

# Acute Tubuler Necrosis Related to Rhabdomyolysis

## *Rabdomiyolize Bağlı Gelişen Akut Tübüler Nekroz*

### ABSTRACT

Rhabdomyolysis is a clinical and laboratory syndrome due to traumatic or non-traumatic injury that leads muscle cell contents participation into circulation. Dehydration and acidosis may cause myoglobinuric acute renal failure in patient with rhabdomyolysis. This case presents a 27-year-old male referred to emergency unit with weakness and abdominal ache who has a story of urine decrease and trauma exposure. Diagnosis of rhabdomyolysis in this case highlights the importance of anamnesis in early diagnosis and treatment.

**KEY WORDS:** Rhabdomyolysis, Acute renal failure, Emergency

### ÖZ

Rabdomiyoliz; travmatik veya nontravmatik olarak çizgili kas hasarına bağlı hücre içeriğinin dolaşıma katılması sonucu oluşan klinik ve laboratuvar sendromdur. Rabdomiyoliz hastalarında dehidratasyon ve asidoz miyoglobürik akut böbrek yetmezliğine neden olabilir. Bu yazıda; 27 yaşında acil servise halsizlik, karın ağrısı şikayeti ile başvuran, hikayesi sorgulandığında travmaya maruz kaldığı ve idrar miktarının azaldığı öğrenilen, yapılan değerlendirme sonucu rabdomiyoliz tanısı alan bir erkek olgu sunularak, anamnezin tanıdaki önemi ile erken tanı ve erken tedavinin önemi vurgulanmak istenmiştir.

**ANAHTAR SÖZCÜKLER:** Rabdomiyoliz, Akut tübüler nekroz, Acil

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### INTRODUCTION

Rhabdomyolysis (RML) is the name of clinic and laboratory syndrome caused by an injury which damages the integrity of the sarcolemma of skeletal muscle, leads to the release of potentially toxic muscle cell components into the circulation (1, 2). Clinical features is a variable. It can be watch as the asymptomatic enzyme elevation or a life-threatening electrolyte imbalance causing to renal failure. The most common trauma causes are, alcohol abuse, muscle compression with heavy exercise, viral infection, toxins (1, 2). The treatment of rhabdomyolysis is elimination of the causes of muscle damage. Intravenous hydration must be initiated as early as possible, provision of adequate urine output with diuretics and urine alkalization (1, 3).

### CASE REPORT

A 27-year-old male patient referred to emergency department with weakness and decreasing at his urine. His medical history included no properties. After the detailed anamnesis of patient, we learned that he was assaulted four days ago, he had urine decreasing in last 2 days and also concentration of urine, vomiting and abdominal pain. Vital signs were stable. He is physical examination was normal but he had pain at his whole body with palpation.

For diagnosis of rhabdomyolysis and acute renal failure blood tests gave the following results: serum creatinine (Cr) 8.81 mg/dl (Normal: 0.84-1.25) Ure:168 mg/dl (17- 43) , aspartate aminotransferase (AST): 196 U/l (0-35) creatine kinase (CK) 32442 U/l (0-171), CK- MB :775 U/l (0- 24),



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serum potassium (K<sup>+</sup>) 4,4mEq/L (3.5-5.2). Urinalysis revealed myoglobinuria, urine color is dark, a pH of 5, 2+ protein, 3+ blood and negative for blood cells.

The patient was held in observation with the diagnosis of rhabdomyolysis. The treatment initially, we started normal saline at a rate of 1.5 L per hour, next 2.5 ml/kg/ per hour infusion, so that the urine pH alkaline range was (> 6.5) NaHCO<sub>3</sub> (132 mEq/day intravenous infusion) and furosemide infusion was begun (100 mg/day intravenous infusion) and forced diuresis. Patient's fluid balance, serum electrolytes, serum Cr and urinary pH, check for 12 hours after initiation of treatment blood tests gave the following results: Cr:9.41 mg/dl, Ure:177 mg/dl, AST: 130 U/l, CK: 21980 U/l, CK- MB :291 U/l , K<sup>+</sup> :4.29mEq/L. Patient was consulted nephrology department and underwent emergency hemodialysis.

After the patient was admitted to the nephrology clinic he underwent hemodialysis for two consecutive days. Patient's Cr and CK levels aimed to decrease so we monitored him by intravenous hydration and urine alkalization. After twelve days his serum levels returned to normal values, vital signs stable and urine output normalized, he discharged from the hospital.

