Acute Renal Failure due to Non-Traumatic Rhabdomyolysis

Non-travmatik Rabdomyolize Bağlı Akut Böbrek Yetmezliği

Nontravmatik Rabdomyoliz / Non-Traumatic Rhabdomyolysis

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Özet

Rabdomiyoliz iskelet kasının travmatik ve nontravmatik nedenlere bağlı olarak görülen klinik ve biyokimyasal sendromudur ve kelime olarak kas erimesi anlamını taşımaktadır. Ezilme tipi travmayı takiben gelişen rabdomiyoliz (Crush sendromu) nadir görülen ancak ABY etyolojisinde iyi tanınan bir klinik durumdur. Nontravmatik rabdomiyoliz ise nadirdir. Bu yazıda akut böbrek yetmezliği (ABY) kliniğinde başvurarak rabdomiyoliz tanısı konulan ve tekrarlayan diyaliz uygulanan bir olgu sunulmaktadır.

Anahtar Kelimeler

Akut Böbrek Yetmezliği; Diyaliz; Nontravmatik; Rabdomiyoliz

Abstract

Rhabdomyolysis is a musculoskeletal clinical and biochemical syndrome which is seen associated with traumatic and non-traumatic causes and is known as muscular dystrophy. Rhabdomyolysis which develops following crush-type trauma (Crush syndrome) is rarely seen but is a well-known clinical event in the etiology of acute renal failure. Non-traumatic rhabdomyolysis is rare. The case is here presented of a patient who was diagnosed with rhabdomyolysis on presentation with acute renal failure and to whom repeated dialysis was applied.

Keywords

Rhabdomyolysis; Non-Traumatic; Dialysis; Acute Renal Failure

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Introduction

Rhabdomyolysis is known as a syndrome which destroys the integrity of the sarcolemma as a result of musculoskeletal damage from traumatic or non-traumatic reasons [1]. In 5-7% of cases that develop acute renal failure, musculoskeletal breakdown is responsible [2]. Reasons in the etiology include medications such as colchicine, lithium and statins, hereditary muscle enzyme deficiencies, trauma, viral infections, excessive exercise and hyothyroidism [3]. The aim of this case presentation was to draw attention to the necessity of keeping in mind the diagnosis of rhabdomyolysis in a patient with no trauma history who presentedwith acute renal failure.

Case Report

An 11-year old male presented at the pediatric emergency polyclinic with the complaint of dark brown coloured urine which had been ongoing for 4 months. From the history it was learned that the patient had had diarrhoea for one week and on the day of presentation had started to experience pain on walking and difficulty standing. In the physical examination, the patient was conscious and co-operative with a weak appearance, pulse 98/min, respiratory count 24/min, and TA 120/85 mmHg. The eyeballs were sunken, the skin had reduced turgor and there was a herpetic lesion on the lower lip. In the apex 3/6 systolic murmur could be heard. Deep tendon reflexes were normo-active and muscle strength in the lower and upper extremities was 3/5. Values in the full blood count were Hb: 11 g/dL, PLT: 136.000/µL, WBC: 6600/µL, RBC: 6.3x106/µL, MCV: 85 fl, MCHC: 34 g/dL, RDW: 13% and in the peripheral blood smear, thrombocyte aggregation, leukocyte and erythrocyte morphology was normal and there was 5% reticulocytosis. In the full urine test, density was 1035, protein 3+ and abundant erythrocytes were determined in microscopy. In the biochemical tests, the values were determined as BUN 20 mg/dL, creatinine 1.28 mg/dL, uric acid 10.5 mg/dL, sodium 123 mmol/L, potassium 5 mmol/L, phosphorus 8 mg/dL, AST 6953 U/L, ALT 721 U/L, lactic dehydrogenase (LDH) 331.910 U/L, creatinine clearance 13.5 mL/min. Complements (C3, C4) and IgA values were within the normal range. Prothrombin time was determined as 15.1 secs, aPTT 62.3 secs, fibrinogen 281 mg/dL, D-Dimer 1278 (normal değer) ng/ml, and the direct Coombs test was negative. Blood gas values were pH 7.42, pCO2 29, HCO3 20.9, and BE -5.1.

The patient was admitted to the pediatric intensive care unit with findings of acute renal failure (ARF), thrombocytopenia and impaired liver function tests. The echocardiographic evaluation was found to be normal, the size of the kidneys was normal on the urinary system ultrasonography and there was seen to be increased parenchyma echogenity (consistent with Grade 2 parenchymal disease). Three times (20 cc/kg) 0.9% NaCl was administered intravenously because of hyponatremic dehydration. Together with parenteral fluid therapy and diuretic treatment, treatments for the raised levels of uric acid and phosphorus were started. Under observation, the patient developed hyperkalemia (K+: 6.3 mg/dL) and hypocalcemia (Ca++: 6.7 mg/dL), which recovered with the administration of 2 cc/kg iv calcium gluconate.

From the history and the acute muscle weakness determined in the physical examination and the determination of creatine kinase (CK) 38.350 U/L, blood and urine myoglobin >3000 ng/mL, the clinical table was thought to be consistent with rhabdomyolysis. There were no findings of hemolysis in the peripheral smear and under observation the thrombocyte count did not decrease. Blood electrolytes recovered with fluid treatment but creatine continued to rise and urine output continued to fall. In the kidney biopsy, acute tubular damage was determined consistent with rhabdomyolysis. The thyroid functions were normal and in viral serology, EBV, CMV, HSV, HIV and toxoplasma were found to be negative, perinuclear anti-neutrophil cytoplasmic antibody (p-ANCA) negative, antinuclear antibody negative, cytoplasmic anti-neutrophil cytoplasmic antibody (c-ANCA) and anti-ds-DNA negative. Proteinuria was determined at 64 mg/m2/hour in 24 hours of urine. Four times hemodialysis and pulse steroid treatments were administered to the patient. Polyuria which had developed under observation receded and renal functions returned to normal. With improvement of the clinical and laboratory values, the patient was administered oral steroid treatment, referred to the polyclinic for follow-up and was discharged.

