Cureus
Part of SPRINGER NATURE

Open Access Case Report

Rhabdomyolysis After a Low-Energy Fall With an Intertrochanteric Fracture: A Case Report

Takshak Shankar [1], Nidhi Kaeley [1], Param P. Singh [1], Parvathy Sasidharan [1], Tarun Rana [1], Deepanjan Mudi [1]

1. Emergency Medicine, All India Institute of Medical Sciences, Rishikesh, IND

Corresponding author: Nidhi Kaeley, drnidhi_kaeley@yahoo.com

Review began 10/30/2026
Review ended 11/02/2026
Published 11/04/2026

© **Copyright** 2026
Shankar et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.119099

Abstract

Rhabdomyolysis is an uncommon but potentially life-threatening complication of traumatic muscle injury. We report an elderly man who developed rhabdomyolysis following an apparently low-energy slip and fall resulting in an isolated intertrochanteric fracture. He presented approximately eight hours after the injury with persistent inability to bear weight and subsequently developed dark-colored urine. Initial investigations revealed elevated creatine kinase and lactate dehydrogenase, with urine dipstick positivity for blood and minimal erythrocytes on microscopy, supporting the diagnosis of rhabdomyolysis. Despite intravenous fluid therapy and discontinuation of atorvastatin, his urine output progressively declined, followed by anuria, acute kidney injury, and refractory hyperkalemia with electrocardiographic changes requiring a single session of hemodialysis. Renal function and urine output subsequently recovered, and definitive fracture fixation was performed. He was discharged ambulatory, and atorvastatin was successfully reintroduced at a lower dose. This case highlights the need to consider rhabdomyolysis after seemingly minor trauma in older adults and emphasizes serial monitoring for evolving renal and electrolyte complications.

Categories: Trauma, Emergency Medicine, Orthopedics
Keywords: acute kidney injury, refractory hyperkalemia, stable intertrochanteric fracture, statin-induced rhabdomyolosis, traumatic rhabdomyolysis

Introduction

Rhabdomyolysis is a clinical syndrome caused by skeletal muscle necrosis, resulting in the release of intracellular components, including myoglobin, sarcoplasmic proteins, and electrolytes, into the circulation. Its clinical spectrum ranges from asymptomatic elevation of serum muscle enzymes to life-threatening complications, including acute kidney injury and severe electrolyte abnormalities [1-3]. Skeletal muscle necrosis may result from diverse insults; however, the final common pathway involves increased intracellular free ionized calcium within the cytoplasm and mitochondria, often due to adenosine triphosphate (ATP) depletion and/or direct plasma membrane injury [4,5]. Membrane disruption further accelerates ATP depletion and cellular damage. Etiologies are broadly classified as traumatic or compression-related, non-traumatic exertional, and non-traumatic non-exertional, including drugs and toxins [6,7]. Although trauma is a major cause, rhabdomyolysis following low-energy trauma is uncommon [3,8]. We report an elderly patient who developed rhabdomyolysis following a slip-and-fall incident at home.

Case Presentation

An elderly man in his mid-eighties presented to the emergency department with left hip pain and an inability to bear weight on the left lower limb following a reported slip and fall in the bathroom at home during the night. His family, alerted by the sound of the fall, attended to him immediately and transported him to a nearby hospital, where he received analgesia before being referred to our tertiary care center. He presented approximately eight hours after the injury. Although his pain had improved considerably following analgesia, he remained unable to bear weight on the affected limb. His medical history was significant for coronary artery disease, for which a stent had been placed in the left anterior descending coronary artery approximately five years earlier. He was receiving aspirin 75 mg and atorvastatin 40 mg and had a documented left ventricular ejection fraction of 40%. He had not followed up with his primary care physician for more than two months and had been taking atorvastatin 40 mg for the preceding six months. There was no history of tobacco, alcohol, or other substance use, and he denied taking any other medications.

On examination, he was conscious, oriented, and hemodynamically stable, with no clinical signs of dehydration. Swelling was noted over the left upper thigh without bruising or features suggestive of compartment syndrome. Distal neurovascular status was intact, and no other injuries were identified. Radiographs of the pelvis and bilateral hips confirmed a closed left intertrochanteric fracture (Figure *1*).

