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【 CASE REPORT 】

Exertional Rhabdomyolysis Combined with Acute Kidney Injury and Complicated by Posterior Reversible Encephalopathy Syndrome

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Abstract:

Posterior reversible encephalopathy syndrome is an uncommon syndrome in exertional rhabdomyolysis. We herein report a case of rhabdomyolysis and acute kidney injury after intense exercise. The patient also had generalized hypertension, headache, and painless complete loss of vision in both eyes, which was consistent with the symptoms of posterior reversible encephalopathy syndrome detected by magnetic resonance imaging of the brain. The patient fully recovered through active fluid replacement, diuresis, blood pressure control, alkalization of urine and blood purification, and supportive measures. This case report describes an infrequent neurological complication of exertional rhabdomyolysis. Understanding the range of complications is critical for improving patient outcomes.

Key words: acute kidney injury (AKI), exercise, posterior reversible encephalopathy syndrome (PRES), rhabdomyolysis

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Introduction

Exertional rhabdomyolysis (ER) is mainly caused by the destruction of skeletal muscle cells due to high-intensity and repetitive exercise and the release of cell contents into the bloodstream, leading to a series of clinical signs and symptoms (1). In addition, exercise-induced rhabdomyolysis can cause serious complications, such as acute kidney injury (AKI) (2). Rhabdomyolysis has been reported frequently for different reasons. However, exercise-induced rhabdomyolysis with posterior reversible encephalopathy syndrome (PRES) is relatively rare. PRES is a clinical syndrome characterized by headache, seizures, various visual abnormalities, and psychiatric abnormalities. Nevertheless, its pathogenesis is not fully understood.

Case Report

A 24-year-old previously healthy man was admitted to our hospital. His major complaints were diffuse myalgia and weakness for one week, dark-colored urine for six days, and headache, painless complete vision loss in both eyes, and anuria for three days. One week before admission, he had engaged in a large amount of exercise. The main sports were long-distance running and rock climbing. He exercised for 5 days, lasting 6-7 hours a day. Three days before admission, the patient suddenly had a headache characterized by paroxysmal fullness and distending pain that resolved spontaneously after 2-3 minutes each time. The headache worsened around 17:00 on the same day, accompanied by sudden painless complete loss of binocular vision and one instance of vomiting. He then consulted a clinic. He had no history of drug or alcohol consumption and had never had hypertension.

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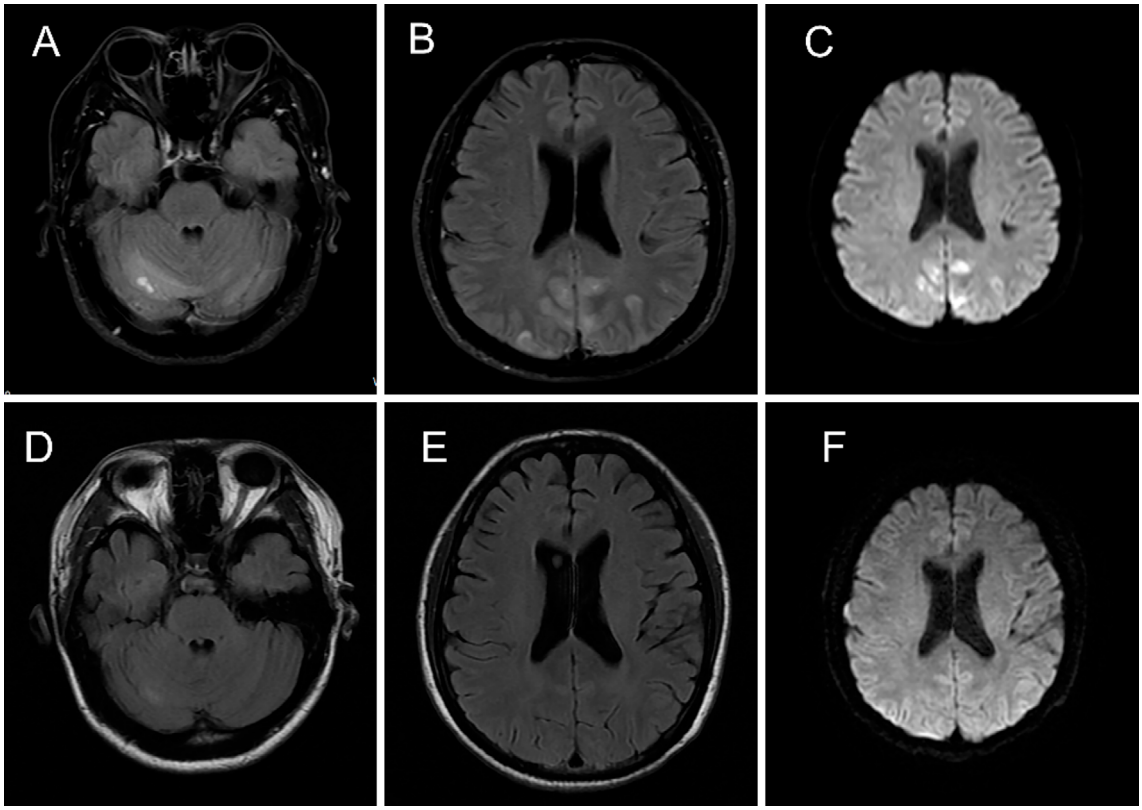