## DISCUSSION

RML is defined as a clinical and laboratory syndrome resulting from the damage of striated muscles with subsequent release of muscle cell contents into the plasma. These contents include myoglobin, CK, aldolase, lactate dehydrogenase, AST and K<sup>+</sup> (1,2,3,4). Myoglobin is freely filtrated by the glomerulus and has a direct toxic effect on tubular epithelial cells. The heme protein in the content of myoglobin induces lipid peroxidation and renal damage. Renal damage in rhabdomyolysis occurs by the common effect of factors like hypovolemia, renal vasoconstriction, heme protein-induced direct toxicity, and tubular obstruction. Therefore, hydration and urine alkalization are important in preventing the development of acute renal failure (ARF) (5-7).

There are about 26,000 known rhabdomyolysis cases in the United States (4). Rhabdomyolysis-induced ARF prevalence and ARF hospitalization in the United States are reported to be 4-33% and 5-15%, respectively (4). The classic presentation of rhabdomyolysis includes myalgia, weakness, red brown urine caused by myoglobinuria, and elevations in muscle enzymes such as CK (2). As its presentation with characteristic physical symptoms has been reported only in 4-15% of the patients, their absence does not exclude diagnosis (3). Weakness, muscle pain, and darkening of urine color were present in our patient. Patient history is usually very helpful in diagnosis. The patient's history of decrease in urine amount, darkening of urine colour, and muscle pain after exposure to blunt trauma supported the diagnosis. The increase in the plasma myoglobin level is the most reliable test in RML diagnosis (2). Myoglobin elevation occurs before CK

elevation after muscle injury and then is rapidly cleared from the plasma through renal excretion and metabolism to bilirubin. Myoglobin enters the urine when the plasma concentration is >1.5 milligrams/d L and causes the typical reddish brown discoloration when urine myoglobin level is >100 milligrams/ Ml. Because myoglobin contains heme, qualitative tests such as the dipstick test(which uses the orthotoluidine reaction) do not differentiate among hemoglobin, myoglobin and red blood cells. Therefore, myoglobinuria should be suspected when the urine dipstick test is positive for blood, but no red blood cells are present on microscopic examination. Radioimmunoassay is slightly more sensitive than the dipstick technique in identifying myoglobinuria, but usually is not necessary. Because myoglobin levels may return to normal within 1 to 6 hours after the onset of muscle necrosis, the absence of an elevated serum myoglobin level or of myoglobinuria does not exclude the diagnosis. In one study, only 19% of patients with rhabdomyolysis had myoglobinuria (1). The most practical method in diagnosis is the increase in the CK level. RML diagnosis is made when serum CK level is found to be five times higher than the normal level (2). Post-trauma CK, serum Cr and K<sup>+</sup> are not included in routine tests in trauma patients. However, in such conditions, it should be taken into account that CK and K<sup>+</sup> values may be elevated due to muscle enzymes and cell destruction. In this way, following rhabdomyolysis, ARF development may be prevented by an appropriate treatment. Although the muscle mass exposed to trauma is important for RML and associated CK level, there is no exact predictive value for the development of acute tubular necrosis (ATN) in these patients (1). Serum CK and K<sup>+</sup> values, independent from the muscle mass exposed to trauma, may reach levels that are likely to cause ATN. RML and secondary ARF treatment includes early and sufficient hydration, urine alkalization and force diuresis (1, 3, 4). Our patient had developed ARF when he applied to the hospital. Still, a controlled hydration was applied and urine alkalization was performed. Hemodialysis was administered when the patient did not respond to these treatments and presented elevated Cr values. The lack of response to the medical treatment is considered to be associated with the fact that the patient presented to the emergency room after he developed acute tubular necrosis.

## CONCLUSION

In patients who present to emergency units for trauma; CK, Cr and K<sup>+</sup> values should be examined, regardless of the affected muscle mass, and early treatment should be initiated to prevent possible complications in cases with high values. Patients with normal laboratory values in acute period should be properly and sufficiently informed at discharge about possible conditions that may develop after trauma. In this way, early diagnosis of rhabdomyolysis can be made and it may be treated before life threatening complications such as acute renal failure and electrolyte disorders develop.

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