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Case Report

Severe Rhabdomyolysis in a 20-year-old Fit Individual: A Case Report and Literature Review

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Abstract: A previously healthy 20-year-old male individual who regularly practiced karate performed moderate physical activity at a gym without adequate hydration. One day after exercising, he developed intense myalgia. After three days, laboratory tests revealed high levels of creatine phosphokinase (CPK), consistent with severe rhabdomyolysis. Other abnormalities were also found, including elevated transaminases and phosphorus. After 5 days, myalgia had been resolved and creatine phosphokinase levels began to decrease, but there was an increase in creatinine and the patient was hospitalized for intravenous hydration. After 1 day of hospitalization, the patient's creatinine normalized and subsequent laboratory tests showed a gradual return of other laboratory markers to normal. We present the pathophysiological mechanisms, diagnosis, and treatment of rhabdomyolysis, highlighting the potential of certain types of exercise to trigger this muscle pathology even in physically active individuals.

Keywords: Rhabdomyolysis; Eccentric Contractions; Acute Kidney Injury.

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1. Introduction

Rhabdomyolysis is a muscle pathology caused by the necrosis of muscle fibers [1, 2], a phenomenon that can have several causes, such as crush injuries, drugs, excessive exercise, and high temperatures [1, 2, 3]. In the case of rhabdomyolysis due to excessive exercise, there are elements intrinsic to the activities performed, the practitioner, and the conditions under which the activities are carried out that exert considerable influence on the severity of muscle damage and its potential complications.

Physical activities involving eccentric muscle contractions are particularly associated with cases of rhabdomyolysis [3, 5], and high temperatures, especially when combined with dehydration, are also important factors that facilitate and aggravate the development of this condition [3]. Given the various potentially fatal complications of rhabdomyolysis, its mostly nonspecific clinical signs [1, 2, 3], and the significant practice of activities involving eccentric contractions by considerable numbers of individuals, we present the case of a previously healthy 20-year-old male patient who regularly practiced karate and developed rhabdomyolysis after performing moderate physical activity at a gym.

2. Case Report

A 20-year-old male patient, previously healthy, developed intense myalgia in the upper limbs one day after performing moderate physical activity (around 30 minutes of weight training involving thoracic/upper limb muscles as well as 20 minutes of aerobic

activities in the form of treadmill walking) without adequate hydration at a gym on October 11, 2025. The patient regularly practices karate twice a week and reported that on particularly intense days of activity, he had previously experienced episodes of mild myalgia. The patient denied the use of any supplements or stimulants. On October 14, 2025 (Figure 1 and 2), he underwent a routine examination that revealed elevated levels of creatine phosphokinase (9,234 U/L), alanine aminotransferase (47 U/L), and aspartate aminotransferase (124 U/L), with other laboratory data showing no abnormalities or maintaining previous levels. Simultaneously, he reported improvement in myalgia and absence of any other symptoms.

Figure 1. Graph showing creatine phosphokinase (CPK) and creatinine values.

