

Intense Training Leading to Dialysis: About an Original Cause of Acute Kidney Injury

Smaali. S^{1*}, Anibar.S¹, EL Ouazzani.M¹, Bouchoual.M¹, Jabrane.M¹, Arrayhani.M¹

¹Nephrology, Hemodialysis, and Kidney Transplantation Department, Souss Massa University Hospital, Faculty of Medicine and Pharmacy, Ibn-Zohr university, Agadir, Morocco

DOI: <https://doi.org/10.36347/sasjm.2025.v1i09.022>

| Received: 19.07.2025 | Accepted: 24.09.2025 | Published: 27.09.2025

*Corresponding author: Smaali. S

Nephrology, Hemodialysis, and Kidney Transplantation Department, Souss Massa University Hospital, Faculty of Medicine and Pharmacy, Ibn-Zohr university, Agadir, Morocco

Abstract

Case Report

Exercise-induced rhabdomyolysis (exRML) is a potentially life-threatening condition characterized by skeletal muscle breakdown following excessive physical exertion. The subsequent release of intracellular components, including myoglobin, creatine kinase, and electrolytes, into the bloodstream can cause severe complications such as acute kidney injury (AKI) and, in rare cases, death. The underlying mechanisms involve disturbances in calcium homeostasis, mitochondrial dysfunction, and structural damage to the muscle extracellular matrix. Although commonly reported in athletes and military personnel, exRML can also occur in otherwise healthy individuals with no prior medical history. What makes the present case noteworthy is that a previously healthy 24-year-old male developed severe AKI requiring hemodialysis after a single intense exercise session. This highlights that exRML can arise in non-athletic young adults and progress rapidly to life-threatening renal failure. The purpose of reporting this case is twofold: first, to raise awareness among clinicians that exRML should be considered even in low-risk populations; and second, to emphasize the importance of early recognition and prompt management to prevent irreversible renal damage and improve outcomes.

Keywords: Rhabdomyolysis, Acute kidney injury, Creatine kinase, Exercise, Myoglobinuria.

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INTRODUCTION

Lately, physical activity has become popular, especially among the young population, due to its widely recognized benefits for both physical and mental health. However, when individuals engage in intense exercise beyond their limits, the risk of musculoskeletal injuries, including exercise-induced rhabdomyolysis (exRML), becomes significantly increased.

Rhabdomyolysis is characterized by skeletal muscle fiber destruction caused by mitochondrial dysfunction and oxidative stress, which collectively damage the sarcolemma and the extracellular matrix. This process results in the uncontrolled release of myoglobin, creatine kinase, and electrolytes into the bloodstream. As a secondary outcome, we observe alteration driven by potassium flux with a focus on the extracellular compartment. Once filtered by the kidneys, myoglobin may precipitate within renal tubules, generate reactive oxygen species, and impair perfusion, thereby contributing to acute kidney injury (AKI). Despite its severity, AKI remains underdiagnosed in exRML [1,2].

ExRML can also lead to electrolyte imbalances, hepatic dysfunction, compartment syndrome, and, in extreme cases, heart failure and arrhythmias [3, 4].

We report the case of young healthy 24-year-old male developed severe AKI requiring hemodialysis after a single intense exercise session. This highlights that exRML can arise in non-athletic young adults and progress rapidly to life-threatening renal failure.

CASE REPORT

A 24-year-old male, with no significant medical history, presented to the emergency department in June with severe pain and swelling in his thighs following an unusually intense workout performed outdoors. Notably, he is usually physically active, but he had never previously engaged in an exercise at this level of intensity before. Despite taking over-the-counter analgesics and nonsteroidal anti-inflammatory drugs (NSAIDs), the pain persisted.

Citation: Smaali. S, Anibar. S, EL Ouazzani. M, Bouchoual. M, Jabrane. M, Arrayhani. M. Intense Training Leading to Dialysis: About an Original Cause of Acute Kidney Injury. SAS J Med, 2025 Sep 11(9): 937-939.

On admission, the patient was hemodynamically stable, with normal blood pressure at 120/80 mmHg. Marked swelling and diffuse tenderness were noted in both thighs, without evidence of ecchymosis. Peripheral pulses were intact and symmetric. Additionally, the patient presented with oliguria, and the urine remained macroscopically clear.

Laboratory tests showed acute kidney injury KDIGO III with elevated serum creatinine (848.64 $\mu\text{mol/L}$) for a normal of 530.41 $\mu\text{mol/L}$ - 1060.82 $\mu\text{mol/L}$, urea (123 mg/dL) (normal: 15-45mg/dL), and significantly elevated hepatic enzyme levels, with Aspartate aminotransferase (AST) :233 IU/L 5.8 $\times$ normal value, and alanine aminotransferase (ALT): 254 IU/L 10 $\times$ normal value. Lactate dehydrogenase (LDH) was also elevated (605 IU/L), but there were no signs of electrolyte abnormalities. Urinalysis revealed microscopic hematuria (30,000 elements/ml),

proteinuria at 1.8 g/24 h, and immunological tests were negative such as complement fraction C3, C4. Given the patient clinical context and presentation and lack of evidence supporting glomerular diseases rhabdomyolysis was highly suspected, and then creatine kinase (CK) levels were measured, revealing a dramatic increase to 10,692 IU/L.

The patient was treated with intravenous hydration and furosemide to induce forced diuresis. However, despite these efforts, fluid retention worsened, reaching up to a 10 kg above his baseline body weight. The patient's renal function continued to deteriorate, requiring hemodialysis after 48 hours of hospitalization. Following three hemodialysis sessions, the patient's condition improved, with restored diuresis and a urine output increase to 4L per day. Progressive decrease of renal function was observed, as indicated by declining serum creatinine and CK levels (Figure 1).