How to cite this article
Shankar T, Kaeley N, Singh P P, et al. (November 04, 2026) Rhabdomyolysis After a Low-Energy Fall With an Intertrochanteric Fracture: A Case Report. Cureus 18(11): e119099. DOI 10.7759/cureus.119099

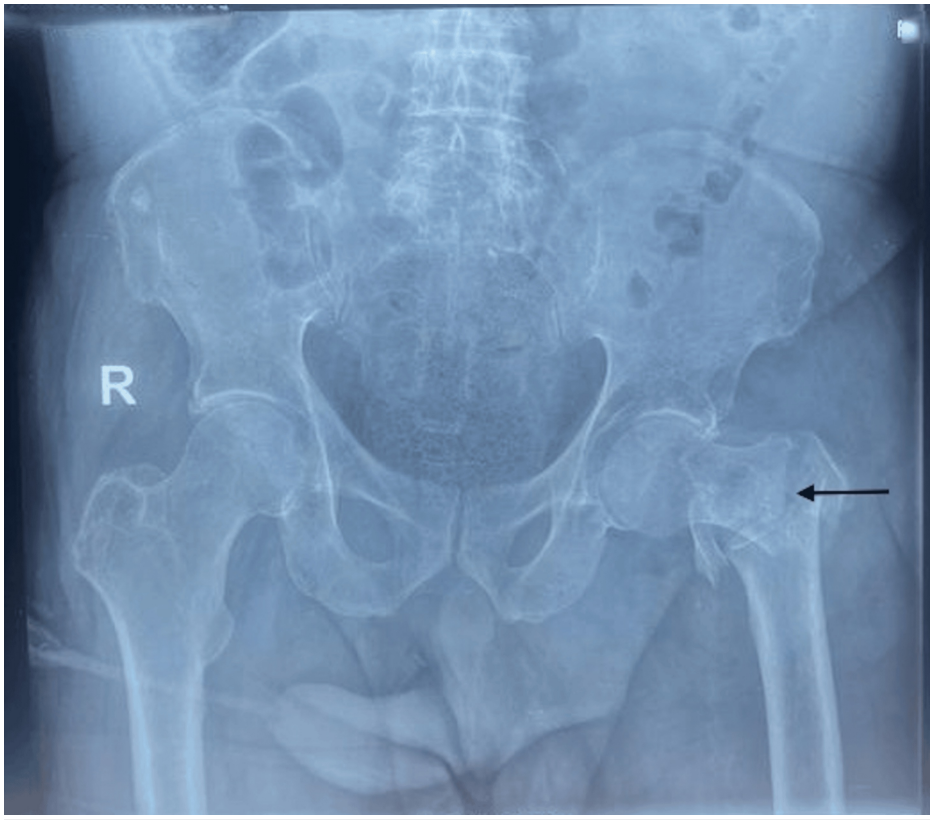


FIGURE 1: Radiograph of pelvis with bilateral hip showing a left intertrochanteric fracture (black arrow)

Following Foley catheterization, dark-colored urine was observed. Intravenous fluid resuscitation was initiated promptly, resulting in urine output of approximately 40 mL/hour; however, the urine remained persistently dark. The laboratory investigations on arrival are summarized in Table *1*.

Parameters	Patient value	Reference range
Hemoglobin (grams per deciliter)	11.7	13-17
Total leucocyte count (cells per cubic millimeter)	7.15	4-11
Platelet count (cells per cubic millimeter)	240	150-400
Total bilirubin (milligrams per deciliter)	0.71	0.3-1.2
Direct bilirubin (milligrams per deciliter)	0.2	0-0.2
ALT (units per liter)	55	0-50
AST (units per liter)	69	0-50
Blood urea (milligrams per deciliter)	61	17-43
Serum creatinine (milligrams per deciliter)	1.03	0.72-1.18
Serum sodium (millimoles per liter)	136	136-146
Serum potassium (millimoles per liter)	5	3.5-5.1
Serum chloride (millimoles per liter)	101	101-109
Serum total calcium (milligrams per deciliter)	7.4	8.8-10.6
Serum uric acid (milligrams per deciliter)	3.7	3.5-7.2
Serum phosphorus (milligrams per deciliter)	3.1	2.5-4.5
INR	1.1	1.02
Creatine kinase (units per liter)	1468	46-171
Lactate dehydrogenase (units per liter)	397	0-248
Urine routine and microscopy	Blood present on dipstick assessment RBC- 1/hpf	

