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Exertional Rhabdomyolysis With Moderate Creatine Kinase Elevation and Marked Transaminase Elevation but Preserved Synthetic Function: A Case Report

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Patient: Male, 43-year-old
Final Diagnosis: Exertional rhabdomyolysis
Symptoms: Anorexia • fatigue • myalgia • nausea
Clinical Procedure: —
Specialty: General and Internal Medicine

Objective: Unusual clinical course

Background: Rhabdomyolysis is a skeletal muscle injury syndrome characterized by release of intracellular contents into the circulation. Abnormal liver biochemical test results are commonly observed in severe rhabdomyolysis. Most published cases have focused on extreme creatine kinase (CK) elevations (often > 30 000 U/L) complicated by fulminant hepatic failure. There is a lack of literature regarding instances in which marked transaminase elevation occurs despite moderate CK elevation.

Case Report: A 43-year-old man exhibited fatigue, nausea, and myalgia after a 3000-m run. Laboratory tests showed moderate CK elevation but striking transaminase elevation. Peak values were: CK, 2828 U/L; aspartate aminotransferase (AST), 2294 U/L; alanine aminotransferase (ALT), 1554 U/L; AST/ALT ratio, 1.48. Total bilirubin peaked at 34.2 µmol/L, the international normalized ratio was 1.22, and albumin level remained normal. Viral hepatitis serology findings were negative. The patient was diagnosed with exertional rhabdomyolysis; treatment comprised intravenous fluids, urine alkalinization, and hepatoprotective agents. CK and transaminase levels rapidly declined. At discharge on day 8, the patient was asymptomatic (ALT, 339 U/L). At 1 week, ALT had decreased to 79 U/L; all laboratory parameters were normal at 1 year.

Conclusions: Exertional rhabdomyolysis can produce marked transaminase elevation despite moderate CK elevation, which may not be linearly correlated with AST and ALT levels. This case documents the unusual phenotype of marked transaminase elevation with preserved synthetic function in a patient showing moderate CK elevation. In selected patients with preserved synthetic function and rapidly declining enzyme levels, conservative management may be appropriate after alternative liver injury etiologies have been excluded.

Keywords: Conservative Treatment • Creatine Kinase • Rhabdomyolysis • Transaminases

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Introduction

Rhabdomyolysis is characterized by injury and necrosis of skeletal muscle cells resulting from various causes (eg, trauma, exertion, hyperthermia, medications, and viral infections), which leads to the release of intracellular contents—including creatine kinase (CK), aspartate aminotransferase (AST), and alanine aminotransferase (ALT)—into the bloodstream [1]. Exertional rhabdomyolysis is induced by physical activity or strenuous exercise. Given the lack of evidence-based guidance for the diagnosis and management of exertional rhabdomyolysis, clinical practice guidelines have recently been updated to aid in decision-making [2]. The classic triad of rhabdomyolysis includes myalgia, weakness, and dark urine, although individual symptoms are often atypical [3]. Patients typically present with elevated CK levels and may also display abnormalities in liver and kidney function, as well as electrolyte imbalances [4].

Previous research has suggested a linear relationship of CK elevation with elevations in AST and ALT [5]. A recent systematic review further characterized the relationship between extreme endurance exercise and biomarkers of liver injury [6]. Most published cases of rhabdomyolysis-associated liver injury have focused on extreme CK elevations (usually > 30 000 U/L) complicated by fulminant hepatic failure requiring intensive care, plasma exchange, or even liver transplantation [7-10]. However, reports of moderate CK elevation but markedly elevated transaminase levels are limited. This report documents an unusual phenotype: marked transaminase elevation with moderate CK elevation and preserved synthetic function in exertional rhabdomyolysis. Any diagnostic or therapeutic implications are presented as hypothesis-generating observations from a single case; they are not intended to serve as validated clinical criteria.

Case Report

A 43-year-old Han Chinese man developed fatigue, anorexia, nausea, and myalgia after participating in an outdoor run on a warm day. The patient was a sedentary office worker who did not engage in regular physical exercise. On the day of presentation, he completed a 3000-m outdoor run (ambient temperature approximately 28 °C) without adequate pre-hydration, which represented unfamiliar strenuous exercise for him. His medical history included hypertension and gastroesophageal reflux disease, without a history of liver disease. A routine health examination 2 months prior to admission showed normal liver function, with an ALT level of 13 U/L, AST level of 17.3 U/L, and total bilirubin (TBIL) level of 19.3 µmol/L. He denied recent alcohol consumption, exposure to hepatotoxic drugs, or risk factors for viral hepatitis. His family history was unremarkable.

