

## Case Report

# Status epilepticus related rhabdomyolysis and acute kidney injury

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## ABSTRACT

Rhabdomyolysis is a syndrome characterized by muscle necrosis and the release of intracellular muscle constituents into the circulation. Creatine kinase levels are typically markedly elevated, and muscle pain and myoglobinuria may be present. Its causes are variable. Acute kidney injury is a common complication of rhabdomyolysis. Our patient is a 51-year-old male who was brought to the hospital with a complaint of generalized tonic-clonic seizures and weakness. His creatine phosphokinase levels increased on the first day of admission to 93,809 U/l. Creatinine level was found to be 9.13 mg/dl in the first biochemistry test. He was diagnosed to have an acute kidney injury and rhabdomyolysis. Hemodialysis was not performed to patient. Sixteen days after first admission, the patient's rhabdomyolysis resolved.

**Keywords:** Rhabdomyolysis, Status epilepticus, Acute kidney injury

## INTRODUCTION

Rhabdomyolysis is characterized by the leakage of muscle-cell contents, including electrolytes, myoglobin, and other sarcoplasmic proteins (e.g., creatine kinase, aldolase, lactate dehydrogenase, alanine aminotransferase, and aspartate aminotransferase) into the circulation. Massive necrosis, which is manifested as limb weakness, myalgia, swelling, and commonly, gross pigmenturia without hematuria, is the common denominator of rhabdomyolysis. The mechanisms involved in the pathogenesis of rhabdomyolysis are direct sarcolemmic injury (e.g., trauma) or depletion of adenosine-tri-phosphate within the myocyte, leading to an unregulated increase in intracellular calcium.<sup>1</sup> Status epilepticus, formerly known as grand mal seizures, comprise two stages: a tonic phase and a clonic phase. These intense seizures can be frightening to experience or observe, as extreme muscle spasms may temporarily arrest breathing. It can rarely cause rhabdomyolysis.<sup>2</sup> Severity of illness ranges from asymptomatic elevations

in serum muscle enzymes to life-threatening disease associated with extreme enzyme elevations, electrolyte imbalances, and acute kidney injury. The most important causes of acute and chronic renal diseases are diabetes mellitus, hypertension and glomerulonephritis.<sup>3,4</sup> Acute kidney injury is the most serious complication of rhabdomyolysis and it may be life-threatening. Anemia and thyroid dysfunctions can be coexist with renal injury.<sup>5,6</sup> Cause of rhabdomyolysis are trauma, muscle hypoxia, thyroidal dysfunctions, genetic defects, infections, body temperature changes, metabolic and electrolyte disorders, drugs, toxins and idiopathic.

## CASE REPORT

A 51 year old man was admitted to this hospital because of weakness and seizure. He had history of hypertension, acute rheumatic fever and tonic-clonic seizure. He took phenytoin and carbamazepine twenty years ago but then stopped after three years. He did not use alcohol, tobacco, over-the-counter or herbal medications, or illicit

substances. His family history was unremarkable. The patient appeared normal weight. The body temperature was 36.7°C, the pulse 67 beats per minute, the blood pressure 98/54 mm Hg, the respiratory rate 16 breaths per minute, and the oxygen saturation 97% while he was breathing ambient air. He had dark urine. The abdomen was protuberant but soft and nontender. Neurologic examination was notable for reduced strength in legs and upper arms. Ankle dorsiflexion and plantar flexion were normal bilaterally. Other systemic examinations of the patient were normal. On admission the serum creatinin kinase (CK) level was 93809 U/l, C-reactive protein- 122.5 mg/l, potassium- 5.2 mmol/l, phosphorus- 7.65 mg/dl, calcium- 6.56 mg/dl, sodium- 128 mmol/l, creatinine- 9.13 mg/dl, urea- 190.2 mg/dl, uric acid- 14.5 mg/dl, alanine aminotransferase- 191 U/l, and aspartate aminotransferase- 457 U/l. On arterial blood gas pH was 7.37, pCO<sub>2</sub>- 40.6 mmHg, HCO<sub>3</sub><sup>-</sup>- 23.1 mEq/l. Urine parameters: color amber, clarity slightly cloudy, protein 1+, blood 3+, glucose negative, erythrocytes 38 and leukocytes 4 per high-power field on the urine test. The white-cell, platelet counts and levels of hemoglobin, magnesium, globulin, bilirubin, alkaline phosphatase, thyrotropin, and antinuclear antibodies were normal. The patient was consulted with the neurology department. Electroencephalography was applied to the patient. Epileptogenic activity was not detected. Levetiresetam and sodium valproate were started. The patient was consulted with the hematology department. No schistocytes were observed in peripheral smear. Microangiopathic hemolytic anemia was excluded. Consultatant of nephrology did not require dialysis for patient. He was diagnosed to have an acute kidney injury and rhabdomyolysis. The patient initiated volume repletion with normal saline promptly at a rate of approximately 400 ml per hour. Patient's creatinine levels began to drop over days. Urinary color improved to normal. The seizure did not recur after the first episode. The patient was discharged after 16 days. The patient had no complaints and his physical examination was normal. Levetirecetam was admister to 500 mg twice daily. One week later, all examinations were normal.

## DISCUSSION

Rhabdomyolysis is a condition that is characterized by the destruction of skeletal muscle and the spillage of its contents into the bloodstream. These include mainly sarcoplasmic proteins (creatine phosphokinase, lactate dehydrogenase), myoglobin, and electrolytes.<sup>7</sup> The etiology of rhabdomyolysis is variable. It's classic triad of myalgia, fatigue, and dark-colored urine. Acute kidney injury is an important complication of rhabdomyolysis. It may be life threatening. Acute kidney injury incidence in rhabdomyolysis is variable. Its ranges from 13% to 50%.<sup>8</sup> In some studies, the rate of acute renal failure due to rhabdomyolysis is 46%.<sup>9</sup> Rhabdomyolysis-induced acute kidney injury causes oliguria and occasionally causes anuria. The electrolyte abnormalities of rhabdomyolysis are hyperkalemia, hyperphosphatemia, hyperuricemia,

high anion-gap metabolic acidosis, and hypermagnesemia.<sup>10</sup> It's characteristic form different from other forms of acute tubular necrosis is low fractional excretion of sodium.<sup>11</sup> Hyperkalemia can be life-threatening in patients with severe traumatic rhabdomyolysis. Rhabdomyolysis that is associated with acute kidney injury needs rapid and urgent treatment. The main step of this condition is aggressive fluid replacement. It's amount depends on the severity of the rhabdomyolysis.<sup>12</sup> Rhabdomyolysis following grand mal seizures is rarely observed in the literature. There was some case reports about this subject.<sup>13</sup> Epilepsy is a common condition, affecting 1% of the population, and it has been shown that a degree of rhabdomyolysis commonly occurs in generalized seizures.

In this case, we conclude that since convulsions may cause rhabdomyolysis and myoglobinuria. It is important to be careful when the patient come with seizure and high CK level. Serial measurements of CK may determine the early stage of acute kidney injury. Increased creatinine level requires closer monitoring for acute kidney injury. Being alert and acting early in treatment is the most important step to reduce the risk of complications.

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