TABLE 1: Laboratory parameters of the patient on arrival

ALT - alanine aminotransferase; AST - aspartate aminotransferase; INR - international normalized ratio

Rhabdomyolysis was diagnosed based on dark-colored urine and elevated serum creatine kinase and lactate dehydrogenase levels in the setting of recent trauma, advanced age, and concomitant statin therapy. Thyroid function was normal. Statin therapy was discontinued during the hospital stay, and intravenous fluids and supportive management were continued. Serum creatine kinase peaked at 2450 U/L on hospital day two. Despite treatment, urine output declined to 20 mL/hour on day two and progressed to anuria for more than 12 hours on day two, with serum creatinine increasing to 2.5 mg/dL, consistent with acute kidney injury. Fluid therapy was adjusted according to his clinical status. He subsequently developed refractory hyperkalemia with electrocardiographic changes, necessitating a single session of hemodialysis. Following dialysis, urine output gradually improved, and serum creatinine returned to baseline. After normalization of laboratory parameters, he underwent closed reduction and intramedullary femoral nailing for definitive fracture management. His hospital course was complicated by hospital-acquired pneumonia, which responded to treatment. He was discharged on hospital day 15 in stable condition and was ambulatory. Atorvastatin 20 mg once daily was initiated at discharge with regular monitoring. At the two-month follow-up, he remained ambulatory and was tolerating statin therapy well. Written informed consent was obtained from the patient for publication of this case report.

Discussion

The etiology of rhabdomyolysis is frequently multifactorial. In a review of 475 hospitalized patients, 60% had more than one risk factor, with drugs being the most common individual contributor, implicated in 46% of cases. Causative agents included both prescription and recreational drugs, while 10% of cases were associated with underlying neuromuscular disorders [9]. Similarly, in another study, trauma was the most common associated condition (26%), followed by immobilization (18%), sepsis (10%), vascular surgery (8%), and cardiac surgery (6%) [10]. Other recognized causes include infections, medications, illicit substances, alcohol, surgery, and strenuous exercise [9-11].

Among trauma patients, risk factors include polytrauma, crush injuries, compartment syndrome, age >55 years, Injury Severity Score >16, penetrating trauma with vascular injury, male sex, and BMI >30 kg/m². Patients with vascular injury or ischemic muscle damage followed by reperfusion are also at increased risk [12-14]. Nevertheless, fewer than one-fifth of rhabdomyolysis cases are attributable to direct traumatic muscle injury, with metabolic and medical causes accounting for most cases [7].

Numerous medications have been associated with rhabdomyolysis, including statins, antipsychotics, antibiotics, antihistamines, and glucocorticoids. An analysis of rhabdomyolysis reports identified statins as the most frequently implicated class [9,15,16]. Statin-associated muscle toxicity ranges from myalgia and myopathy to myositis and rhabdomyolysis. Proposed mechanisms include reduced coenzyme Q10 levels, mitochondrial dysfunction, impaired cellular respiration, altered calcium homeostasis, activation of apoptotic pathways, and inhibition of HMG-CoA reductase activity [17,18]. Although rhabdomyolysis is an uncommon adverse effect of statin therapy, men are approximately twice as likely as women to develop rhabdomyolysis. Advanced age is an additional risk factor for statin-associated muscle injury [17,18].

Clinical manifestations of rhabdomyolysis range from asymptomatic elevation of muscle enzymes to severe systemic illness. Common features include muscle pain or tenderness, weakness, swelling, and tea-colored urine resulting from myoglobinuria. However, this classic triad of muscle pain, weakness, and dark urine occurs in only 1-10% of patients. Skin changes associated with ischemic tissue injury, including discoloration and blistering, also occur in fewer than 10% of cases [7,19]. Severe disease may present with fever, nausea, vomiting, abdominal pain, malaise, and tachycardia [4]. Systemic complications include acute kidney injury, cardiac arrhythmias, and coagulopathy, while common metabolic abnormalities include hypovolemia, hypocalcemia, hyperkalemia, hyperphosphatemia, hyperuricemia, and high-anion-gap metabolic acidosis [3,6,7].