On admission, vital signs were as follows: temperature, 36.5 °C; heart rate, 70 beats/min; blood pressure, 154/103 mm Hg; respiratory rate, 18 breaths/min; and body mass index, 25.9 kg/m^2. Physical examination revealed no jaundice, scleral icterus, or stigmata of chronic liver disease. The abdomen was soft and nontender, without hepatosplenomegaly or ascites. Neurologic examination was normal, without asterixis or encephalopathy. Bilateral lower extremity muscle tenderness was present, but there was no swelling or evidence of compartment syndrome.

Initial laboratory tests performed in the outpatient clinic showed markedly elevated transaminase levels (ALT, 1554 U/L; AST, 2294 U/L). Other laboratory markers were as follows: TBIL, 25.7 µmol/L; direct bilirubin (DBIL), 9.1 µmol/L; albumin, 45 g/L; lactate dehydrogenase (LDH), 1786 U/L; CK, 1952 U/L; alkaline phosphatase, 65 U/L; creatinine, 98 µmol/L; uric acid, 675 µmol/L; and potassium, 3.3 mmol/L. The procalcitonin level was 0.846 ng/mL, and the serum myoglobin level was 156 ng/mL. The coagulation profile showed an international normalized ratio (INR) of 1.22 and D-dimer level of 1.41 mg/L. Abdominal ultrasound demonstrated slightly coarse hepatic echotexture without focal lesions or biliary dilation. Repeat laboratory testing after admission later that night (10: 30 PM) showed the following: ALT, 1355 U/L; AST, 1444 U/L; LDH, 1047 U/L; CK, 2048 U/L; TBIL, 26.3 µmol/L; DBIL, 10.2 µmol/L; and INR, 1.22. Urinalysis revealed proteinuria (3+), glucosuria, ketonuria, and hematuria. The hepatitis B surface antigen test result was negative, whereas the hepatitis B surface antibody test result was positive, consistent with prior vaccination. Hepatitis A immunoglobulin M (IgM), hepatitis C antibody, and hepatitis E IgM test results were negative; the hepatitis E immunoglobulin G (IgG) test result was positive, indicating past infection. The patient had no fever, altered mental status, or evidence of disseminated intravascular coagulation, effectively excluding heat stroke as a complicating factor.

Initial treatment consisted of: (1) aggressive intravenous fluid resuscitation (4000-5000 mL/day for the first 48 hours); (2) urine alkalinization with sodium bicarbonate (5% $NaHCO_3$, 125 mL intravenously once daily for 3 days; target urine pH, 7.0-7.5); (3) hepatoprotective agents (glycyrrhizin, glutathione, and ademetionine); and (4) potassium supplementation. No invasive procedures, including liver biopsy or muscle biopsy, were performed. N-acetylcysteine, plasma exchange, continuous renal replacement therapy, and liver transplantation were not required.

On admission, the patient reported dark urine with reduced urine output. Urine output was initially low (170 mL during the first 24 hours) but increased to 2850 mL per 24 hours after fluid resuscitation on day 2 and remained adequate thereafter (3000 mL per 24 hours on day 3). Urine color gradually changed from dark brown to clear yellow by day 3, consistent with resolution of myoglobinuria.