Figure. **Magnetic resonance imaging (MRI) T2 fluid attenuated inversion recovery (FLAIR) images showed high signal intensities involving mainly the subcortical white matter. (A) Parietal and occipital lobe areas. (B) Cerebellar hemisphere area. Diffusion weighted imaging (DWI) images showed a high signal on the parietal and occipital lobes (C). Brain MRI showed that the lesion almost disappeared on the parietal and occipital lobes (D, F) and in the cerebellar hemisphere (E).**

Table. **Laboratory Values in Serum during Hospitalization.**

Variable	Day 1	Day 3	Day 5	Day 7	Day 9
Cr (μmol/L)	1,551	675	863	284	413
BUN (mmol/L)	57.6	27.4	16.5	10.6	8.7
CK (U/L)	5,800	3,071	689	255	180
Mb (ng/mL)	>1,200	242.3	140.6	108.9	108,6
CK-MB (U/L)	144.8	92	23.8	14.2	14.2
BNP (pg/mL)	14,225	8,701	8,583	3,004	1,200

Cr: creatinine, BUN: blood urea nitrogen, CK: creatine kinase, Mb: myohemoglobin, CK-MB: creatine kinase isoenzyme, BNP: brain natriuretic peptide

His blood pressure was 156/112 mm Hg. He was completely blind in both eyes on a physical examination. However, wet rales could be heard in both lower lungs. The rest of the cardiovascular and respiratory examinations were regular, as was the abdominal examination. His consciousness was clear, he was able to walk and stand normally, with no abnormalities noted on a cranial nerve examination. The Glasgow Coma Scale score was 15/15. The laboratory tests showed an elevated white blood cell count (12.59×10^9/L), and elevated levels of urea nitrogen (56.1 mmol/L), creatinine (1,422 μmol/L), creatine kinase (5,800 U/L) (normal range: 50-310 U/L), creatine kinase isoenzyme (150 U/L) (normal range: 0-24.0 U/L), myoglobin (1,200 ng/mL) (normal range: 17.4-105.7 ng/mL), and B-type natriuretic peptide assay (14,225 pg/mL). We diagnosed him with ER and AKI based on heavy exercise, diffuse myalgia, weakness, anuria, and markedly elevated levels of creatine kinase, creatinine, and myoglobin. Urgent brain magnetic resonance imaging (MRI) showed multiple areas of increased signal intensity in the bilateral temporal, occipital, parietal, and cerebellar lobes on T2-fluid-attenuated inversion recovery (FLAIR) imaging. On diffuse-weighted imaging, the signal lesions showed limited diffusion (Figure). The magnetic resonance venography (MRV) was normal. The imaging features were consistent with PRES.

