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Severe Atraumatic Rhabdomyolysis Complicated by Bilateral Lower-Extremity Compartment Syndrome: A Case Report

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Abstract

Rhabdomyolysis involves breakdown of skeletal muscle with release of intracellular contents into circulation. Although classically linked to trauma, nontraumatic and nonexertional etiologies now account for most hospitalized cases. Compartment syndrome is a well-documented complication in which massive myocyte injury and edema raise intracompartmental pressure and propagate a cycle of ischemia and necrosis. The co-occurrence of severe atraumatic rhabdomyolysis and compartment syndrome without an identifiable precipitant is rare.

A 48-year-old Chuj-speaking male with no known past medical history presented with severe bilateral lower extremity pain, swelling, and paresthesias beginning on awakening with associated periumbilical abdominal pain, nausea, and emesis. He reported only moderate social alcohol intake the prior evening and denied trauma, prolonged immobility, exertion, heat exposure, illicit drug use, medications, or recent illness. He was tachycardic, tachypneic, and hypoxemic with tense lower extremity compartments and a right-sided pulse deficit identified only by Doppler. Creatine kinase (CK) was profoundly elevated at 255,720 U/L; CK-MB 1,897 ng/mL, with leukocytosis, polycythemia (hemoglobin 20.1 g/dL) reflecting hemoconcentration, acute kidney injury (AKI), aspartate aminotransferase (AST)-predominant transaminitis, hyperphosphatemia, and severe high anion-gap metabolic acidosis (lactate 8.9 mmol/L). Serum ethanol was 76 mg/dL. EKG decreased the likelihood of acute coronary syndrome. Imaging excluded fracture and deep vein thrombosis (DVT). CT angiography showed proximal right anterior tibial and peroneal artery occlusion with preserved distal perfusion and bilateral muscle edema consistent with rhabdomyolysis and secondary bilateral compartment syndrome. The arterial and hematologic abnormalities likely represented downstream consequences.

The patient underwent emergent right thigh and bilateral four-compartment lower leg fasciotomies. His 26-day hospitalization was complicated by dialysis-dependent AKI requiring continuous renal replacement therapy (CRRT), transition to intermittent hemodialysis, staged fasciotomy closures, and new-onset cardiac dysfunction. He demonstrated renal recovery with a final hemodialysis session approximately one month after presentation and creatinine declining to 1.4 mg/dL.

No acquired etiology was definitively identified, but the presentation may be consistent with unwitnessed positional rhabdomyolysis in the setting of ethanol exposure. This is supported by onset on awakening, dependent right-sided predominance, and alcohol myotoxicity with sustained compression. Limited interpretation in the patient's preferred language may have impacted history-taking. Equitable care requires professional interpretation in the patient's preferred language. This case underscores the need for a high index of suspicion for compartment syndrome in any patient with severe rhabdomyolysis regardless of mechanism, as early recognition and aggressive management are critical to preventing AKI, limb loss, and death. When no acquired cause is found, systematic evaluation for inherited myopathy is warranted. An inherited myopathy cannot be excluded in this case.

Categories: Emergency Medicine
Keywords: alcohol use, creatine kinase, emergency fasciotomy, language barrier, metabolic acidosis with acute kidney injury, rhabdomyolysis and compartment syndrome

Introduction

Rhabdomyolysis is characterized by the breakdown of skeletal muscle and release of intracellular contents such as creatine kinase (CK), myoglobin, potassium, and phosphate into circulation [1,2]. The etiologies of rhabdomyolysis are categorized as traumatic, exertional, and nontraumatic nonexertional [2]. Nontraumatic causes include exogenous toxins (alcohol, statins, illicit drugs), infections, electrolyte disturbances (hypokalemia, hypophosphatemia), endocrine abnormalities (hypothyroidism, diabetic ketoacidosis), autoimmune myopathies, and genetic or metabolic myopathies [1-3]. Although rhabdomyolysis is classically associated with crush injuries and trauma, nontraumatic etiologies now represent the majority of

hospitalized cases [1-3].

Compartment syndrome occurs when pressure increases within a fascial compartment and compromises tissue perfusion, leading to ischemia and necrosis [4]. Although most commonly associated with direct trauma, up to 30% of cases of acute extremity compartment syndrome occur without fracture, and rhabdomyolysis is a well-documented secondary cause [5,6]. The pathophysiology in this setting involves massive muscle edema from myocyte injury, which raises intracompartmental pressure and creates a positive feedback loop of ischemia, further muscle necrosis, and worsening swelling [6,7].

We present a 48-year-old man with no known medical history who developed severe atraumatic rhabdomyolysis, with a peak CK above 255,000 U/L, complicated by bilateral lower extremity compartment syndrome without an identifiable acquired precipitant. Compartment syndrome complicating atraumatic rhabdomyolysis is usually unilateral, and bilateral involvement is infrequently described. In this case, diagnosis was further complicated by a proximal arterial occlusion on CT angiography, which raised concern for a primary vascular cause, and by limited language concordance during history-taking. This combination of bilateral compartment syndrome, extreme CK elevation without a trigger, and a confounding vascular finding, made harder to diagnose by a language barrier that limited history-taking, has, to our knowledge, rarely been described in a single patient.