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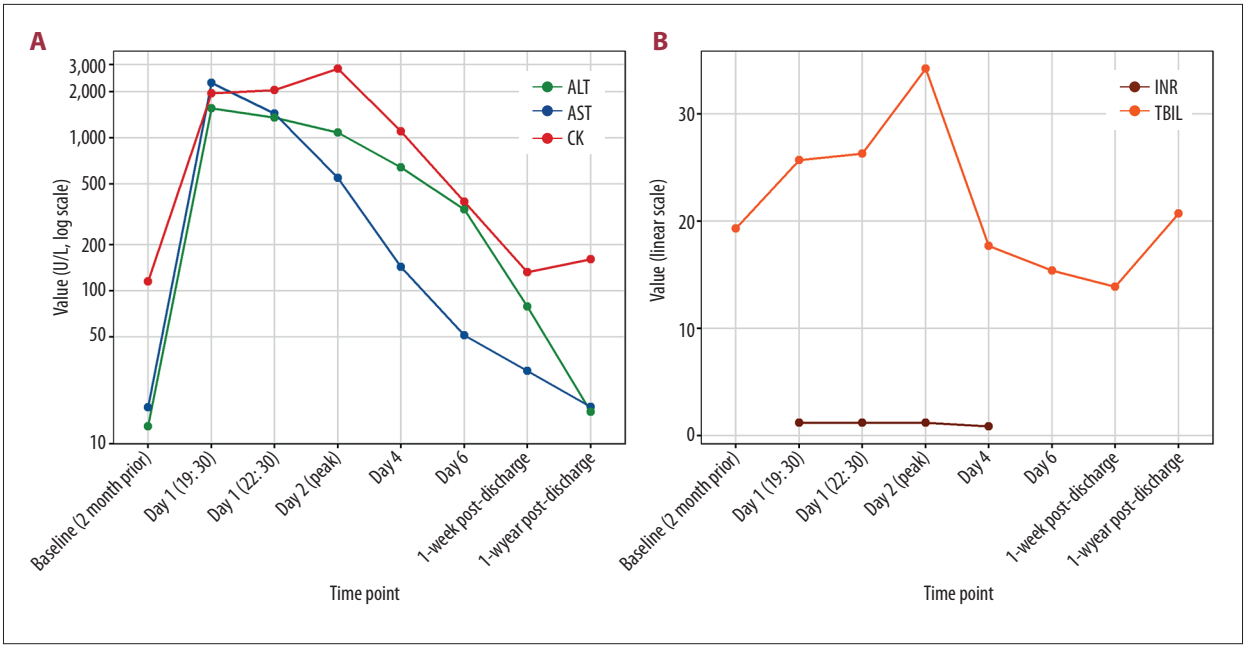

Figure 1. Temporal trends in CK, AST, ALT, TBIL, and INR during hospitalization and follow-up. (**A**) Left y-axis (log scale): CK, AST, and ALT (U/L). (**B**) Right y-axis (linear scale): TBIL (μmol/L) and INR. X-axis: time points (baseline, hospital days 1-6, and follow-up at 1 week and 1 year). No laboratory tests were performed on days 3 and 5; therefore, data points are connected by straight lines for visualization only, and values are shown only at measured time points. Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; INR, international normalized ratio; mo, months; TBIL, total bilirubin.

On day 2, the CK level peaked at 2828 U/L. Other levels were ALT, 1083 U/L; AST, 548 U/L; LDH, 538 U/L; TBIL, 34.2 μmol/L; DBIL, 12.5 μmol/L; gamma-glutamyl transferase, 63 U/L; and INR, 1.22. Urine output improved to 2850 mL per 24 hours after fluid resuscitation. Subsequently, all laboratory parameters continued to improve. On day 4, levels were CK, 1100 U/L; ALT, 640 U/L; AST, 143 U/L; TBIL, 17.7 μmol/L; INR, 0.88; and serum myoglobin, 51.8 ng/mL. On day 6, levels were CK, 381 U/L; ALT, 339 U/L; AST, 51 U/L; and TBIL, 15.4 μmol/L. The patient was discharged on day 8 in good condition and remained asymptomatic. The ALT level remained mildly elevated (339 U/L), but all other laboratory parameters had normalized. No laboratory tests were performed on the day of discharge.

One week after discharge, the patient remained asymptomatic. Follow-up laboratory testing showed the following: ALT, 79 U/L (still mildly elevated); AST, 30 U/L; TBIL, 13.9 μmol/L; and CK, 132 U/L. At the 1-year follow-up (February 11, 2026), all laboratory parameters were normal: ALT, 16.2 U/L; AST, 17.4 U/L; TBIL, 20.7 μmol/L; and CK, 160.4 U/L. No laboratory testing was performed during the intervening period, and the patient reported no symptoms.

Figure 1 illustrates dynamic changes in CK, AST, ALT, TBIL, and INR during hospitalization and follow-up. **Table 1** summarizes key laboratory values at baseline, admission, peak, discharge, and follow-up. **Table 2** presents the differential diagnosis of transaminase elevation in the setting of rhabdomyolysis.

