

Exercise and Acute Kidney Injury, a Rare Combination

Egzersiz ve Akut Böbrek Hasarı, nadir bir kombinasyon

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Abstract

Rhabdomyolysis is a common cause of acute kidney injury(AKI). Although it has been previously described in exercise-induced rhabdomyolysis, few cases have been reported, usually in athletes and in the soldiers training in army school. We define a case of rhabdomyolysis induced by exercise in a 19-year-old male patient who is a medical student and a farmer. Early and aggressive fluid therapy could prevent renal damage without the need for renal replacement therapy in rhabdomyolysis-induced AKI.

Key words: Exercise, Kidney Failure, Crush Syndrome

Özet

Rabdomiyoliz, akut böbrek hasarının sık görülen bir nedenidir.Daha önce egzersize bağlı rabdomiyoliz olarak tarif edilmesine rağmen, genellikle sporcular ve ordu okullarında eğitim alan askerlerde bildirilmiştir. Biz bir tıp öğrencisi ve çiftçi olan 19 yaşındaki bir erkek hastada egzersizle indüklenmiş rabdomiyoliz olgusunu sunuyoruz. Erken ve agresif sıvı

tedavisi rabdomiyoliz kaynaklı akut böbrek hasarında renal replasman tedavisine gerek kalmadan böbrek hasarını önleyebilir.

Anahtar kelimler: Egzersiz, Böbrek yetmezliği, Crush sendromu

Introduction

Rhabdomyolysis refers to the dissolution of muscle fibers. In the medical literature, it is defined as a pathological condition characterized by destruction of muscles in which intracellular enzymes such as creatine phosphokinase (CK), lactate dehydrogenase, aldolase, myoglobin, and electrolytes are extensively released into the circulation. In general asymptomatic increase of the enzymes and AKI are the presentations in the clinical ground(1).

Etiology is miscellaneous. The most commonly reported causes are drugs, alcohol abuse, muscle diseases, trauma, convulsions, and immobilization. Vigorous exercise is a rare cause in the published series(2). Marathon running, mountain climbing, weight lifting, and strenuous military training are among the reported cases. CK elevation and myoglobinemia after heavy physical activity are common conditions without serious complication(3). The development of AKI caused by rhabdomyolysis due to exercise seems to be rare in literature(4). We present a young patient with AKI associated with rhabdomyolysis. He is also a medical student working as a farmer during holidays.

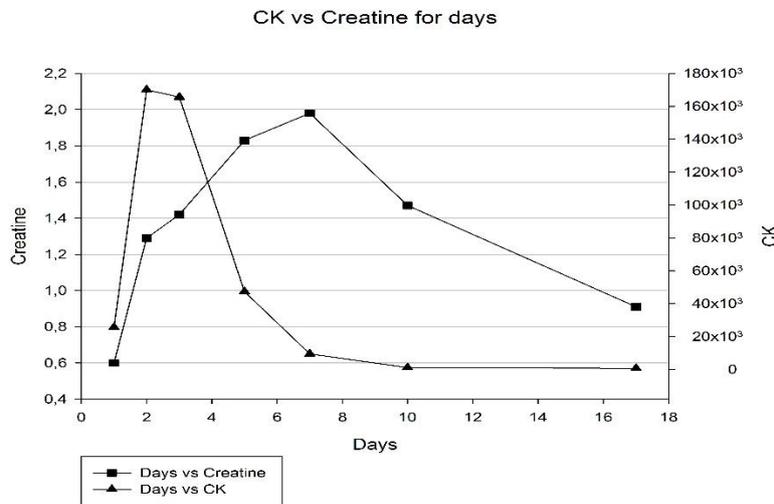
Case report

18-year-old male patient was admitted to the emergency department complaining leg pain, fatigue, and dark urine. There was no history of previous disease and hereditary disorder. He did not use alcohol, cigarettes, and any drugs. In the physical examination, the body temperature of 37.8°C, pulse rate of 88/minute, arterial blood pressure of 130/80 mm Hg was measured. Heart and lung examination revealed no pathological findings. Muscle tenderness was detected in both lower extremities. The patient stated that he had worked in the field for about 12 hours in hot weather the day before. In laboratory parameters; white blood cell count 16,300 / L, hemoglobin 14 g / dL, platelets 255.000/L, INR 1.17, glucose 91 mg/dL, creatinine 0.9 mg / dL, BUN 19 mg / dL, AST 2900 U / L, ALT 540 / L, ALP 124 U / L,