Laboratory evaluation typically demonstrates elevated serum creatine kinase (CK), myoglobin, lactate dehydrogenase (LDH), aspartate aminotransferase (AST), potassium, and creatinine. CK is the biochemical hallmark, with levels exceeding five times the upper limit of normal or >1000 IU/L generally considered diagnostic. Serial CK measurements are recommended to monitor disease progression until peak levels are reached, typically within 24-72 hours, and may be discontinued once a consistent downward trend is established [3,11,20].

Management is primarily supportive and aims to prevent pigment-induced acute kidney injury, correct fluid and electrolyte abnormalities, eliminate precipitating factors, and treat complications. Early intravenous fluid resuscitation is the cornerstone of treatment in patients at risk of acute kidney injury. Lactated Ringer's solution or saline may be used, with an initial infusion rate of approximately 400 mL/hour and subsequent goal-directed adjustment to maintain urine output of 1-3 mL/kg/hour, up to 300 mL/hour. Current evidence does not support routine sodium bicarbonate or diuretic therapy, including mannitol and loop diuretics, for prevention of acute kidney injury because available studies have important methodological limitations [3].

This case illustrates that rhabdomyolysis may occur following an apparently low-energy traumatic event in a susceptible older adult. Although the intertrochanteric fracture and associated soft-tissue injury were temporally associated with rhabdomyolysis, the etiology was likely multifactorial. Advanced age and concurrent atorvastatin therapy may have increased susceptibility to muscle injury; however, atorvastatin exposure was considered a potential contributing factor rather than a proven cause. The absence of reported interacting medications and normal thyroid function reduced, but did not exclude, the possibility of statin-associated muscle toxicity. The subsequent successful reintroduction of atorvastatin at 20 mg daily further supports a cautious interpretation of causality. The presence of dark-colored urine, urine dipstick positivity for blood with few erythrocytes on microscopy, and elevated serum creatine kinase supported the diagnosis of rhabdomyolysis.

The subsequent progression to oliguria, anuria, hyperkalemia, and acute kidney injury highlights the importance of serial monitoring, even when the initial creatine kinase elevation is modest. Importantly, the magnitude of creatine kinase elevation may not reliably predict the severity of renal impairment [21]. This case underscores the need for early assessment and serial monitoring of creatine kinase, renal function, electrolytes, and urine findings in older adults following low-energy falls, particularly when additional risk factors for muscle injury are present.

Conclusions

Rhabdomyolysis is a potentially life-threatening syndrome with diverse etiologies and a wide clinical spectrum. Although commonly associated with significant trauma, it may also occur following seemingly minor injuries, particularly in individuals with additional predisposing factors such as advanced age and concomitant medication use. This case emphasizes the importance of maintaining a high index of suspicion for rhabdomyolysis even after low-energy trauma. Early recognition, appropriate laboratory evaluation, and timely initiation of supportive management are crucial to prevent acute kidney injury and other potentially serious complications. Prompt diagnosis and individualized management can significantly improve outcomes in susceptible patients.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Takshak Shankar, Nidhi Kaeley, Param P. Singh, Parvathy Sasidharan

Acquisition, analysis, or interpretation of data: Takshak Shankar, Nidhi Kaeley, Param P. Singh, Parvathy Sasidharan, Tarun Rana , Deepanjan Mudi

Drafting of the manuscript: Takshak Shankar, Nidhi Kaeley, Param P. Singh, Parvathy Sasidharan, Tarun Rana , Deepanjan Mudi

Critical review of the manuscript for important intellectual content: Takshak Shankar, Nidhi Kaeley, Parvathy Sasidharan