Case Presentation

Emergency department (ED) course

A 48-year-old Chuj-speaking male with no known past medical history presented to the ED for severe bilateral lower extremity pain. His pain began upon awakening and was associated with swelling, paresthesias, and difficulty with ambulation. The right leg was more severely affected than the left. He also experienced periumbilical abdominal pain with associated nausea and emesis. The patient reported consumption of a moderate amount of alcohol the evening prior but denied excessive alcohol consumption or chronic use. He denied any injury, trauma, prolonged immobility or irregular positioning, significantly increased physical activity or heat exposure, dehydration, insect bites, illicit substance use, or recent illness. He denied a family history of a similar occurrence. Additionally, he was not taking any prescription medications.

On examination, the patient was ill-appearing and in acute distress secondary to pain. He was afebrile and maintained a blood pressure around 105/75 mmHg, but he was tachycardic at 138 beats per minute, tachypneic at 24 breaths per minute, and required two liters of supplemental oxygen by nasal cannula due to a decrease in oxygen saturation to 89% on room air. Cardiac and pulmonary examinations were unremarkable. Abdominal examination revealed a soft abdomen with tenderness to palpation in the periumbilical region without guarding or rebound tenderness. Examination of bilateral lower extremities revealed tense compartments, particularly in the calves, with the right leg more severely affected. Sensation was grossly intact in the bilateral lower extremities, but range of motion and motor examination were limited by pain. There was no erythema, abrasion, deformity, or rash on either lower extremity. Popliteal and femoral pulses were palpable bilaterally. The dorsalis pedis (DP) and posterior tibialis (PT) pulses were palpable on the left but difficult to identify on the right; they were ultimately identified on the right by Doppler ultrasonography.

The initial differential diagnosis included infectious myositis, necrotizing soft tissue infection (NSTI), rhabdomyolysis, deep vein thrombosis (DVT) with or without pulmonary embolism, acute arterial occlusion, compartment syndrome, and occult fracture.

Laboratory studies, shown in Table *1*, revealed markedly abnormal results across multiple domains. Most notably, CK was profoundly elevated. A complete blood count demonstrated significant leukocytosis, polycythemia, and thrombocytosis. The comprehensive metabolic panel was notable for acute kidney injury (AKI), hyperglycemia, markedly elevated transaminases, elevated alkaline phosphatase, hypocalcemia, and hyperphosphatemia. Venous blood gas analysis revealed severe metabolic acidosis with markedly elevated lactate. The urine drug screen was positive for benzodiazepines (administered prior to collection) and opiates/fentanyl (administered in the ED). The volatile alcohol screen was negative, and the serum ethanol level was mildly elevated.

Laboratory Test	Patient Value	Reference Value
Creatine kinase (CK)	255,720 U/L	~30–200 U/L
CK-MB	1,897 ng/mL	~0–5 ng/mL
WBC	34.1 × 10^3/μL	4.0–11.0 × 10^3/μL
Hemoglobin	20.1 g/dL	13.5–17.5 g/dL (male)
Hematocrit	59.40%	41–53% (male)
Platelets	546 × 10^3/μL	150–450 × 10^3/μL
Creatinine	2.36 mg/dL	0.7–1.3 mg/dL
eGFR/GFR	33.1 mL/min/1.73 m^2	≥60 mL/min/1.73 m^2
Glucose	199 mg/dL	70–99 mg/dL (fasting)
AST	3,958 U/L	~10–40 U/L
ALT	773 U/L	~7–56 U/L
Alkaline phosphatase	138 U/L	~40–129 U/L
Calcium	8.0 mg/dL	8.5–10.5 mg/dL
Phosphorus	10.2 mg/dL	2.5–4.5 mg/dL
Venous pH	7.18	7.31–7.41
Venous pCO_2	28 mmHg	~41–51 mmHg
Bicarbonate (HCO_3^-)	10 mEq/L	22–29 mEq/L
Lactate	8.9 mmol/L	~0.5–2.2 mmol/L
Serum ethanol	76 mg/dL	0 mg/dL
Volatile alcohol screen	Negative	Negative
Urine benzodiazepines	Positive	Negative
Urine opiates/fentanyl	Positive	Negative

TABLE 1: Laboratory studies obtained in the emergency department

This table displays laboratory values obtained for this patient during his time in the Emergency Department prior to his inpatient hospitalization.