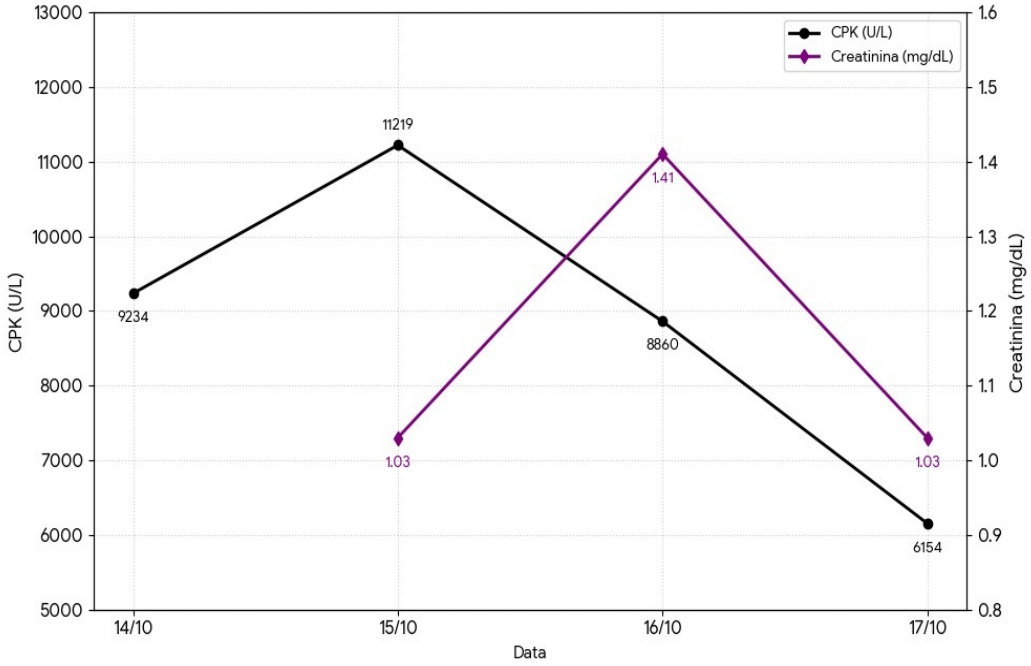
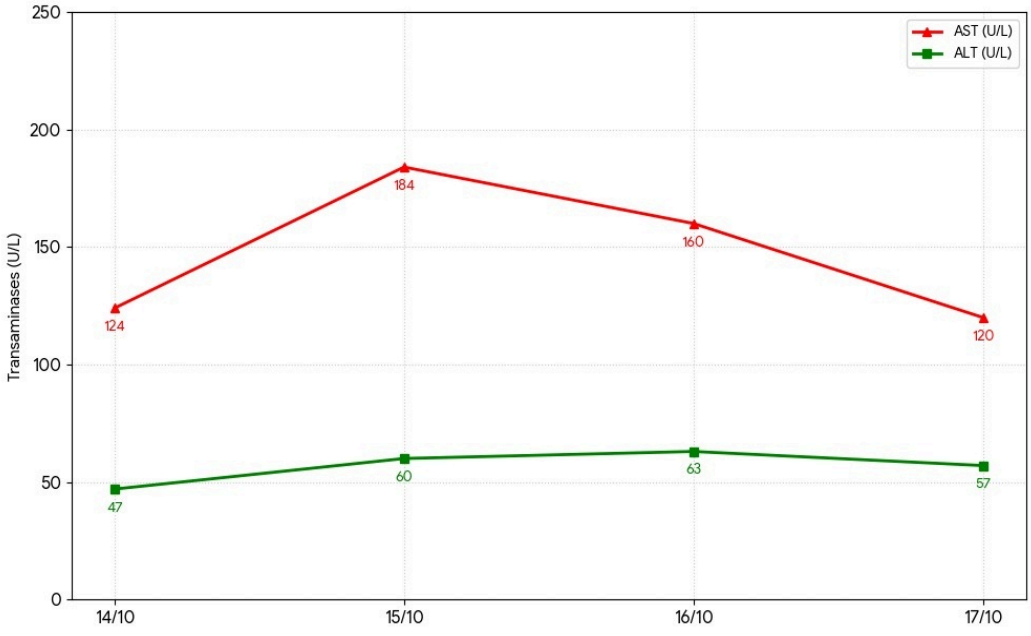


Figure 2. Graph showing aspartate aminotransferase (AST) and alanine aminotransferase (ALT) values.



Due to the very high creatine phosphokinase value, the patient underwent another examination on October 15, 2025 (Figure 1 and 2), which again revealed elevated levels of creatine phosphokinase (11,219 U/L), alanine aminotransferase (60 U/L), and aspartate aminotransferase (184 U/L), in addition to increased phosphorus (4.8 mg/dL), with other laboratory data showing no abnormalities or maintaining previous levels (with creatinine at 1.03 mg/dL). Despite the very high creatine phosphokinase levels, the accompanying clinician chose an expectant approach based on vigorous oral hydration because of the maintenance of urine output and of the creatinine levels.