Discussion

In the present case, rhabdomyolysis occurred after a 3000-m run, an intensity generally considered safe for healthy adults. This finding suggests that even moderate exercise can trigger rhabdomyolysis in susceptible individuals, particularly in the presence of high ambient temperature, dehydration, poor physical conditioning, or underlying metabolic susceptibility. Our patient had a body mass index of 25.9 kg/m^2 and slightly coarse hepatic echotexture on ultrasound, suggesting possible underlying nonalcoholic fatty liver disease, although this possibility was not confirmed by biopsy or dedicated imaging. Such a condition could potentially amplify myoglobin-induced oxidative stress [11,12]. Similar observations have been reported after indoor spinning [13,14] and military training [15,16]. A systematic review of indoor spinning-induced rhabdomyolysis indicated that an increasing number of patients are being diagnosed with this condition; common presentations include myalgia, dark urine, and muscle weakness [17]. The disproportionate biochemical response to a 3000-m run in our patient suggests individual susceptibility and emphasizes that this observation may not be generalizable to all individuals with similar exercise exposure. Clinicians should maintain

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Table 1. Key laboratory values at baseline, admission, peak, discharge, and follow-up.

Values (reference range)	Baseline (3 months prior)	Admission (Day 1, 7: 30 PM)	Day 1 (10: 30 PM)	Day 2 (peak)	Day 4	Day 6 (most recent at discharge)	Follow-up (1 week)	Follow-up (1 year)
CK (2-200 U/L)	114.7	1952	2048	2828	1100	381	132	160.4
AST (15-40 U/L)	17.3	2294	1444	548	143	51	30	17.4
ALT (0-40 U/L)	13	1554	1355	1,083	640	339	79	16.2
AST/ALT ratio	1.33	1.48	1.07	0.51	0.22	0.15	0.38	1.07
TBIL (0-26 μmol/L)	19.3	25.7	26.3	34.2	17.7	15.4	13.9	20.7
DBIL (0-6.8 μmol/L)	6.4	9.1	10.2	12.5	6.7	5.1	3.7	7.3
ALB(35-50 g/L)	-	45	46	46	48	43	48	50.1
INR (0.8-1.2)	-	1.22	1.22	1.22	0.88	-	-	-
Cr (57-97 μmol/L)	88.9	98	94	90	88	79	93	92.9
Mb (28-72 ng/mL)	-	156	211	129	51.8	-	-	-
LDH (120-250 U/L)	183.3	1786	1047	538	279	200	197	183.7

Abbreviations: ALB, albumin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; Cr, creatinine; DBIL, direct bilirubin; INR, international normalized ratio; LDH, lactate dehydrogenase; Mb, myoglobin; TBIL, total bilirubin.

Table 2. Differential diagnosis of transaminase elevation in rhabdomyolysis.

Etiology	Key features	Exclusion criteria applied
Viral hepatitis (A, B, C, E)	ALT often > AST; positive serology	HAV IgM (-), HBsAg (-), HCV antibody (-), HEV IgM (-); only HEV IgG (+)
Drug-induced liver injury	Temporal relationship; exposure to hepatotoxic drugs	No exposure to hepatotoxic drugs during prior week
Alcoholic hepatitis	AST/ALT ratio > 2; heavy alcohol use	Remote history of alcohol intake (4 oz/week) but no recent use; AST/ALT ratio = 1.48
Autoimmune hepatitis	Elevated IgG; ANA/ASMA-positive	Not tested; unlikely due to acute self-limited clinical course
Ischemic hepatitis (shock liver)	Marked transaminase elevation with hypotension	No hypotension; BP remained stable throughout
Acute biliary obstruction	Predominant ALP/GGT elevation; biliary duct dilation	ALP normal; ultrasound showed no biliary dilation
Muscle-derived transaminase elevation	AST/ALT ratio > 3 early, rapid AST decline, normal INR, normal albumin, elevated CK	Elevated CK; AST declined by 76% within 48 hours; INR 1.22; albumin normal

Abbreviations: ALP, alkaline phosphatase; ALT, alanine aminotransferase; ANA, antinuclear antibody; ASMA, anti-smooth muscle antibody; AST, aspartate aminotransferase; BP, blood pressure; CK, creatine kinase; GGT, gamma-glutamyl transferase; HAV, hepatitis A virus; HBsAg, hepatitis B surface antigen; HCV, hepatitis C virus; HEV, hepatitis E virus; IgG, immunoglobulin G; IgM, immunoglobulin M; INR, international normalized ratio.

a low threshold for laboratory evaluation in patients presenting with myalgia, fatigue, or dark urine after unfamiliar exercise, regardless of perceived exercise intensity.