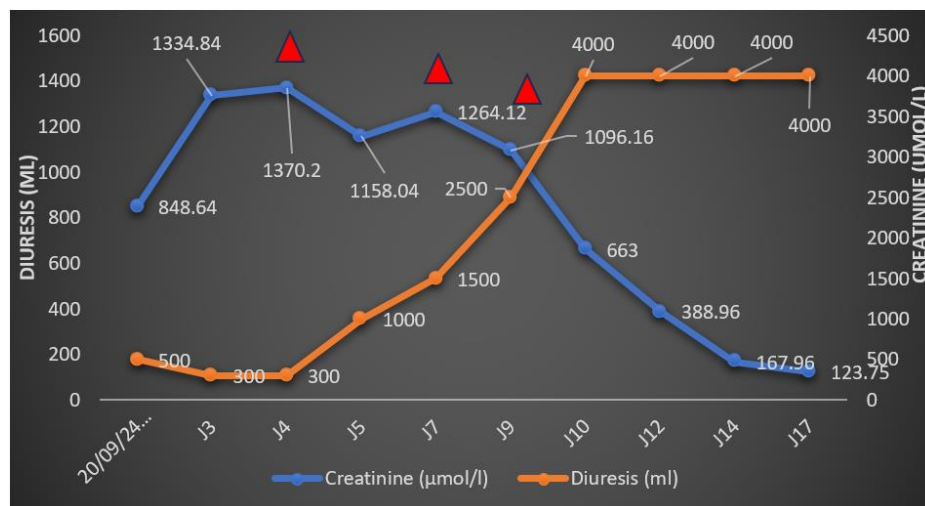


Figure 1: Depicts the progression of diuresis, serum creatinine levels, and dialysis sessions (denoted by red triangles) during the patient's hospitalization

DISCUSSION

The rapid destruction of muscle fibers after intense physical exertion can cause exRML. This breakdown releases intracellular muscle components, primarily myoglobin, into the bloodstream, which can lead to renal injury. Although rhabdomyolysis can be triggered by trauma, substance abuse, or prolonged immobility, it is particularly common in athletes, military personnel, and individuals undergoing strenuous physical activity [5]. In military recruits, for example, the incidence of exRML is reported to be approximately 22.2 cases per 100,000 participants during basic training [6].

Mechanical stress and biochemical changes within muscle cells during extreme exertion contribute to exRML. Structural damage to muscle fibers results in the release of myoglobin, creatine kinase (CK), lactate dehydrogenase (LDH), and potassium into the extracellular compartment [7]. When myoglobin levels exceed the physiological filtration capacity of the kidney,

it leads to myoglobinuria and simultaneously exerts direct tubular toxicity [5]. The toxic effects of myoglobin on the kidneys are further compounded by oxidative stress and the formation of myoglobin-induced renal casts, which obstruct renal tubules, impairing kidney function and contributing to AKI [8].

During intense exercise, excessive mechanical stress on muscle fibers leads to the dysregulation of calcium ions, which activates proteolytic enzymes such as calpains and phospholipases. These enzymes degrade muscle cell membranes, releasing more intracellular components, including myoglobin. Additionally, elevated calcium levels contribute to mitochondrial dysfunction, impairing ATP production and accelerating cell death processes, including necrosis [2, 9].

The diagnosis of rhabdomyolysis observed is based on clinical presentation and laboratory findings. Elevated CK levels are typically one of the first

indicators of muscle injury. However, it is important to note that this can also be observed in healthy individuals after strenuous exercise. In cases of rhabdomyolysis, CK levels are often more than 10 times the normal upper limit [10]. Myoglobinuria, characterized by dark brown or tea-colored urine, is another critical diagnostic feature. Hepatic enzyme elevations, such as AST and ALT, may also be observed due to muscle cell breakdown, as these enzymes are released from injured muscle fibers [11].

Exercising in hot, humid conditions is particularly dangerous, as it increases the likelihood of muscle injury and impairs the body's ability to dissipate heat effectively [12] which was the case of our patient. AKI is one of the most serious complications of exertional rhabdomyolysis, occurring in approximately 33% of cases. It results from the toxic effects of myoglobin on the kidneys, which leads to tubular necrosis and renal dysfunction [13]. Clinicians must closely monitor serum creatinine, potassium, and calcium levels in suspected rhabdomyolysis cases to guide therapeutic interventions.

The primary treatment for rhabdomyolysis-induced AKI involves aggressive hydration to dilute the myoglobin concentration in the bloodstream and promote its excretion via the kidneys. Urine alkalinization, achieved through bicarbonate infusion, may also help prevent further renal injury, as myoglobin is less nephrotoxic at higher urinary pH levels [14]. In severe cases, hemodialysis may be necessary to manage fluid overload and correct electrolyte imbalances [15].

Preventive strategies to reduce the risk of exRML include proper warm-up and cool-down routines, which can reduce muscle injury during intense physical activity [16]. Additionally, avoiding excessive exertion in hot and humid conditions and ensuring adequate hydration before, during, and after exercise can mitigate the risk of rhabdomyolysis. Monitoring other biomarkers, such as lactate dehydrogenase (LDH), may also help predict the risk of AKI in individuals with rhabdomyolysis [17].

CONCLUSION

Exercise-induced rhabdomyolysis is a severe and potentially life-threatening condition that requires prompt diagnosis and intervention. Early detection of rhabdomyolysis and appropriate treatment, including fluid resuscitation and, when necessary, dialysis, are critical to preventing complications such as acute kidney injury. The condition is preventable with proper training and warm-up protocols, and healthcare providers should remain vigilant, especially when managing athletes or individuals engaging in high-intensity exercise regimens.

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