Once again, an examination was repeated on October 16, 2025 at 10 AM (Figure 1 and 2), which again revealed elevated levels of creatine phosphokinase (8,860 U/L), alanine aminotransferase (63 U/L), aspartate aminotransferase (160 U/L), phosphorus (4.9 mg/dL), and importantly, increased creatinine (1.41 mg/dL) and myoglobin (161 ng/mL). He also underwent a urinalysis, which showed, apart from an increased pH of 8, no other abnormalities, including no findings of positive hemoglobin (representative of urinary myoglobin), and a density of 1.015. On this day, October 16, 2025, the patient no longer had myalgia and reported no changes in urinary habits or urine color, being therefore completely asymptomatic. Due to the increased creatinine, however, he was hospitalized the same day for fluid replacement through intravenous administration of normal saline, initiated around 8 PM. Venous blood gas on patient admission showed a normal pH of 7.35 as well as a slightly increased serum bicarbonate concentration of 28 mmol/L

On October 17, 2025 (Figure 1 and 2), another examination was performed while the patient was still hospitalized, which again revealed elevated levels of creatine phosphokinase (6,154 U/L), alanine aminotransferase (57 U/L), and aspartate aminotransferase (120 U/L), with normalized levels of phosphorus and creatinine, the latter of which fell to 0.89 mg/dL. On the same day, around 4 PM, the patient was discharged and remained asymptomatic.

3. Discussion

Rhabdomyolysis is a muscle pathology caused by the necrosis of muscle fibers [1, 2], a phenomenon that can have several causes, such as crush injuries, drugs, excessive exercise, and high temperatures [1- 3]. Despite these different causes, the underlying pathological mechanism is intracellular calcium excess [2, 3]. This occurs due to dysfunction of transport pumps because of ATP depletion inside myocytes, which in turn causes an increase in intracellular sodium concentration [2, 3]. The increase in intracellular sodium stimulates the activity of sodium-calcium exchangers, which, when performing this exchange, increase the amount of intracellular calcium [2, 3]. The accumulation of calcium in the cytoplasm, when reaching more significant dimensions, leads to activation of phospholipase A2 and calcium-dependent proteases. The activity of these enzymes causes degradation of phospholipids and various proteins, compromising cell membrane integrity and allowing more calcium to flow into the cells. The increasingly greater sarcoplasmic calcium concentration leads to greater ion flow into mitochondria, impairing oxidative phosphorylation and increasing the production of reactive oxygen species [2, 3], which contribute to the degradation of lipids, proteins, and nucleotides [3]. Eventually, muscle fibers undergo necrosis, with their contents released into the extracellular space [1, 2, 3].

Despite the shared underlying mechanism, how the pathology develops is subject to variation. In cases of crush injury, external pressure prevents adequate blood flow to the affected musculature, causing ischemia and ATP depletion, which initiates the cascade detailed above [1, 2]. Among drugs that can cause rhabdomyolysis, statins constitute a common trigger, a fact associated with the central action of such medications, the inhibition of HMG-CoA reductase enzyme [2, 4]. This enzyme, in addition to acting as an essential component in cholesterol synthesis, also participates in the production of ubiquinone, an important component of the mitochondrial respiratory chain whose absence can compromise ATP production [3]. Furthermore, excessive exercise is another possible cause of

rhabdomyolysis, which occurs when energetically demanding activities are performed to the point of ATP depletion and subsequent transporter protein dysfunction [2, 3].

Rhabdomyolysis is associated not only with exercise intensity but also with its modality. Physical activities involving eccentric contractions (those in which muscle elongation occurs concomitantly with contraction) are especially implicated in the development of this pathology [3, 5]. Because they involve a muscle elongation process, eccentric contractions, especially when practiced by unconditioned individuals, can cause myofibril disruption [6]. Such disruption can directly damage muscle fiber cell membranes and, moreover, the elongation process itself stimulates the opening of some ion channels [6], two processes intimately associated with increased intracellular calcium. High temperatures, resulting from exercise itself or environmental conditions, impair the function of the sodium-potassium pump, causing intracellular sodium excess that eventually generates repercussions on calcium, thus constituting another means for rhabdomyolysis or its aggravation. In this sense, dehydration can potentiate the severity of the situation due to water's important function in body thermal homeostasis [3].

The main problem associated with rhabdomyolysis is not the necrosis of muscle fibers itself, but rather the systemic effects caused by the release of internal components of myocytes [1, 2, 3]. Potassium is released in large quantities from necrotic muscle fibers, potentially generating hyperkalemia. This ion, with an important function in muscle fiber repolarization, can generate, when in excess, bradyarrhythmias, which, when progressing to complete cardiac arrest, can be lethal [1, 2]. Considerable portions of phosphate also reach the extracellular space and, when combined with calcium, can generate the formation of calcium phosphate crystals in different tissues, a process that, by involving calcium, intensifies the already decreased levels of this cation, which undergoes a decline process due to the large influx into stressed myocytes. Hypocalcemia, in turn, can intensify the cardiotoxic effects of hyperkalemia, potentiating the severity of the situation [1, 2].