albumin 3.4 g / dL, calcium 9.9 mg / dl, phosphorus 4.3 mg / dL, sodium 138 mmol / L, potassium 4 mmol / L, CK-MB 4944 U / L (0-25), LDH 2701 U / L (140-280), CK 25 596 U / L (29-200) were obtained. In his urine analysis; pH 6, protein 3+ were detected with light red color urine, 30 erythrocytes were seen on urine microscopy without dysmorphism. HBsAg, anti-HBsAb, anti-HCV and anti-HIV were negative, pH 7.36; PCO₂ 46.6 mm Hg, HCO₃ 25.8 mmol / L were in the arterial blood gas analysis. Spot urine protein / creatinine ratio, parathyroid hormone and TSH were 0.60 mg / mg, 80.3, 1.95 mIU / L, respectively. CRP 31.6 mg/L, sedimentation 2 mm/h were measured. There were no pathological findings on chest radiography. Echocardiography showed normal left ventricular functions. In abdominal ultrasonography; liver, gallbladder, pancreas, and spleen were normal, right kidney size 128X52 mm, left kidney size 130X54 mm, parenchymal thickness were reported as normal. Renal artery and vein pathology were not detected in the Doppler examination.

Initially, despite being considered an acute nephritis, the patient was hospitalized with a diagnosis of rhabdomyolysis in the nephrology service due to increased CK values and the presence of strenuous exercise history.

Daily 7000 cc fluid hydration and furosemide infusion was started. Urine was alkalinized with 50 mmol / day of sodium bicarbonate. The amount of urine began to increase after the first three days in which he was oliguric. Creatinine and CK levels decreased and came back to normal (Graph 1). The patient was discharged without any complication when the kidney function had normalized.

Graph 1. Creatinine and CK levels

CK: creatine phosphokinase (U/L), Creatine mg/dL

Discussion

Whatever the etiology of rhabdomyolysis, pathophysiological events end at an ordinary point. Na / K ATPase pump protects the negative gradient of the cell membrane pumping out the intracellular sodium. Calcium pumps also prevent the rise of pathological levels of intracellular calcium. All these mechanisms are dependent on the energy. ATP deficiency that occurs in severe exercise leads to disruption of these mechanisms. Accumulation of intracellular sodium triggers the increase of calcium concentration. Intracellular calcium activates the proteolytic enzymes leading to cell destruction thereby excessive amounts of potassium, aldolase, phosphate, myoglobin, aspartate transaminase pass into the bloodstream(1).

Rhabdomyolysis may be seen in normal individuals with strenuous physical activity. Creatine kinase elevation, myoglobinemia, and myoglobinuria have been identified in marathon runners and soldiers in the military training class. The development of AKI in these individuals, however, is not common. Physical inactivity, exercise in warm and humid weather, NSAID use and dehydration increase the risk of rhabdomyolysis(5,6). Different mechanisms are associated with AKI due to rhabdomyolysis such as hypovolemia,

intraluminal obstruction by myoglobin, uric acid casts, direct myoglobin toxicity, renal ischemia secondary to muscular vasoconstrictors, and production of free radicals (7-9). Rhabdomyolysis and AKI developed in our patient on a hot summer day after strenuous physical activity in an area that made farming.

The primary symptoms of exercise-induced muscle damage are pain, tenderness, swelling and stiffness. Pain after exercise begins within hours intensified in 48 hours. Although the pain symptoms are not severe, compartment syndrome has been reported in some cases(10). It was undeveloped except for pain in our case.

Although the correlation between CK and myoglobin values, CK values were not significantly associated with the risk of developing AKI in studies. A CK level has been shown to be elevated even in very light exercise. AKI did not develop in the majority of cases even in CK levels of 80000. In fact, according to some authors, renal failure from severe exercise alone is required the presence of factors such as genetic predisposition and environmental heat stress(10).

Exercise type also plays a significant role in the suggested risks related to rhabdomyolysis. It has been reported that eccentric type of activities had a higher likelihood of muscle damage. Mountain climbing, plyometric exercises, weight lifting are examples of this kind of activity(6). We observed the intense eccentric type of activity in our case.

If AKI occurs due to rhabdomyolysis, supportive treatment should be more intense and long lasting. Fluid resuscitation should be initiated early to prevent the development of AKI especially in patients with CK values above 20000 U/L(11). Fluid deficit should be corrected very quickly, and 2.5 ml / kg / h should be continued so that 2 ml / kg / hour urine output should be provided. Many experts recommend the urine alkalization and diuretic therapy, but the effectiveness of these approaches has not yet been proven(12,13).

Consequently, rhabdomyolysis should be considered in the differential diagnosis of AKI, and essential investigation must be made with the aim of complete recovery that can be achieved without hemodialysis through early and vigorous fluid treatment.

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