Discussion

Following damage to the striated muscle cells, intracellular elements pass into systemic circulation resulting in the clinical and laboratory findings of rhabdomyolysis. This situation can show variations from a table of temporary hypocalcemia, hyperkalemia, increased creatin kinase and myoglobin as far as a Crush syndrome table characterised by hypovolemic shock, cardiac arrest and ARF. When muscle cells are damaged, proteins such as myoglobin, CK, aldolase, LDH, potassium, and AST and cellular components apart from proteins are expressed into the plasma [2, 4]. Determination of myoglobin at high levels in the serum may be an indicator of early stage damage in the muscle or the muscle membrane.

Rhabdomyolysis may occur for traumatic reasons such as earthquakes, electric shocks or mining accidents or for many different reasons such as alcoholism and medication use (especially statins), excessive physical activity, long-term lack of movement, epileptic seizures, hyperthermia, hypothermia, infections, electrolyte imbalances (especially hypokalemia, hypophosphatemia). Rhabdomyolysis is also seen in many congenital diseases of the metabolism, in glycolytic enzyme deficiency diseases, in some diseases with abnormal lipid metabolism and in malignant hyperthermia [2].

Frequently seen complications of rhabdomyolysis are cardiac arrest, hypovolemia, arrythmia, compartment syndrome, widespread intravascular coagulation and ARF. As the most common complication, ARF develops associated with impaired renal perfusion and myoglobin damage. Of all ARF cases, 5-25% are known to be associated with rhabdomyolysis and approximately 10-40% of rhabdomyolysis cases develop ARF. The most significant laboratory finding of rhabdomyolysis is the increased level of serum myoglobin. With a normal plasma level of <0.003 mg/dL, myoglobin is completely cleared from the plasma in approximately 6 hours with a very short half life of 1-3 hours. As the half life is short, the level is generally found to be normal on presentation [5]. In healthy individuals, as approximately 50-85% is associated with plasma globulins, the transfer of myo-

globin to the urine is negligible. Myoglobinuria has been seen to be much greater in muscle mass which has suffered necrosis. The main factors affecting the formation of myoglobinuria are the level of blood myoglobin, the glomerular filtration rate and urine flow rate [6]. Although the current patient had impaired kidney function, the myoglobin level in the urine was found to be >3000 ng/mL.

Sometimes rhabdomyolysis may develop without any trauma and this is known as non-traumatic rhabdomyolysis. The current case was thought to be non-traumatic rhabdomyolysis and the fluid loss due to diarrhea and reduced oral intake were considered to have facilitated acute renal failure.

The most practical and meaningful test in the diagnosis of rhabdomyolysis is an increase in creatin kinase showing muscle damage. As 70% of potassium in the body is found in the muscles, severe hyperpotassemia may be seen in rhabdomyolysis cases. In the current case potassium was determined at 6.1 mg/dL. While the ARF table is initially prerenal character in rhabdomyolysis patients, at an advanced stage, acute tubular necrosis occurs [7]. In the current patient, despite sufficient appropriate fluid treatment, oliguria continued suggesting acute tubular necrosis.

In patients developing ARF asociated with Crush syndrome, it is not necessary to perform a kidney biopsy in all cases but in cases that do not start the recovery process of ARF, a biopsy can be performed to ascertain the underlying cause. Due to the raised levels of creatinine in the current case, kidney biopsy was performed and acute tubular necrosis associated with rhabdomyolysis was determined.

Conclusion

Rhabdomyolysis, which develops for various reasons and becomes evident with widesprread muscle damage, must be kept in mind for patients presenting with ARF table even if there is a history of trauma. It should not be forgotten that despite appropriate treatment, it may lead to 2-5% mortality [8].

Competing interests

The authors declare that they have no competing interests.

References

1. Vanholder R, Sever MS, Erek E, Lameire N. Rhabdomyolysis. J Am Soc Nephrol 2000;11:1553-61.
2. Ward MM. Factors predictive of acute renal failure in rabdomyolisis. Arch Intern Med 1998;148:1553-7.
3. Melli G, Chaudhry V, Cornblath DR. Rhabdomyolysis: an evaluation of 475 hospitalized patients. Medicine (Baltimore) 2005;84:377-85.
4. Dönmez O, Meral A, Yavuz M, Durmaz O. Crush Syndrome of children in the Marmara earthquake, Turkey. Pediatr Int 2001;43: 678-82.
5. Palmer MS. Hemoglobin and myoglobin induced acute renal failure in rats. Role of iron in nefrotoxicitiy. Am J Physiol 1988;255:539-44.
6. Knochel JP: Rhabdomyolysis and myoglobinuria. Semin Nephrol 1981;1:75-86.
7. Torres PA, Helmstetter JA, Kaye AM, Kaye AD. Rhabdomyolysis: pathogenesis, diagnosis, and treatment. Ochsner J 2015;15(1):58-69.
8. Rosa NG, Silva G, Teixeria A, Rodrigues F, Araujo JA. Rhabdomyolysis Acta Med Port 2005;18:271-81.

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