The patient was admitted to the hospital. Nitroprusside sodium was administered to lower the blood pressure gradually. We started intermittent hemodialysis thrice a week from the day of admission. He was treated with active rehydration, diuresis, alkalinization of urine, and supportive measures. The clinical symptoms improved within four days of admission. The blood pressure was controlled and normal, his vision returned to normal, and the headache was significantly relieved. The creatine kinase level dropped considerably (Table), and the daily urine volume was about 1,500 mL. Antihypertensive therapy was switched to an oral calcium channel blocker. Cranial MRI on the eighth day of ad-

mission showed the resolution of the signals (Figure).

After 14 days of treatment, the patient was discharged from the hospital. At one-week follow-up, the patient had no complaints, and his laboratory findings were standard. At three-month follow-up, he showed complete recovery without long-term renal or neurological consequences.

Discussion

Exercise is an important cause of rhabdomyolysis (3). High-intensity exercise leads to necrosis of skeletal muscle cells, releasing cellular contents and causing pain, swelling, fatigue, and potential organ damage (4). The patient had the clinical features of ER. In addition, the patient's clinical presentation and imaging features on MRI supported the diagnosis of AKI and PRES.

Rhabdomyolysis has been shown to be associated with PRES in the published literature (5, 6). However, reports of exercise-induced rhabdomyolysis combined with PRES are relatively rare. A reported case of AKI secondary to rhabdomyolysis in a heavily trained soldier with PRES included convulsive seizures as the associated symptom of PRES (7). A similar case was reported in an adolescent with AKI complicated by PRES following strenuous exercise without a diagnosis of rhabdomyolysis but with PRES-associated symptoms of transient loss of consciousness, visual agnosia, and convulsions (8). In the present case, our patient's PRES-related symptoms were headache and painless complete loss of binocular vision, which differed from previously reported cases. Visual symptoms in rhabdomyolysis can be used as a differential diagnosis for an early diagnosis and treatment.

PRES is a clinical radiological syndrome mainly manifested by the continuous destruction of the blood-brain barrier and posterior vascular edema. It is a rare disease (9, 10). In some patients, sharp waves can be seen on an electroencephalogram (EEG) (11). The exact physiopathology of PRES has not been fully elucidated, but hypertension and endothelial injury are almost always present (12). Excessive exercise can easily lead to skeletal muscle lysis and the entry of skeletal muscle cell contents into the bloodstream, resulting in disturbances of the body's internal environment and often leading to AKI, acute hypertension, and multiorgan dysfunction. At the same time, acute hypertension and AKI are common causes of PRES. Therefore, it was speculated that PRES occurred in this patient.

In the present patient, the increase in cerebral blood flow caused by acute hypertension exceeded the regulatory ability of the cerebrovascular system, which caused brain tissue edema. In addition, the endothelial cerebrovascular dysfunction caused by endogenous or exogenous toxin circulation led to cerebral edema. The patient had been healthy in the past, had no symptoms of infection, was conscious, and had normal serum lactate levels. Therefore, viral encephalitis could be ruled out. Unfortunately, no cerebrospinal fluid and EEG examinations were performed. In addition, PRES should be differentiated from demyelinating disease, posterior circulation embolism, venous sinus thrombosis, and acute toxic encephalopathy (13).

In conclusion, ER can lead to severe complications, and exercise training can reduce the incidence of ER. Early and massive fluid replacement is the central principle of treatment. When AKI occurs, aggressive hemodialysis should be administered. When rhabdomyolysis is associated with AKI, hypertension, headache, and visual symptoms, it is necessary to consider the possibility of PRES, and brain MRI should be performed as soon as possible. In most cases, after timely and effective treatment, patients' clinical symptoms usually recover within a few hours to a few days. However, if treatment is not promptly performed, the condition may deteriorate or even lead to death, and neurological sequelae are likely to occur.

The authors state that they have no Conflict of Interest (COI).

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