Clinical Case Report

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Exertional rhabdomyolysis in a 21-year-old healthy man resulting from lower extremity training

A case report

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Abstract

Rationale: The incidence exercise-induced rhabdomyolysis is increasing in the healthy general population. Rhabdomyolysis can lead to the life-threatening systemic complications of acute kidney injury (AKI), compartment syndrome, and disseminated intravascular coagulopathy.

Patient concerns: A 21-year-old man had bilateral lower limb pain and soreness, dark brown urine after lower exremity training. Laboratory results showed that creatinine kinase (CK) and myoglobin (Mb) increased to 140,500 IU/L and 8632 μg/L respectively, with elevated liver enzymes, Scr, and proteinuria.

Diagnoses: Exercise-induced rhabdomyolysis with AKI.

Interventions: The patient was hospitalized and treated with vigorous hydration and sodium bicarbonate for 6 days.

Outcomes: After 6 days of treatment, the patient had a significant decrease in the CK and Mb levels. His renal function returned to normal. His laboratory tests had completely normalized during 2-week follow-up.

Lessons: Exercise-induced rhabdomyolysis can cause serious complications such as AKI. Delayed diagnosis can be critical, so timely manner should be taken to achieve a favorable prognosis.

Abbreviations: AKI = acute kidney injury, ALT = alanine aminotransferase, AST = aspartate aminotransferase, BUN = blood urea nitrogen, CK = creatinine kinase, ER = exertional rhabdomyolysis, KDIGO = Kidney Disease: Improving Global Outcomes, LDH = lactate dehydrogenase, Mb = myoglobin, Scr = serum creatinine.

Keywords: acute kidney injury, creatinine kinase, exercise, myoglobinuria, rhabdomyolysis

1. Introduction

Exertional rhabdomyolysis (ER) has been increasingly identified in the healthy general population after exercise.[1,2] Rhabdomyolysis is a syndrome caused by disruption of skeleton muscle with the release of muscle tissue content into the circulation that can lead to the life-threatening systemic complications of acute kidney injury (AKI), compartment syndrome, and disseminated intravascular coagulopathy.[3] Here we present a case of exercise-induced rhabdomyolysis caused by a lower extremity training.

1.1. Consent statement

Written informed consent was obtained from the patient for the publication of this study.

2. Case report

A 21-year-old man presented to the emergency department with a 2-day history of lower extremity pain and soreness and 1 day of gross hematuria. He reported that the pain began the 2nd day after he went to the gym doing lower extremity exercise training. The day prior to his presentation, the patient developed gross hematuria, without back pain and fever. He reported no history of previous medical conditions or medications.

Physical examination showed muscle tenderness but no edema.

Urinalysis showed protein 2+ and urine occult blood +/−. Laboratory work-up demonstrated a creatinine kinase (CK) of 140,500 IU/L, alanine aminotransferase (ALT) of 316 IU/L, aspartate aminotransferase (AST) of 1319 IU/L, and myoglobin (Mb) of 8632 μg/L. His blood urea nitrogen (BUN) and serum creatinine (Scr) were 7.38 mmol/L and 94 μmol/L, respectively.

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FP and XL contributed equally to this study.

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Table 1

Laboratory values during hospitalization and follow-up.

Variable	Hospital admission	Day 1	Day 3	Day 6	Follow-up (3 wks after admission)
CK, IU/L	124,600	75,866	27,624	2750	220.4
CK-MB, IU/L	n/a	572.5	187.5	40.2	13.1
AST, IU/L	1279	950	521	97	19
ALT, IU/L	304	296	235	129	14
Mb, IU/L	6062	1594.0	226.4	84.4	<21
LDH, IU/L	n/a	1304.5	513.9	291	205.7
α-HBDH, IU/L	n/a	533	357	n/a	n/a
Scr, μmol/L	94	57.2	61.9	71	70
BUN, mmol/L	7.38	4.99	3.73	3.1	3.45

ALT = alanine aminotransferase; AST = aspartate transferase; BUN = blood urea nitrogen; CK = creatine kinase; CK-MB = creatine kinase-MB; Mb = myoglobin; LDH = lactate dehydrogenase; α-HBDH = α-hydroxybutyrate dehydrogenase; Scr = serum creatinine.

Based on his markedly elevated CK, myalgia, and myoglobinuria, he was diagnosed and hospitalized with exercise-induced rhabdomyolysis. After treatment with vigorous hydration and sodium bicarbonate, his pain improved and his CK, ALT, AST, Mb, BUN, and Scr decreased to 124,600 IU/L, 304 IU/L, 1279 IU/L, 6062 μg/L, 4.99 mmol/L, and 57.2 μmol/L, respectively. His urine color returned to normal without urinalysis abnormalities. After 6 days of hydration, his CK, ALT, AST, Mb, and lactate dehydrogenase (LDH) decreased to 2750 IU/L, 129 IU/L, 97 IU/L, 84.4 μg/L, and 291 IU/L (Table 1), respectively. So the patient was discharged.

By 2 weeks postdischarge, his Scr was 70 μmol/L. His CK level, liver enzymes and other laboratory tests had normalized.

3. Discussion

Lower extremity exercise training in gyms requires strenuous leg movements such as kick-back, squats, and prone leg bending with body-building apparatus. The heat in the gyms, dehydration, and the excessive muscle activities increase susceptibility to rhabdomyolysis.[3] Moreover, recurrence to rhabdomyolysis is thought to be related to specific genetic defects and more than 60 monogenic genes have been found associated with rhabdomyolysis.[4] Increasing cases have reported ER caused by conditioning such as spin and crossfit.[5,6] Meanwhile, reports showed low-intensity exercise[7] and low-load high-repetition resistance exercise[8] can also cause ER.

