

Report of a Case with Status Epilepticus Associated Rhabdomyolysis

Status Epilepticus İlişkili Rabdomiyoliz Olgusunun Sunumu

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Abstract

Status epilepticus is one of the important neurological emergencies due to the high rates of morbidity, mortality and workforce loss it causes. Status epilepticus is a condition that needs to be recognized and treated quickly because it is both life-threatening and causes serious sequelae. Status epilepticus can lead to Rhabdomyolysis, although it is a rare occurrence. In this case report, a forty-one-year-old female patient with a history of epilepsy is presented. She had eight generalized tonic-clonic seizures within seven hours. At the time of admission, the patient's creatinine kinase level was measured as 11434 U/L. Creatinine kinase level decreased with hydration therapy. No acute kidney injury occurred. As a result, patients with status epilepticus are accompanied by muscle damage. Especially patients at risk for acute kidney injury should be closely monitored for creatinine kinase and urine output.

Keywords: Status epilepticus, rhabdomyolysis, tonic-clonic seizures

ÖZ

Status epileptikus, yüksek morbidite, mortalite ve iş gücü kaybına neden olan önemli nörolojik acil durumlar arasında yer almaktadır. Status epileptikus, hem yaşamı tehdit edici hem de ciddi sekellerle sonuçlanabilen bir durum olduğu için hızlı bir şekilde tanınması ve tedavi edilmesi gereken bir durumdur. Status epileptikus, nadir bir sonuç olmasına rağmen Rabdomiyolizise yol açabilir. Bu vaka raporunda, epilepsi öyküsü olan kırk bir yaşındaki bir kadın hasta sunulmaktadır. Hasta, yedi saat içinde sekiz generalize tonik-klonik nöbet geçirdi. Yatış anında, hastanın kreatin kinaz seviyesi 11434 U/L olarak ölçüldü. Hidrasyon tedavisi ile kreatin kinaz seviyesi azaldı. Akut böbrek yetmezliği oluşmadı.

Sonuç olarak, status epileptikuslu hastalarda kas hasarı eşlik edebilir. Özellikle akut böbrek yetmezliği riski altındaki hastaların kreatin kinaz ve idrar çıkışı açısından yakından izlenmesi önemlidir.

Anahtar Kelimeler: Status epileptikus, rabdomiyoliz, tonik-klonik nöbet

Highlights

- Status epilepticus is a life-threatening condition.
- Even though rhabdomyolysis is a rare consequence of epilepsy, it should not be forgotten.
- Early diagnosis and treatment of rhabdomyolysis may prevent kidney failure.

Introduction

Status epilepticus stands out as a critical neurological emergency, contributing significantly to morbidity, mortality, and productivity loss. As with epilepsy, the causes of the status vary. It may develop after acute brain injury or as a symptom of epilepsy. Status epilepticus is a condition that needs to be recognized and treated quickly because it is both life-threatening and causes serious sequelae (1). Status epilepticus is divided into three subtypes: tonic-clonic, focal SE with impaired consciousness, and absence. For diagnosis, two time intervals have been defined. The time to initiate emergency treatment, t1, is within 5-15 minutes. The onset of neurological consequences is determined to occur within 30-60 minutes (2).

Rhabdomyolysis (Rb) is the release of toxic muscle content into the circulation as a result of damage to striated muscles due to traumatic or non-traumatic causes and destruction of muscle tissue (3). While drugs, muscle diseases and neuroleptic malignant syndrome are prominent among non-traumatic causes, status epilepticus is among the rare causes (4).

In this case report, we aimed to discuss the clinical course and treatment approach of Rb caused by status epilepticus in the light of current literature.

Case presentation

A forty-one-year-old female patient presented to our clinic reporting seven seizures within a six-hour period. Her medical history revealed a background of epilepsy and generalized anxiety disorder, and she was regularly taking quetiapine (300 mg), alprazolam (0.5 mg), and levatiracetam (500 mg) daily. Importantly, it was discovered from the patient's relatives that she had not been adhering to her medication regimen for the past week. The physical examination on admission indicated a blood pressure of 128/78 mmHg, temperature of 37.1°C, and a heart rate of 78 beats/minute. The patient exhibited full muscle strength in both lower extremities with no sensory or motor deficits, but appeared prone to sleep, consistent with a postictal state. No abnormalities were noted in other system examinations.

