

Exercise-induced exertional rhabdomyolysis

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ABSTRACT

Rhabdomyolysis is a condition resulting from skeletal muscle breakdown that can present in several ways, ranging from no symptoms to a life threatening renal disorder. A variety of insults, including trauma, toxins, drugs, infections, and exercise, can lead to muscle breakdown. Complications include compartment syndrome, electrolyte imbalance, and cardiac arrest. Rhabdomyolysis is a clinical challenge due to the range of its presentations. We report a 22-year-old male college student who came to the emergency department with mild thigh soreness and dark urine. A full work-up showed his serum creatine kinase was significantly elevated to 178,786 U/L and he had acute kidney injury. Our patient had no toxin or drug exposure, no infection, no trauma, and no crush injuries, but he had attended a 45-minute spinning class several days prior to admission, indicating a case of exercise-induced exertional rhabdomyolysis. He was hospitalized and treated with IV hydration to protect his kidneys. After eight days of conservative treatment with IV fluids, the patient's creatine kinase level normalized. This case illustrates that even patients with minimal risk factors for rhabdomyolysis can present with severe kidney injury requiring prolonged hospitalization.

Keywords: exertional rhabdomyolysis, spinning, acute kidney injury

INTRODUCTION

Rhabdomyolysis is an infrequent but potentially dangerous condition that requires prompt workup and appropriate treatment. There are several possible causes of rhabdomyolysis, and they are broadly divided into three categories. The first is traumatic causes, which include muscle compression as a result of crush injuries or prolonged periods of immobilization. The second is non-traumatic and non-exertional causes, such as drugs, toxins, infections, or disorders of electrolytes. The third category is non-traumatic exertional causes. This type of rhabdomyolysis is due to marked exertion by trained or untrained athletes, hyperthermia, or rare metabolic myopathies, including McArdle disease. As the number of individuals participating

in organized exercise continues to increase, the incidence of exertional rhabdomyolysis will also increase.¹ It is vital to understand how to recognize and treat these patients, as their presentations may vary. We present a non-traumatic, exertional case of rhabdomyolysis resulting in acute kidney injury which required sustained treatment with intravenous fluids to ensure full recovery.

CASE

A 22-year-old Hispanic male college student presented to the emergency department with a two-day history of bilateral thigh soreness and a one-day history of orange urine. He came to the hospital to be evaluated and to acquire a school excuse for missing class that day. He attended a 45-minute spinning class at the recreational center with a friend three days prior; he had no recent trauma. After the onset of dark urine, the patient began to hydrate with 7 regular-sized water bottles, which did not change the color

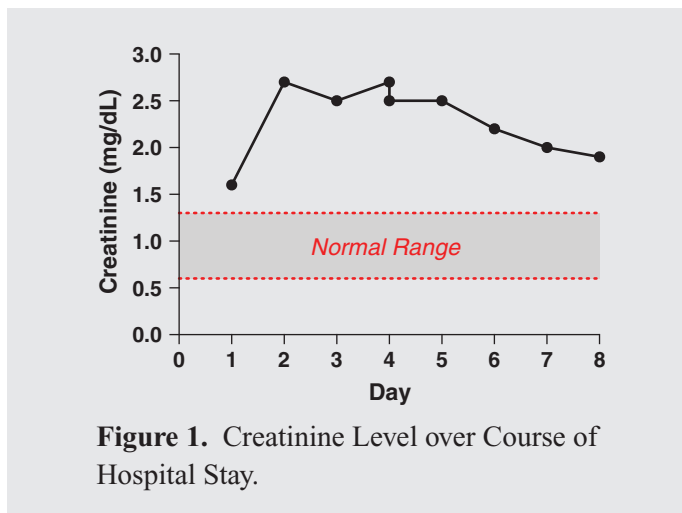
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DOI: 10.12746/swrccc.v6i23.463

of his urine. His social history was negative for any drug, tobacco, or alcohol use. He had no medical or surgical history before this encounter. His only family history was maternal hypertension, and he had no known family history of any metabolic, autoimmune, or neuromuscular diseases. He was on no home medications, including no supplements or workout enhancing medications. He occasionally rode his bike for leisure, approximately 3 miles 3 times per week during the school year, but did not routinely participate in spinning classes.

On physical examination, the patient was alert and cooperative and in mild distress. His BMI was in normal range, and he was of average build. He was hypertensive at 149/101mmHg; other vitals were within normal limits. Head, neck, respiratory, cardiovascular, and abdominal examinations all showed normal findings. Musculoskeletal examination showed decreased strength and increased tenderness in his lower extremities. The patient had normal pulses and no edema in his extremities. His skin was free of bruising, lesions, or rashes. He was neurologically intact with normal cranial nerve function. His capillary refill was less than 2 seconds, and his mucous membranes were moist and showed adequate hydration.