Our patient had a presentation of elevated CK of 140,500 IU/L, myoglobinuria, and myalgia. Clinically, a triad of symptoms was described including myalgia, weekness, and dark brown urine.[9] Although there is not a clearly consistent cutoff threshold of serum CK level, a concentration 5 times the upper limit of normal, ranging from 1500 to over 100,000 IU/L is used by most clinicians.[10] Detection of Mb in urine may be absent in 25% to 50% of patients with rhabdomyolysis due to the rapid clearance of Mb, and Mb levels also decrease rapidly in patients with renal failure.[11,12] Over 50% patients with rhabdomyolysis did not have myalgia.[13] Chavez et al conducted a systematic search and indicated that clinical studies mostly diagnosed rhabdomyolysis based on CK levels and not usually took symptoms as diagnostic references.[10] A consensus criteria of rhabdomyolysis definition is needed to support clinical studies and diagnosis.

The Scr of our patient was 94 μmol/L at admission, and decreased to a baseline value between 60 and 70 μmol/L during hospitalization and follow-up visits. Initially, his Scr increased by ≥0.3 mg/dL (≥26.5 μmol/L) within 48 hours. According to Kidney Disease: Improving Global Outcomes,[14] any item that conforms to the following can be defined as AKI: Scr increased by ≥0.3 mg/dL (≥26.5 μmol/L) within 48 hours; Scr increased to 1.5 times over the baseline value, which is known or presumed to have occurred within the previous 7 days; urine output <0.5 mL/(kgh) for 6 hours. Based on the guideline, he had AKI. He was diagnosed 2 days after exercise and treated with early vigorous hydration, and did not develop subsequent severe kidney injury. Although the Scr value can be within the normal range of the general, the baseline value is different for each individual and possible AKI may be overlooked. Thus, timely diagnosis of AKI is necessary.

The AKI is rarely reported in young ER patients without underlying renal diseases. Alpers and Jones found that the lower incidence of AKI in patients with ER vs rhabdomyolysis from other causes in a retrospective cohort.[15] In addition, Kenney et al reported that patients with exertional rhabdomyolysis were younger, generally healthier, had higher elevation of CK than those who developed rhabdomyolysis from other etiologies, but had a lower incidence of severe complications.[16] But some cases progressed rapidly and violently. Bhalla and Dick-Perez reported a man with rhabdomyolysis and evidence of renal insufficiency, developed bilateral compartment syndrome and renal failure requiring dialysis.[17] Therefore, it is important to diagnose AKI timely.

Some studies reported the predictors of AKI and mortality in patients with rhabdomyolysis. It is reported that initial Scr levels were associated with progression to AKI and mortality at 30 days.[18] A study indicated that the value of serum Mb was a more sensitive marker of acute myoglobinuric kidney injury and considered 15 to 20 mg/L as an appropriate Mb cutoff.[19] A retrospective study suggested that the Mb/CK ratio more than 0.2 was related to the increased development of AKI.[20] Some studies found that serum CK levels had been regarded as a predictor of AKI and mortality in severe rhabdomyolysis with elevated serum CK levels of more than 10,000 IU/L.[21,22] However, some conflicted results were reported. Clarkson et al presented data on measures of renal function (potassium, osmolality, BUN, Scr, phosphorus, and uric acid) in 203 subjects who performed 50 maximal eccentric contractions of the elbow flexor muscles.[23] None of these participants developed AKI, though 111 of them had CK values at 4 days postexercise >2000 IU/L, and 51 had values >10,000 IU/L. Another retrospective cohort analysis showed that CK levels did not predict mortality.[18] McMahon et al proposed a risk prediction score to identify high-risk patients with rhabdomyolysis.[24] The risk prediction score model

included age, sex, race, cause of rhabdomyolysis, and independent risk factors such as initial Scr, CK >4000 IU/L, calcium, bicarbonate, phosphate, and inpatient laboratory work-up. It was validated among more than 2000 patients with rhabdomyolysis and turned out that a score of <5 was of low risk of renal replacement therapy or in-hospital mortality, whereas a score of >10 was of high risk. It needs more studies to confirm these.

Clinically, major management of rhadomyolysis is fluid therapy with NaCl 0.9%.[13] It relieves the obstruction of Mb casts by improving blood flow and glomerular filtration rate to prevent AKI.[25] Mannitol and acetaminophen were also recommended for the prevention of AKI.[26] Prescribing bicarbonate into regimen can correct and avoid electrolyte balance disorder.[13] These were confirmed very useful in our patient. Our patient was treated with early vigorous replacement with saline and bicarbonate to improve urine pH and urine output, and received favorable prognosis.

The recurrence of well-healed young patients with ER is low.[15] It has been shown that destructed muscle fibers regenerate after a few weeks and grow well with good circulation to the necrotic regions.[27] Some studies presented cases of recrudescence, especially in patients with underlying sickle cell trait, malignant hyperthermia, carnitine palmitoyltransferase II deficiency, or other hereditary diseases.[28,29] To enable patients return to sport without recurring ER, Schleich et al outlined a 4-phase progressive program implemented to successfully return each athlete to sport after an ER diagnosis requiring hospitalization.[30] Athletes returned to activities of daily living for 2 weeks after discharge in phase I and were not allowed to train until the CK level was below 5 times normal (1000 IU/L). Then, it moved forward to phases II to IV, in which recovery training began from low-load exercise to high-resistance training. No athletes in the program had a relapse of rhabdomyolysis and it suggested an individual regime based on patients' improvement of physical and laboratory indicators.

Global exercise is a trend, and anyone participates in sports is likely to develop ER. To improve the prognosis of ER, we hope that there will be more authoritative consensus in diagnosis, more effective, and accurate prediction system of AKI.

Author contributions

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