Elevated serum transaminase levels are common in rhabdomyolysis. A systematic review of the definition of rhabdomyolysis recommended defining the condition as a clinical syndrome of acute muscle weakness, myalgia, and muscle swelling combined with a CK level above 1000 IU/L or greater than 5 times the upper limit of normal [1]. The pathophysiology is multifactorial and includes direct release of AST from damaged muscle [18], oxidative stress caused by myoglobin-generated free radicals leading to hepatic ischemia-reperfusion injury [5], protease-mediated tissue injury [19], and

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ischemic hepatitis in cases of hypoperfusion [5]. In a cohort of patients with CK levels of at least 1000 U/L, Weibrecht et al reported AST elevation (> 40 U/L) in 93.1% and ALT elevation in 75.0% [20]. Lim et al found a moderate positive correlation between CK and AST ($r = 0.62$, $P < 0.001$) [4]. However, the magnitude of transaminase elevation in our case (AST > 2200 U/L and ALT > 1500 U/L) is remarkable given the peak CK level of 2828 U/L. In a study of muscle necrosis induced by extreme exercise, median AST and ALT levels were 2466 U/L and 497 U/L, respectively, with a mean CK level of 43 763 U/L. Our patient's AST level was comparable, whereas the ALT level was markedly higher. Published cases of rhabdomyolysis-associated liver injury typically involve CK levels in the tens of thousands. For example, a marathon runner had a CK level above 266 000 U/L [7], a high-altitude climber had a CK level of 33 765 U/L [8], and military trainees had CK levels above 50 000 U/L [21]. Our patient's CK level was substantially lower, yet his transaminase elevations were similar or even greater, suggesting that CK level does not reliably predict the degree of transaminase elevation.

Most reports have focused on patients with CK levels above 30 000 U/L and fulminant hepatic failure [6,7,10]. Recent case series and reviews similarly indicate that severe liver injury in rhabdomyolysis is almost always associated with CK levels greater than 10 000 to 50 000 U/L [22,23]. The novelty of the present case lies not in describing a new disease but in documenting a rare presentation of exertional rhabdomyolysis where moderate CK elevation was accompanied by transaminase levels typically observed in fulminant hepatic failure, whereas synthetic liver function remained preserved. This underrecognized pattern adds a boundary-condition example to the existing literature, which has largely focused on extreme CK elevations with overt liver failure.

Because skeletal muscle contains abundant AST, and even ALT—historically considered more liver specific—is present in skeletal muscle at lower concentrations [18], it is often unclear whether the marked transaminase elevations noted in patients with exertional rhabdomyolysis reflect true hepatocellular injury or simply the release of enzymes from damaged skeletal muscle [5,24]. This diagnostic dilemma may lead to unnecessary invasive investigations and treatments, including liver biopsy [5].

True acute liver failure associated with rhabdomyolysis typically presents with INR values above 2.5 [10,25]. Al Manasra et al noted that prothrombin time, bilirubin, and albumin levels can help identify concomitant liver injury [10]. Mohammad et al reported a similar case in which normal gamma-glutamyl transferase and INR values suggested a muscle source despite markedly elevated transaminase levels [22]. In our patient, the INR remained near normal throughout hospitalization (peak, 1.22), and bilirubin was only mildly elevated, indicating preserved synthetic function.

The AST level declined much more rapidly than the ALT level (76% vs 30% over 48 hours). This pattern is consistent with the shorter half-life of AST (~ 17 hours vs ~ 47 hours for ALT) and is characteristic of muscle-derived transaminase elevation [27]. Weibrecht et al observed that during recovery from rhabdomyolysis, AST declines in parallel with CK, whereas ALT declines more slowly [20]. On admission, our patient's AST/ALT ratio was 1.48. In skeletal muscle, the AST/ALT activity ratio is approximately 16.6: 1, compared with 2.58: 1 in the liver [18], which explains why AST is disproportionately elevated in muscle disorders. Jo et al confirmed that rhabdomyolysis causes AST-predominant transaminase elevation, regardless of underlying liver disease [26]. In our patient, although the ALT level was markedly elevated, the AST level remained higher than the ALT level. Furthermore, the TBIL level peaked at 34.2 µmol/L (~ 2.0 mg/dL) without coagulopathy, consistent with mild hepatic stress rather than true liver failure. In contrast, patients with heat stroke-related acute liver failure often have bilirubin levels above 3 mg/dL and severe coagulopathy [10,29].

