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# Severe Elevation in Serum Creatinine Kinase in a Patient with Rhabdomyolysis Following Intensive Exercise: A Case Report

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**Abstract**

Background: Rhabdomyolysis is a clinical and biochemical syndrome characterized by the release of cellular contents into circulation due to skeletal muscle injury. Important indicators of muscle damage include myoglobin, creatinine kinase (CK), and lactate dehydrogenase. The severity of the condition can vary from asymptomatic increases in muscle enzymes to life-threatening electrolyte disturbances and renal failure. Electrolytes released into circulation along with myoglobin secondary to myocyte membrane damage are responsible for the development of complications. Acute kidney injury can occur, especially when myoglobin accumulation in renal tubules is accompanied by hypovolemia and renal vasoconstriction. This case presentation aims to discuss an exercise-related rhabdomyolysis case with a CK level of 71150 IU/L, in conjunction with current literature. Case Presentation: A 32-year-old male patient presented to the emergency department with complaints of weakness and severe pain in both legs after intense exercise. His work up revealed a CK level of 71150 IU/L, alanine aminotransferase (ALT) 92 IU/L, aspartate aminotransferase (AST) 691 IU/L, and potassium (K) 5.7 mEq/L. He is diagnosed with acute rhabdomyolysis due to exercise and intravenous fluids administered. He didn't require hemodialysis. On 17$^{th}$ day of admission, he is discharged with full recovery. Conclusions: Physicians should be aware of rhabdomyolysis in patients presenting with weakness and muscle pain, especially in those reporting intensive physical exercise.

**Keywords**

Acute kidney injury, Creatinine kinase, Exercise, Rhabdomyolysis

**Article History**

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## 1. Introduction

Rhabdomyolysis is a clinical syndrome characterized by the release of cellular contents into circulation due to damage to skeletal muscles. This condition can be triggered by various factors, with the most common causes including trauma, excessive exercise, medication use, and dehydration. Trauma [1], strenuous exercise [2], drugs/toxins [3], infections [4], and seizure are among the causes of rhabdomyolysis.

Exercise-induced rhabdomyolysis (EIR) is a clinical syndrome marked by skeletal muscle breakdown triggered by intense or unaccustomed exercise, resulting in the systemic release of intracellular components, most notably creatinine kinase (CK) and myoglobin, into the bloodstream [5]. It is one of the most common causes of exertional rhabdomyolysis. Other causes of exertional rhabdomyolysis (ER) include heat stroke, high humidity, and dehydration. While rigorous physical activity normally causes CK elevations of up to ~20× the upper normal limit, EIR represents a pathological breach in this spectrum where muscle damage becomes substantial enough to prompt clinical symptoms and potential systemic complications [6]. Serial works in the literature reported that EIR disproportionately affects young, healthy males, often between their late teens and thirties, who engage in endurance events (≈ 54%) or weight training (≈ 15%) without proper conditioning [7]. Average CK levels in diagnosed cases typically reach >30000 IU/L, with peaks soaring above 38000 IU/L—far exceeding normal thresholds [8]. Importantly, myoglobinuria, evidenced by dark, tea-colored urine, is a hallmark feature and often accompanied by severe muscle pain, weakness, and swelling [6]. Mechanistically, EIR stems from ATP depletion in muscle fibers during extreme exertion, which leads to ion channel dysfunction, excess intracellular calcium, and activation of proteases and phospholipases that disrupt cellular integrity—releasing CK, myoglobin, and other muscle proteins into circulation [6]. Although CK and myoglobin leakage themselves are notable, the most concerning sequela is the risk of acute kidney injury (AKI) from myoglobin-mediated nephrotoxicity, which has been documented in approximately 10%-30% of EIR cases [9].

The management of exercise-related rhabdomyolysis cases is critical for early diagnosis and treatment. Symptoms typically present as muscle pain, weakness, and brown urine, which can lead patients to seek emergency care. However, it should be noted that these symptoms may conceal a more serious condition.

In present report we aimed to present a case of rhabdomyolysis occurring after exercise and to review the diagnosis, follow-up, and treatment processes related to this condition.