On admission, lab studies revealed WBC count $15 \times 10^3/\text{mm}^3$, creatinine 1.6 mg/dL, and serum creatine kinase (CK) 178,786 U/L (normal: 35-252). Other lab values included AST 2,351 U/L and ALT 575 U/L. Urinalysis showed straw-colored urine, a large amount of blood, a high level of urine myoglobin (427 mcg/L), and high urine protein (17 mg/dL). Urine drug screen was negative. ECG showed normal sinus rhythm with no abnormalities. His chest x-ray was normal. Renal ultrasound revealed no focal lesions, no hydronephrosis, and a small amount of free fluid in the pelvis. The myoglobin in the urine and the elevated serum CK with the patient's history confirmed the diagnosis of rhabdomyolysis, likely due to the spinning class he attended three days prior to admission.

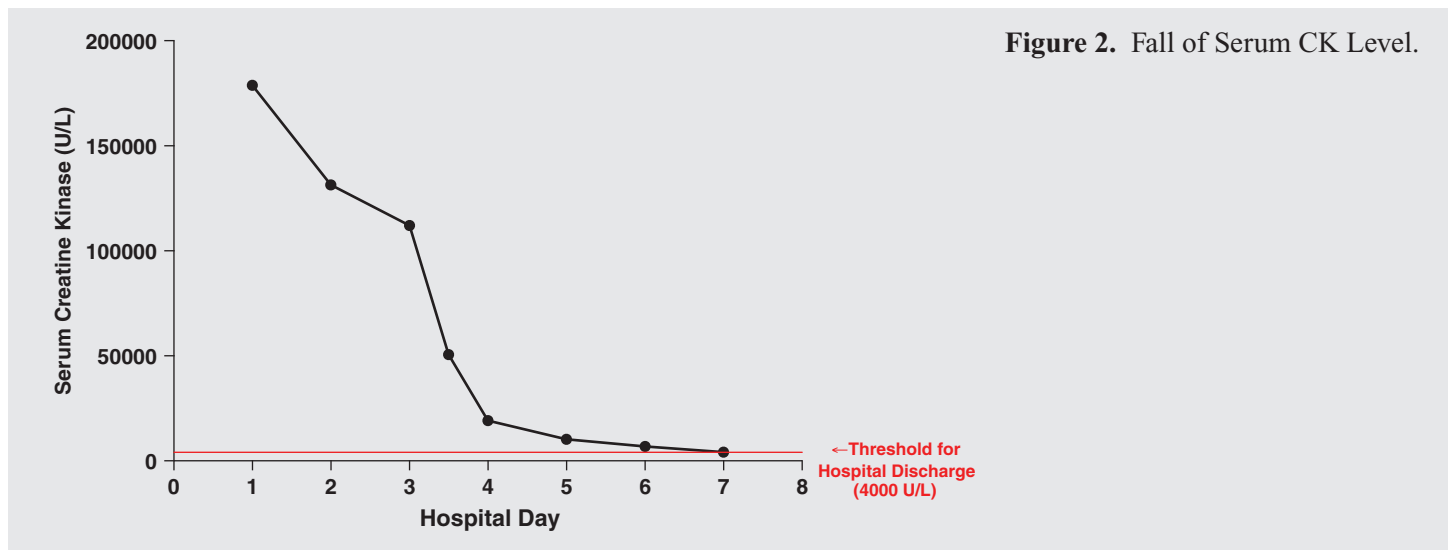
The patient was admitted to inpatient care for acute kidney injury secondary to severe rhabdomyolysis. Treatment was initiated with a 3 liter bolus of normal saline and was continued at 200 mL/hr to



achieve adequate urine output. His prolonged hospital stay was necessary due to his very high CK and AST/ALT levels and abnormal renal function. The day after admission, his creatinine increased to 2.7 mg/dl from 1.6 mg/dl and then trended downward (Figure 1). His hospital course was unremarkable, and he continued to improve on IV fluids alone with no sodium bicarbonate and no renal replacement therapy. His serum CK levels dropped from 178,786 U/L on admission to 4,144 U/L on Day 8 (Figure 2). AST and ALT levels normalized after eight days. He was discharged and followed up in outpatient clinic; he continues to be stable with no recurring episodes of rhabdomyolysis.

DISCUSSION

Our patient had no predisposing factors, did not use drugs, and exercised on a regular basis; yet, one spinning class resulted in acute kidney injury. Cases of exertional rhabdomyolysis are rare but probably underreported, and the incidence of acute kidney injury is lower in exercised-induced rhabdomyolysis than in other causes of rhabdomyolysis.² The mechanism of kidney injury is due to casts of myoglobin forming within the tubules, which lead to direct toxicity, intrarenal vasoconstriction, and a decrease in the glomerular filtration rate.³ Our case illustrates the damage that can be done from low-impact physical activity and the importance of a thorough history and physical examination on admission. The severity



of symptoms on presentation does not necessarily correlate with the severity of rhabdomyolysis, as indicated by this patient's extremely high serum CK level with kidney injury, but relatively normal physical examination. There are case reports of patients with rhabdomyolysis with lower serum CK levels who had worse complications, including compartment syndrome necessitating fasciotomy.² Similar to our patient, this patient also presented to the ED with exertional rhabdomyolysis four days after her first spinning class but had increased intra-compartmental pressures and was emergently taken to the OR for compartment release.²

