

A case of rhabdomyolysis due to immobilization and compression

To the Editor,

Rhabdomyolysis is a complex clinical picture characterized by damage to skeletal muscles, participation of intracellular elements in the systemic circulation and clinical and laboratory findings which develop in relation (1). Rhabdomyolysis has traumatic and non-traumatic causes. Traumatic causes include earthquakes, traffic and mine accidents, electric shock, excessive exercise and staying in certain positions for a long time. In the pathogenesis of rhabdomyolysis related to traumatic causes, compression on the muscle (baromyopathy) has a major role. The clinical picture ranges from transient mild hyperpotassemia and increased creatinine phosphokinase (CK) level to "Crush" syndrome characterized by life-threatening shock, cardiac arrhythmias and acute renal failure (ARF) (2). Here, a pediatric case of rhabdomyolysis and related acute renal failure possibly due to an uncomfortable and long travel is presented.

A seven-year-old male patient presented with complaints including weakness and pain in the arms and legs, inability to walk and dark colored urine a few hours after an overnight bus trip during which he was stuck between two people. His personal and familial histories were natural. His physical examination was normal except for a decrease in muscle strength and deep tendon reflexes predominantly in the lower extremities and marked myalgia. The initial laboratory tests revealed a urinalysis as follows: density: 1029, pH:6, protein (-), blood (+3), leucocytes (-), nitrite (-), no cells on microscopic examination. Complete blood count, erythrocyte sedimentation rate and C-reactive protein were found to be normal. Biochemical tests were as follows: alanine aminotransferase 668 IU/L, aspartate aminotransferase 3264 IU/L, CK 242 694 IU/L, blood urea nitrogen (BUN) 48 mg/dL, creatinine 1.9 mg/dL, albumin 1.9 g/dL, electrolytes: normal.

A diagnosis of acute renal failure related to rhabdomyolysis was made because of increased muscle enzyme levels and renal dysfunction in the patient who had weakness and myalgia

which started suddenly. Hydration and alkalization treatment was started. Since renal dysfunction increased gradually, hypertension developed and urine output decreased in the follow-up, hemodialysis was started. In addition, mechanical ventilation was started, since respiratory distress developed and blood gases deteriorated gradually. Since no traumatic cause including traffic accident, electric shock or excessive exercise was found, thyroid function tests, autoimmune and viral serology, urinary and blood amino acid chromatography and carnitine and homocysteine levels were investigated in the patient for conditions which might have caused rhabdomyolysis and found to be normal. Electromyography revealed primary muscle fiber involvement and muscle biopsy revealed an appearance compatible with rhabdomyolysis. Rhabdomyolysis secondary to staying in the same position for a long time during an uncomfortable trip a short time before the complaints began was considered and the treatment was continued.

Hemodialysis and mechanical ventilation treatment were discontinued, when renal function tests and blood pressure returned to normal and respiratory findings improved in the follow-up. The patient was discharged in a short time with a good general status.

Rhabdomyolysis develops due to traumatic or non-traumatic causes (3). In the pathogenesis of rhabdomyolysis which develops as a result of traumatic causes, baromyopathy has a major role. In baromyopathy, the permeability of the sarcolemma is disturbed, substances including potassium, myoglobin, CK and creatinine which are found in high amounts in the muscle get outside the cell, while sodium, chloride, water and calcium flow into the cell. Thus, edema is developed in the cell. This edema leads to compartment syndrome clinically (1). Rhabdomyolysis due to a traumatic cause was considered in our patient who had increased potassium, muscle enzymes and creatinine because of history of a long trip during which the patient was stuck in the same position.

In traumatic rhabdomyolysis, systemic findings show variance in different stages of the disease, though local

symptoms (myalgia, muscle weakness, muscle rigidity) and pain are predominant. These systemic findings include hypotension, shock, cardiac arrhythmia, cardiac and respiratory failure, hypovolemia and acute renal failure (3,4,5,6). Our patient had muscle weakness and developed acute renal failure and respiratory failure in the follow-up.

The most common laboratory findings in patients with rhabdomyolysis include increase in muscle enzymes, myoglobinuria, findings of prerenal-renal failure, anemia, leucocytosis, thrombocytopenia, hyperpotassemia, hyperphosphatemia, hypocalcemia, hypoalbuminemia and metabolic acidosis (6,7). However, a serum CK level 5-fold higher than the normal value is enough for a diagnosis of rhabdomyolysis (2,8). Our patient had increased muscle enzymes (CK level 5-fold higher than the normal value), increased potassium, BUN and creatinine levels, myoglobinuria and hypoalbuminemia.

In rhabdomyolysis, one of the most important complications which affects the prognosis is acute renal failure. Even if acute renal failure develops, complete recovery is the rule unless the patient is lost as a result of complications including hyperpotassemia, heart failure and infections and no persistent damage is expected in the kidneys in the long-term (9). In our patient, acute renal failure regressed in a short time.

Conclusively, trauma is important in the etiology of rhabdomyolysis as well as infections and toxic causes. Trauma is not necessarily severe. As observed in our case, it may occur

as a result of an uncomfortable interurban travel and lead to serious problems including acute renal failure.

Hurşit Apa, Meral Torun Bayram, Hasan Ağin, Ceyhun Dizdarer

Dr. Behçet Uz Pediatric Diseases and Pediatric Surgery Education and Research Hospital, İzmir, Turkey

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