In the examinations performed, hemoglobin 11.3 g/dl (12.0-15.5 g/dl), white blood cell 24.7 $10^3/\mu\text{l}$ ($4.0-11.0 \times 10^3/\mu\text{L}$), platelet 304 $10^3/\mu\text{l}$ ($150-450 \times 10^3/\mu\text{L}$), alanine transferase 17 U/L (7-56 U/L), aspartate transferase 26 U/L (5-40 U/L), alkaline phosphatase 125 U/L (30-120 U/L), gamma glutamyl transferase 55 U/L (9-48 U/L), lactate dehydrogenase 1016 U/L (140-280 U/L), sodium 135 mEq/L (135-145 mEq/L), calcium 9.05 mEq/L (8.5-10.5 mEq/L), potassium 4.35 mEq/L (3.5-5.1 mEq/L), blood urea nitrogen 13.6 mg/dL (7-20 mg/dL), creatinine 0.83 mg/dL (0.6-1.3 mg/dL), C-reactive protein 41 mg/dL (0-5 mg/dL), creatine kinase (CK) 11434 U/L (26-192 U/L), blood gas parameters were within normal limits. In the urinalysis, the color of the urine was light yellow and clear, and no erythrocytes were seen. Cranial computed tomography and magnetic resonance imaging were within normal limits.

The patient had a generalized tonic-clonic seizure lasting 10 minutes in the first hour of follow-up in the emergency department. Diazepam 5 mg was administered intravenously. It was observed that the seizure activity stopped. Levatiracetam was administered at a dose of 30 mg/kg. The patient was diagnosed with seizure-related Rb and started on intravenous fluid therapy.

During the follow-up, there was no decrease in the amount of urine, acidemia did not develop, kidney function tests were within normal limits and the complaints decreased. At the thirty-fourth hour of his treatment, CK decreased to 507 U/L. The patient was explained that he needed to take his medications and was discharged.

Discussion

Epilepsy is a disease that requires the patient to be prepared for physical changes, compliance with daily medication use, recurrent medical examinations and acute medical emergencies, and negatively affects the quality of life (5). An important clinical presentation of epilepsy is status epilepticus. Status epilepticus is an important cause of mortality and morbidity in epilepsy patients. In status epilepticus, ischemia and permanent neuronal damage may occur in the brain tissue due to the inability to meet the excessively increased oxygen demand in the brain tissue during excessive neuronal activity (6). Additionally, the lack of respiratory effort during a tonic-clonic seizure results in ventilation failure. Lack of ventilation causes widespread tissue hypoxia. Lactate, the most well-known biomarker of tissue hypoxia, is used to differentiate seizures from pseudo-seizures due to this pathogenesis. Similar to neuronal tissue, increased oxygen consumption experienced during tonic-clonic contractions in muscle tissue causes ischemia in muscle tissue. Considering the lack of ventilation, we reveal the cornerstones that explain muscle damage in status epilepticus (7).

Rhabdomyolysis is a syndrome characterized by muscle necrosis and the release of intracellular muscle components into the circulation. Causes of Rb include drugs, toxins, infections, muscle trauma, convulsive seizures, hyperthermia, electrolyte imbalances, muscle enzyme defects, cocaine and alcohol use (9). Symptoms of Rb usually have an acute onset and include myalgia, stiffness, weakness, malaise, low-grade fever, and dark (usually brown) urine. However, symptoms related to the musculoskeletal system are observed in only half of the cases (10). In severe Rb, symptoms such as nausea, vomiting, abdominal pain and tachycardia may be observed. In some cases, mental status changes may occur secondary to urea-induced encephalopathy. In a small number of cases, edema, tenderness in the affected muscle groups and hemorrhagic discoloration of the overlying skin may be observed. Muscle edema may not occur until rehydration with intravenous fluids. Depending on the cause, the affected muscle groups may be localized or widespread. Postural muscles of the thighs, spine and lumbar region

are usually affected. Acute Rb may develop without any of these signs and symptoms, and the patient's physical examination findings may be normal. Therefore, the diagnosis is usually made by detailed anamnesis (recent cocaine use, etc.), detection of increased serum CK levels, or detection of myoglobinuria in routine laboratory tests (11).

Although prolonged tonic-clonic seizure was held responsible for the basic pathogenesis in our case, it is also mentioned in the literature that antiepileptics may be the cause of Rb. Especially levatiracetam stands out in terms of Rb (12). The rationale for not considering antiepileptic-related Rhabdomyolysis in our case was the concurrent elevation of tissue hypoxia markers and the subsequent decrease in CK levels following the seizure.

Conclusion

Ppatients experiencing status epilepticus are susceptible to muscle damage. Vigilant monitoring of CK and urine output is particularly crucial, especially for patients at risk of acute kidney injury.

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