Furthermore, the release of acids such as lactic acid and uric acid can generate metabolic acidosis that intensifies hyperkalemia [2]. The protein myoglobin, an important oxygen storage molecule in muscle fibers, is also released. This protein enters the bloodstream and is deposited in nephrons, especially under acidic conditions. This causes urinary flow obstruction and an intense vasoconstriction of renal arterioles, favoring ischemia and tubular epithelial necrosis. Myoglobin contributes to renal injury in yet another way by releasing iron from its heme group, which causes the production of reactive oxygen species [1, 2]. Dehydration, in addition to increasing the damaging effects of high temperatures, can contribute to the severity of the process by reducing renal blood flow, aggravating the ischemic process and myoglobin deposition [7].

Regarding diagnosis, laboratory findings are of considerable importance, since the traditional triad of rhabdomyolysis, consisting of myalgia, muscle weakness, and dark urine due to myoglobinuria, occurs in only about 10% of affected individuals or even lower percentages. Most symptomatic patients only exhibit myalgia and weakness, highly nonspecific symptoms that make diagnosis difficult [1, 2]. Therefore, detection of increased amounts of the enzyme creatine phosphokinase (CPK) in plasma is the main finding indicative of rhabdomyolysis, since this enzyme shows elevation of its levels 2 to 12 hours after muscle injury and remains in the plasma for up to 10 days, with a peak between 3 and 5 days [1, 2, 3, 8]. Excess myoglobin, in turn, despite being more specific for the rhabdomyolysis process, usually disappears from plasma in a total of 6 to 8 hours, with drastic reduction in its sensitivity [1, 2, 3]. Aminotransferases are also elevated in the rhabdomyolysis process [1, 2, 9], with emphasis on aspartate aminotransferase (AST), which is more abundant in muscles than alanine aminotransferase (ALT), which is more specific for the liver [9]. Like CPK, these enzymes maintain elevated levels for several days. AST rises after about 1 day and has a peak between 3 and 4 days, while ALT usually increases after 2 days, with a peak between 4 and 5 days [9].

Regarding specific values, there is controversy about which exact CPK level constitutes a diagnostic criterion for rhabdomyolysis, but levels above 1,000 U/L or those above 5 times the usual levels for that patient are typically considered [1, 2]. The amount of CPK detected in plasma is associated with the degree of muscle damage [1, 3], with levels less than 10 times the usual being indicators of mild rhabdomyolysis, levels between 10 and 49 times the usual being indicators of moderate rhabdomyolysis, and levels greater than 50 times the usual being indicators of severe rhabdomyolysis [3]. Furthermore, a CPK level above 5,000 U/L shows a strong correlation with the occurrence of acute kidney injury [10]. The amount of myoglobin normally found in plasma is less than 85 ng/ml and, in cases of rhabdomyolysis, values above 15,000 ng/ml have been associated with renal failure and the need for hemodialysis [10], with visible myoglobinuria occurring only with myoglobin values from 1,000,000 ng/ml upwards [1]. There is no specific aminotransferase values used as diagnostic criteria for rhabdomyolysis, but an AST to ALT ratio greater than 1 is expected, with this ratio being, on average, 3 [9].

Treatment of rhabdomyolysis is based on hydration to prevent myoglobin deposition in tubules and thus prevent the occurrence of acute kidney injury. It is also essential to treat underlying causes, when possible, such as discontinuing causative drugs (like statins) or treating infectious agents that affect muscles [1, 2]. Regarding the specific hydration approach, there should be intravenous administration of fluids in the form of isotonic saline ideally 6 hours after the event occurs and replacement should be such as maintain a urinary output of 300 ml/h or more in the first 24 hours. To maintain this amount, mannitol administration may be necessary, but only if such output is not achieved simply with increased fluid replacement [1, 2]. In some cases, intravenous infusion of sodium bicarbonate may also be necessary to correct potential metabolic acidosis [1].

