaining 46.7% being hypopotassemic, figures consistent with the previous literature. Life-threatening hyperpotassemia in the first days then gave way to hypopotassemia. We think this may be ascribed to aggressively applied fluid therapy, urine alkalinization, and even intensive hemodialysis.

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The incidence of hypocalcemia was 46.7% on the first day, rising to 100% on the fifth day, followed by a continual decrease. All patients were asymptomatic for existing hypocalcemia. Initial hypocalcemia strengthens the cardiotoxic effect of hyperpotassemia [4]. Safari et al. [3] reported a hypocalcemia prevalence of 100%. Hypocalcemia may result from a high level of fluid resuscitation, urine alkalinization, and also hyperphosphatemia [10]. Calcium in cytoplasm associated with muscle cell breakdown also passes into the blood and causes a hypercalcemic effect in the late period [11,12].

The breakdown of muscle cells leads to hyperphosphatemia [13]. However, this not a life-threatening electrolyte disorder. Hyperphosphatemia is frequently observed in crush syndrome [14,15]. Hyperphosphatemia was also frequently observed in the present study but gave way to normophosphatemia over 10-day follow-up.

Our study was a single-center study, and this constitutes the most important limitation of the study. In addition, the number of patients is relatively small because it is a single-center study. this constitutes another limitation of the study.

Conclusion

In conclusion, severe electrolyte disturbances can occur in crush syndrome. While some of these are not life-threatening, others can lead to severe outcomes and even death. The results of this study showed that electrolyte abnormalities in the first days of follow-up of patients with crush syndrome can be replaced by abnormalities in the opposite direction with rapid aggressive treatment. Management should therefore be changed after the first five days in the treatment of electrolyte disturbance in crush syndrome.

Conflict of Interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Ethical Approval
Approval for the study was granted by the Inonu University Health Sciences Non-Interventional Clinical Research Ethical Committee (2023/4837).

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