Although the exact pathophysiology of spinning-induced exertional rhabdomyolysis is unknown, previously reported case series have revealed characteristics shared by patients who develop the disease. Some of the factors are gender, age, exercise experience level, and intensity of exercise. In a case series and literature review done in South Korea, there were 11 cases of spinning-related rhabdomyolysis within a nine month period.⁴ All of the patients were female, and 10 out of 11 were under the age of 27 (one was age 46). They all presented with CK levels >11,000 U/L, but none developed acute kidney injury like our patient did. In another clinical series from New York, three patients developed rhabdomyolysis after spinning.⁵ All three

were under age 28, had dark urine, elevated CKs, and normal creatinine levels on admission. One of the patients had juvenile myoclonic epilepsy; two were female. The BMI of the patients ranged from 18.5 to 32.3 kg/m². All maintained normal renal function throughout their hospitalizations. A 2016 *American Journal of Medicine* case series reviewed 46 reported cases of exertional rhabdomyolysis after a spinning class.⁶ Forty-two cases followed a first spin class; only three of the 46 developed acute kidney injury. These case series indicate some common features: many of the patients who develop exertional rhabdomyolysis from spinning were first time participants, under age 35, and female. Factors that can vary are BMI, clinical presentation, and exercise experience level.

Cases of patients with recurrent episodes of rhabdomyolysis can provide insight into some relevant clinical factors involved in the mechanism of developing the disease. An athlete who had two episodes of rhabdomyolysis two weeks apart, one after a frisbee tournament and one after a karate class, was found to have a mutation in the CPT II (carnitine palmitoyltransferase) gene.⁷ Another patient with recurrent rhabdomyolysis had long-chain 3-hydroxyacyl-coenzyme A dehydrogenase deficiency, a recessive disorder.⁸ Single episodes of rhabdomyolysis are likely caused by trauma, exercise, or alcohol use; recurrent episodes are likely caused by a metabolic myopathy,

including disorders of fat metabolism, carbohydrate metabolism, or mitochondrial function. In a retrospective cohort study done in 2016, it was found that U.S. Army soldiers with sickle cell trait had a significantly elevated incidence of exertional rhabdomyolysis compared to those without the sickle cell trait (hazard ratio, 1.54; 95% CI 1.12-2.12; $P=.008$); however, there was no higher risk of death.⁹ It is hypothesized that the sickling of red blood cells and polymerization of hemoglobin S lead to microvascular thrombosis, predisposing to muscle ischemia and capillary occlusion, which then lead to a higher risk of rhabdomyolysis. Patients who have a single episode of rhabdomyolysis and no genetic myopathies may theoretically have an acquired myopathy from dysregulation of the muscle repair mechanism.¹⁰ Another potential explanation is that patients have varying composition of type II muscle fibers, making them “high” responders or “low” responders based on CPK levels after physical stress.¹⁰ The predisposing factors in healthy patients remain unclear, but as more cases are studied, our understanding of the mechanisms should improve.

Conservative management with IV fluids allowed our patient to recover without major acid-base abnormalities or chronic kidney damage. Prompt treatment was vital; delayed diagnosis and treatment could have resulted in muscle necrosis, nerve damage, or compartment syndrome, as seen in other patients. The right balance of adequate hydration without volume overload is essential to prevent complications. The current standard of care for treating rhabdomyolysis includes IVF hydration, urine alkalinization with sodium bicarbonate, and diuretics, such as mannitol; however, IV fluids are the only treatment supported by data from randomized controlled studies.¹¹ Our patient received IV hydration but did not receive sodium bicarbonate or mannitol. Studies have shown that treatment with mannitol and bicarbonate does not prevent renal failure, dialysis, or mortality in patients with CK levels greater than 5,000 U/L.¹² Dialysis is another option for treatment for acute kidney injury secondary to rhabdomyolysis, but evidence-based medicine indicates that conservative treatment is non-inferior to renal replacement therapy with regards to mortality rates.¹³ Indications for renal replacement therapy

include persistent hyperkalemia >6.5 mmol/L, severe oliguria/anuria, or persistent metabolic acidosis with a pH <7.1 .¹⁴

KEY STATEMENTS

1. This case demonstrates that rhabdomyolysis can occur in anyone, including relatively young and fit individuals with no risk factors, and cause acute kidney injury.
2. Future studies should investigate recurrence rates in these individuals and evaluate them for evidence of subclinical muscle disease.

Article citation: Rao A. Exercise-induced exertional rhabdomyolysis. *The Southwest Respiratory and Critical Care Chronicles* 2018;6(23):12–16

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Submitted: 1/7/2018

Accepted: 3/8/2018

Reviewers: Michael Phy DO

Conflicts of interest: None

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