Finally, the rapid normalization of liver biochemical parameters with conservative treatment strongly suggests a self-limited process. In the Acute Liver Failure Study Group series, heat stroke-induced liver failure was associated with substantial mortality and frequent requirement for liver transplantation [9]. Although the incidence of liver dysfunction among patients with rhabdomyolysis can reach 25%, liver function usually recovers [30-32]. Therefore, we believe that the transaminase elevation in our patient was predominantly muscle derived.

It is important to acknowledge the borderline nature of this case. Our patient's CK level (2828 U/L) clearly exceeded the diagnostic threshold for rhabdomyolysis (> 1000 U/L) but remained well below the values typically observed in severe cases (> 10 000-50 000 U/L). Similarly, the transaminase elevations were striking by liver disease standards but—given the normal INR and albumin findings—were most likely muscle derived. This borderline presentation is precisely where clinical decision-making is most challenging and, in our view, where this case has its greatest educational value. It demonstrates that even when transaminase levels reach values commonly associated with liver failure, conservative management may be appropriate if synthetic liver function remains preserved and alternative causes of liver injury have been excluded.

This comparison emphasizes that most published cases involve CK levels above 30 000 U/L with clinically significant coagulopathy; our case is distinguished by the combination of moderate CK elevation, marked transaminase elevation, and preserved synthetic function (**Table 3**).

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Table 3. Comparison of the present case with previously reported cases of rhabdomyolysis-associated liver injury.

Feature	Aquilina et al (2018) [7]	Yeh et al (2022) [8]	Al Manasra et al (2018) [10]	Present case
Trigger	Marathon	High-altitude climbing	Post-hepatectomy	3000-m run
Peak CK (U/L)	> 266 000	33 765	14 635	2828
Peak AST (U/L)	> 6000	2882	1656	2294
Peak ALT (U/L)	> 2000	2259	1134	1554
INR	> 2.5	Prolonged (17.8 s)	Normalized with hydration	1.22 (normal)
Treatment	ICU, dialysis	ICU, N-acetylcysteine	Aggressive hydration	Conservative management only
Outcome	Recovered	Recovered	Recovered	Recovered

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; ICU, intensive care unit; INR, international normalized ratio.

The present findings suggest that, in cases of exertional rhabdomyolysis with moderate CK elevation but marked transaminase elevation, conservative management with close monitoring may be appropriate when the patient is hemodynamically stable, liver synthetic function is preserved, AST exceeds ALT, and the AST level declines by more than 50% within 48 hours.

This case has several limitations. A liver biopsy was not performed; therefore, mild hepatocellular injury cannot be definitively excluded. Autoimmune hepatitis and other rare causes were not investigated, although the clinical course makes these diagnoses unlikely. Additionally, inherited metabolic myopathies (eg, carnitine palmitoyltransferase II deficiency and McArdle disease) were not evaluated by genetic testing. However, the patient had no history of recurrent exercise intolerance, myalgia, or episodes of dark urine, making these conditions less likely. Urine myoglobin was not quantitatively measured; notably, the progressive normalization of urine color and restoration of adequate urine output suggested resolving myoglobinuria. Given the single-case nature of this report, the findings cannot be generalized without confirmation in larger studies. Furthermore, the interpretive pattern described here was observed in a single hemodynamically stable patient without shock, encephalopathy, or overt coagulopathy and should not be extrapolated to patients exhibiting progressive hyperbilirubinemia or other signs of primary liver failure. Accordingly, this case is best regarded as a teaching example for the differential diagnosis of marked transaminase elevation, rather than as a challenge to existing liver failure guidelines.

Conclusions

Exertional rhabdomyolysis can produce marked transaminase elevation despite moderate CK elevation; the degree of CK elevation may not be linearly correlated with AST and ALT levels. This case documents the unusual phenotype of marked transaminase elevation (AST > 2200 U/L and ALT > 1500 U/L) with preserved synthetic liver function in a patient showing moderate CK elevation (peak, 2828 U/L). The borderline nature of this case—CK above the diagnostic threshold but well below levels typically observed in severe rhabdomyolysis—highlights the clinical importance of recognizing that marked transaminase elevation does not necessarily indicate primary liver injury when synthetic liver function is preserved. In selected patients, conservative management may be appropriate after alternative causes of liver injury have been excluded. Such findings require validation in larger studies and should not replace a thorough evaluation for liver disease in patients without a clear alternative explanation.

Department and Institution Where Work Was Done

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Patient Consent

Written informed consent was obtained from the patient for publication of this case report.

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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