## 2. Case Report

A 32-year-old male patient presented to the emergency department with complaints of weakness and severe pain in both legs. His medical history revealed no previous illnesses or medication use. It was learned that he had performed intense exercise without a coach the day before. The following day, he experienced widespread muscle pain, particularly in the legs, which worsened in intensity. Further inquiry revealed that he had noticed his urine turning brown and had a decreased urine output one day after the exercise. The patient reported difficulty walking. He denied having fever, chills, nausea, vomiting, burning sensations, or abdominal/flank pain, and stated he did not use any illicit drugs or dietary supplements. Upon admission to the emergency department, the patient was cooperative and oriented. Physical examination showed tenderness in the lower extremities, but no other significant findings. Vital signs were as follows: blood pressure 120/83 mmHg, body temperature 36.5 °C, pulse 81 bpm, and $SpO_2$ 98% (room air).

Laboratory findings revealed: CK 71150 IU/L, alanine aminotransferase (ALT) 92 IU/L, aspartate aminotransferase (AST) 691 IU/L, and potassium (K) 5.7 mEq/L, with no other abnormalities in serum biochemistry, including blood urea nitrogen (BUN), serum creatinine, and other serum electrolytes. His McMahon risk score was calculated as 5 (low risk for rhabdomyolysis). The patient was admitted with a preliminary diagnosis of rhabdomyolysis for further monitoring and treatment. Intravenous hydration of 4000 cc daily was initiated, resulting in a decrease in CK levels (as shown in Figure 1, and potassium and liver function test levels returned to normal ranges. The patient did not require hemodialysis and all of his abnormal laboratory findings, including CK, returned to normal range during hospitalization. He was discharged after a 17-day hospitalization with a recommendation for follow-up in the internal medicine outpatient clinic.

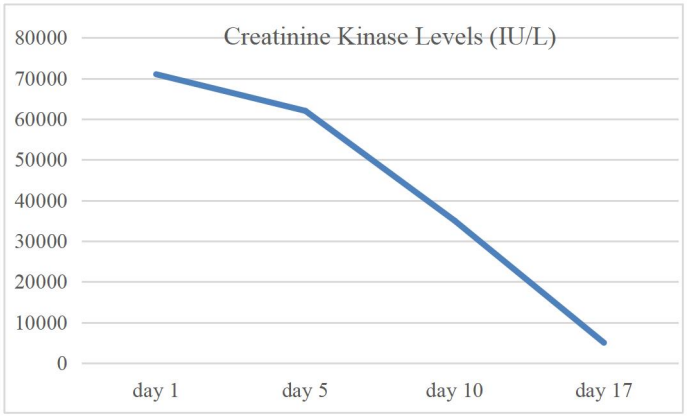


**Figure 1.** Trend of the patient's serum CK levels during follow-up.

## 3. Discussion

We presented here a case with severe CK elevation, accompanied with increment in AST and serum potassium levels, in a patient following intense exercise.

Rhabdomyolysis was first described by Fleisher in 1881 after observing hemoglobinuria in soldiers following a long march. The pathogenesis of rhabdomyolysis was later linked to myoglobin entering circulation due to muscle damage and acute tubular necrosis by Bywaters and Beall in 1941 [10]. It is a clinical syndrome characterized by the breakdown of skeletal muscle fibers, leading to the release of intracellular contents into the bloodstream [11].

Ion channels in muscle cells maintain low levels of sodium ($Na^+$) and calcium ($Ca^{2+}$) ions, while potassium ($K^+$) ions are kept at high concentrations. The breakdown of muscle cells or the inability to produce energy triggers rhabdomyolysis. When energy production is impaired, ATP-dependent ion pumps, such as Na/K ATPase and $Ca^{2+}$ ATPase, cannot function properly [12,13].

CK levels typically rise within 2 to 12 hours after muscle injury, peak at around 24 to 72 hours, and then decline over several days if no further muscle damage occurs. CK levels can exceed 5000 IU/L and sometimes reach above 100000 IU/L in severe cases [3]. In our case, the CK level was determined to be 71150 IU/L.