In cases of severe acute kidney injury, severe electrolyte disturbances, and/or hypervolemia, more aggressive interventions should be considered, such as hemodialysis and other renal replacement therapies [1, 2]. Special attention should be given to hyperkalemia, as after performing renal replacement therapies, hyperkalemia can rebound, emphasizing the importance of careful monitoring of potassium levels [1]. Hypocalcemia, on the other hand, should not be corrected, especially because calcium introduction can intensify calcium phosphate crystal precipitation, which causes tissue damage. Furthermore, during the recovery phase, hypocalcemia converts to hypercalcemia, a phenomenon that serves as an additional contraindication to calcium replacement [1].

The values observed in the patient were consistent with severe rhabdomyolysis, since his baseline CPK levels were 187 U/L (identified as such through past routine examinations) and the highest recorded amount, 11,219 U/L, represented an increase of approximately 60 times [3]. Values above 5,000 U/L are already predictive of acute kidney injury [10], a reality that was confirmed by the patient's creatinine increase from 1.03 mg/dL to 1.41 mg/dL. The increase in aminotransferases also occurred as expected in rhabdomyolysis, with greater AST increase and slower ALT decline, which on October 16, 2025, was still increasing, while AST was already declining. The ratio between AST and ALT was around 3, another expected relationship for such a condition [9], further supporting primary muscle injury as the cause for such elevated transaminases.

There was also a slight phosphorus increase, constituting mild hyperphosphatemia, a finding consistent with rhabdomyolysis [1, 2]. One notable finding is the elevated myoglobin (161 ng/mL) 5 days after muscle injury, when a return of this protein to usual levels would be expected a few hours after the precipitating event [1, 2, 3]. This potentially leads to ongoing muscle necrosis even after the resolution of symptoms and/or impaired renal clearance, which would be in accordance with the rise in creatinine.

Regarding clinical manifestations, the patient reported severe myalgia in the absence of other symptoms typically expected in rhabdomyolysis events, which was not a cause for alarm for him, given the occurrence of other myalgic episodes, although milder, after his regular karate training sessions. Furthermore, the patient also reports not having con-

sumed water while performing the exercises, a fact that may have contributed to the development of the pathology [3, 7]. That said, the development of severe rhabdomyolysis in an individual who already regularly performed intense physical activity is notable. While no genetic tests were performed by the patient, he had undergone several previous routine examinations, including some only a few days after karate training sessions, that showed not abnormally elevated CPK levels, strengthening the association with the exercises practiced at the gym setting.

An explanation for this apparent "paradox," beyond the contribution of dehydration, is the performance of exercises of a different nature at the gym, specifically weight training, marked by the predominance of eccentric contractions, which present, due to different factors, a greater potential to trigger rhabdomyolysis [6]. Karate, on the other hand, despite also presenting movements with eccentric contractions, is marked by a significant presence of concentric and isometric contractions, which are associated with the fast and explosive movements of the practice [11, 12].

Regarding patient treatment, intravenous sailing alone proved capable of achieving the urinary output of 300 ml/h in the first 24 hours, leading to a rapid normalization of creatinine levels, which fell to 0.89 mg/dL by the start of the next day (October 17th). Additionally, venous blood gas on patient admission showed a normal pH of 7.35 as well as a slightly increased serum bicarbonate concentration of 28 mmol/L, precluding the administration of intravenous sodium bicarbonate.

6. Conclusion

In summary, the reported case demonstrates that severe rhabdomyolysis can occur through moderate exercises of a specific nature, those notably focused on performing eccentric contractions, in individuals with good physical conditioning considering the regular practice of intense physical exercises. This emphasizes the importance of adequate hydration and the guided and careful practice of activities, especially those marked by eccentric contractions, such as weight training. Furthermore, the fact that the reported patient had only intense myalgia as a symptom, with maintenance of urinary habits even in a context of declining renal function, reinforces that the diagnosis of rhabdomyolysis can often go unnoticed, including severe cases as presented. Therefore, the need for healthcare professionals to be alert to reports of myalgia after intense exercises or exercises different from those usually practiced by that individual is reinforced, given the potentially fatal complications of this pathology.

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Conflicts of Interest: All other authors declare no conflicts of interest.

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