Supervision: Takshak Shankar, Nidhi Kaeley

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Aleckovic-Halilovic M, Pjanic M, Mesic E, Storrar J, Woywodt A: From quail to earthquakes and human conflict: a historical perspective of rhabdomyolysis. Clin Kidney J. 2021, 14:1088-96. 10.1093/ckj/sfaa075
2. Mokhtari AK, Maurer LR, Christensen MA, et al.: Rhabdomyolysis in severe COVID-19: male sex, high body mass index, and prone positioning confer high risk. J Surg Res. 2021, 266:35-43. 10.1016/j.jss.2021.03.049
3. Kodadek L, Carmichael Ii SP, Seshadri A, et al.: Rhabdomyolysis: An American Association for the Surgery of trauma critical care Committee clinical consensus document. Trauma Surg Acute Care Open. 2022, 7:e000836. 10.1136/tsaco-2021-000836
4. Giannoglou GD, Chatzizisis YS, Misirli G: The syndrome of rhabdomyolysis: pathophysiology and diagnosis . Eur J Intern Med. 2007, 18:90-100. 10.1016/j.ejim.2006.09.020
5. Khan FY: Rhabdomyolysis: a review of the literature . Neth J Med. 2009, 67:272-83.
6. Rout P, Chippa V, Adigun R: Rhabdomyolysis. StatPearls Publishing, Treasure Island, FL; 2026.
7. Gabow PA, Kaehny WD, Kelleher SP: The spectrum of rhabdomyolysis . Medicine (Baltimore). 1982, 61:141-52. 10.1097/00005792-198205000-00002
8. Banerjee S, Gupta A, Jeshwanth N: Acute kidney injury secondary to rhabdomyolysis: a rare complication of low-energy trauma. J Orthop Case Rep. 2021, 11:16-9. 10.13107/jocr.2021.v11.i01.1946
9. Melli G, Chaudhry V, Cornblath DR: Rhabdomyolysis: an evaluation of 475 hospitalized patients . Medicine (Baltimore). 2005, 84:377-85. 10.1097/01.md.0000188565.48918.41
10. McMahon GM, Zeng X, Waikar SS: A risk prediction score for kidney failure or mortality in rhabdomyolysis . JAMA Intern Med. 2013, 173:1821-8. 10.1001/jamainternmed.2013.9774
11. Gupta A, Thorson P, Penmatsa KR, Gupta P: Rhabdomyolysis: revisited. Ulster Med J. 2021, 90:61-9.
12. Brown CV, Rhee P, Chan L, Evans K, Demetriades D, Velmahos GC: Preventing renal failure in patients with rhabdomyolysis: do bicarbonate and mannitol make a difference?. J Trauma. 2004, 56:1191-6. 10.1097/01.ta.0000130761.78627.10
13. Oda J, Tanaka H, Yoshioka T, et al.: Analysis of 372 patients with Crush syndrome caused by the Hanshin-Awaji earthquake. J Trauma. 1997, 42:470-6. 10.1097/00005373-199703000-00015
14. Brown CV, Rhee P, Evans K, Demetriades D, Velmahos G: Rhabdomyolysis after penetrating trauma. Am Surg. 2004, 70:890-2.
15. Oshima Y: Characteristics of drug-associated rhabdomyolysis: analysis of 8,610 cases reported to the U.S. Food and Drug Administration. Intern Med. 2011, 50:845-53. 10.2169/internalmedicine.50.4484
16. Teng C, Baus C, Wilson JP, Frei CR: Rhabdomyolysis associations with antibiotics: a pharmacovigilance study of the FDA Adverse Event Reporting System (FAERS). Int J Med Sci. 2019, 16:1504-9. 10.7150/ijms.38605
17. Sharma U: Statin-induced delayed rhabdomyolysis. BMJ Case Rep. 2019, 12:e231125. 10.1136/bcr-2019-231125
18. Morris R, Bu K, Han W, et al.: The association between statin drugs and rhabdomyolysis: an analysis of FDA Adverse Event Reporting System (FAERS) data and transcriptomic profiles. Genes (Basel). 2025, 16:248. 10.3390/genes16030248
19. Huerta-Alardín AL, Varon J, Marik PE: Bench-to-bedside review: rabdomyolysis - an overview for clinicians . Crit Care. 2005, 9:158-69. 10.1186/cc2978

20. Stahl K, Rastelli E, Schoser B: A systematic review on the definition of rhabdomyolysis . J Neurol. 2020, 267:877-82. 10.1007/s00415-019-09185-4
21. Młynarska E, Krzemińska J, Wronka M, Franczyk B, Rysz J: Rhabdomyolysis-induced AKI (RIAKI) including the role of COVID-19. Int J Mol Sci. 2022, 23:8215. 10.3390/ijms23158215