Causes of ER include heat stroke, high humidity, intensive exercise and dehydration. Normal intense exercise can raise CK significantly, up to 10× the upper normal limit (e.g., from ~100 IU/L baseline to ~6000 IU/L), peaking about 3-4 days post-workout then declining gradually [14-16]. However, CK elevations beyond ~5000 IU/L, especially with symptoms or myoglobinuria, may signal ER [17,18]. ER is diagnosed when muscle breakdown leads to CK levels commonly >10× normal, plus signs like dark (myoglobin-positive) urine, muscle pain/swelling, and potential complications [19]. A large systematic review (772 individuals post-endurance/weight training) found mean CK ≈31500 IU/L (range: 164-106000), with peak CK averaging ~38500 IU/L in documented cases [5]. A Swiss emergency-department review of CK ≥1000 IU/L cases found 2.1% due to exertion, average CK ~16900 IU/L. Nearly 43% developed AKI [20]. Military recruit data show ER incidence of ~22 per 100000 per year; civilian studies similarly show young adult males (19-29 years, BMI ≥25) are at higher risk, the majority recover with hydration [17]. Dehydration, high heat/humidity, unaccustomed or high-eccentric exercise, and inadequate conditioning heighten the risk [3]. CK levels begin rising within 12 h of injury, peak between 1-3 days, then decline over 3-5 days if no further muscle damage occurs [1]. CK half-life is approximately 36 hours, longer than serum myoglobin, making CK a reliable muscle damage marker [21]. CK levels correlate with severity: levels above ~5000 IU/L show more muscle injury and risk of AKI; dramatically higher levels further increase acute complications [10].

Most ER cases are managed with aggressive IV hydration, monitoring electrolytes and renal function, and CK levels usually decline rapidly [9]. About 40%-43% of ER patients develop AKI [3]. Other risks include electrolyte imbalances (like hyperkalemia), compartment syndrome, arrhythmias, transaminase elevations, and rare coagulopathy [19]. Reported extreme cases include one athlete with CK-induced AKI, disseminated intravascular coagulation, and compartment syndrome requiring fasciotomy and dialysis [18]. However, present case did not suffer from AKI. Similarly, authors reported high CK levels without AKI in a patient in literature [22].

Rhabdomyolysis can result in severe complications, most notably AKI [23]. The pathophysiology of rhabdomyolysis-induced AKI is multifactorial. Recent studies have highlighted that AKI develops in a significant proportion of patients with rhabdomyolysis, with incidences ranging from 13% to 60%, depending on the underlying cause and patient population [24]. Myoglobin, released from damaged muscle cells, plays a central role by inducing oxidative stress, inflammation, and direct tubular toxicity within the kidneys. Additionally, factors such as hypovolemia, electrolyte imbalances, and aciduria exacerbate renal injury [25]. Following cell damage secondary to trauma, electrolytes and enzymes, particularly potassium, myoglobin, and CK, as well as uric acid, phosphate, lactate dehydrogenase, and AST,

increase in serum [26,27]. When myoglobin levels rise and exceed plasma binding capacity, it can form glomerular casts, potentially leading to AKI [10]. Accordingly, our patient had elevated levels of CK, AST, and potassium at the time of admission.

ER, resulting from intense physical activity, has been increasingly recognized, especially among professional athletes and military personnel [28]. While many cases are self-limiting, some progress to AKI, underscoring the importance of early recognition and management The three most common symptoms are muscle pain, tea-colored urine due to myoglobinuria, and decreased urine output. Our patient presented with the classic triad of symptoms associated with rhabdomyolysis [29].

Treatment primarily consists of intravenous hydration. However, the efficacy of adjunctive therapies such as urine alkalinization with sodium bicarbonate and the use of diuretics remains controversial, with limited evidence supporting their routine use. In cases where AKI progresses, renal replacement therapy may become necessary, although its initiation should be based on clinical indications rather than solely on laboratory values [28]. While elevated CK levels due to exercise usually follow a benign course, there have been rare cases requiring hemodialysis reported in the literature. Our patient responded well to IV hydration and was discharged in good condition without developing AKI.

## Conclusion

In conclusion, physicians should consider rhabdomyolysis in patients presenting with muscle pain and weakness after intense exercise. Prompt treatment with intravenous fluids should not be delayed once the diagnosis is established to avoid renal injury.

## Conflict of Interest

The authors declare that there are no conflicts of interest.

## Generative AI Statement

The authors declare that no Gen AI was used in the creation of this manuscript.

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