AST: aspartate aminotransferase; ALT: alanine aminotransferase; eGFR: estimated glomerular filtration rate

Initial EKG showed sinus tachycardia and no acute ischemic changes. Bilateral lower extremity radiographs showed no acute osseous abnormality, and bilateral lower extremity venous duplex ultrasonography showed no evidence of DVT. Computed tomography angiography (CTA) of the aorta, abdomen, and lower extremities with runoff shown in Figure *1* demonstrated occlusion of the right anterior tibial and peroneal arteries proximally, with the posterior tibial artery patent below the ankle. Low-attenuation changes were identified throughout bilateral thigh and calf musculature, right worse than left, and this was found to be likely consistent with myositis or edema.

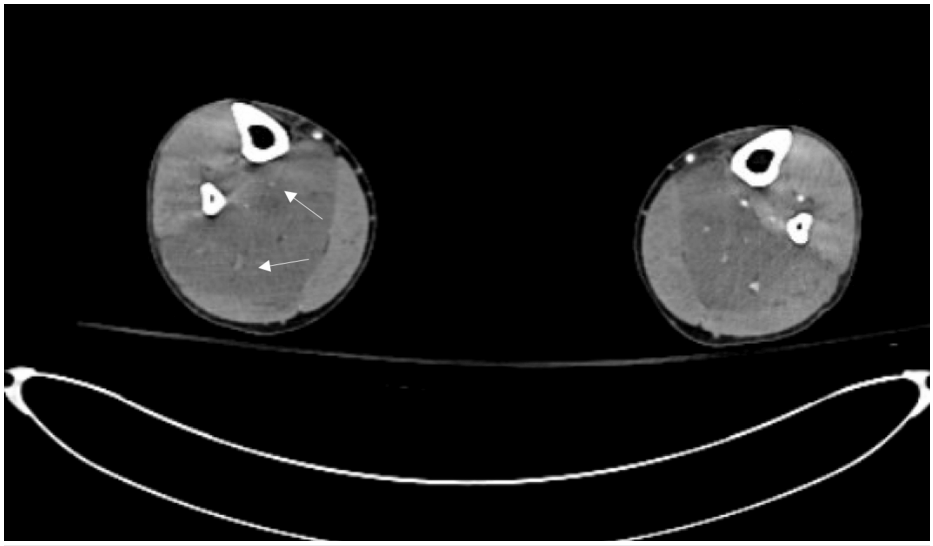


FIGURE 1: CTA of lower extremities distal to right-sided arterial occlusion

This figure shows the CTA image of the bilateral lower extremities distal to the arterial occlusion in the right anterior tibial and peroneal arteries, with low-attenuation changes identified throughout the bilateral thigh and calf musculature. The arrows point to areas that lack arterial flow secondary to the occlusion in the right lower extremity.

CTA: computed tomography angiography

The patient received ketorolac in the ambulance and was placed on supplemental oxygen at 2 L/min via nasal cannula upon arrival due to low oxygen saturation described above. In the ED, he received acetaminophen, morphine, ondansetron, and intravenous fluid resuscitation with both lactated Ringer's and normal saline boluses. Given concern for possible NSTI, empiric broad-spectrum antibiotics were initiated, and fentanyl was administered for refractory pain. Trauma/acute care surgery (TACS), orthopedic surgery, vascular surgery, and the medical intensive care unit (MICU) were consulted. Orthopedic surgery promptly evaluated the patient and made a clinical diagnosis of compartment syndrome without measuring compartment pressures; they recommended the immediate performance of lateral posterior compartment fasciotomy of the right thigh and four-compartment fasciotomy of the bilateral lower legs in the operating room (OR). Vascular surgery evaluated the right anterior tibial artery occlusion and recommended no acute intervention given a patent posterior tibial artery with intact distal perfusion.

Hospital course

Following the initial procedure, the patient was hospitalized for a total of 26 days, during which time the surgical team performed a series of staged fasciotomy closures and wound care procedures. The patient initially required ICU admission with intubation and continuous renal replacement therapy (CRRT) but was later extubated successfully and transitioned to intermittent hemodialysis. His course was complicated by dialysis-dependent AKI, electrolyte derangements, and new-onset mild left ventricular systolic dysfunction with severely reduced right ventricular function (ejection fraction 41-45%). At the time of discharge, the patient was afebrile and ambulatory with physical therapy, pain had improved, and all fasciotomy sites were closed. Initial CK levels had dropped from a peak in the ED of 255,730 U/L to 565 U/L by day 24 of admission. Following discharge, the patient followed up with vascular surgery, cardiology, and primary care and presented to the ED three times weekly for hemodialysis. The patient demonstrated renal recovery, undergoing his final hemodialysis session approximately one month after initial presentation, with a corresponding decline in serum creatinine to 1.4 mg/dL.

Discussion

Several features distinguished this presentation of severe atraumatic rhabdomyolysis with associated compartment syndrome. First, the onset of symptoms on awakening rather than during or after exertion or injury initially raised concern for atraumatic causes and prompted thorough questioning. Although rhabdomyolysis is classically associated with crush injury, metabolic and medical causes predominate over trauma [3]. Common causes include exogenous toxins (alcohol, cocaine, opioids, phencyclidine, statins), positional injury, infection (influenza, Epstein-Barr virus, *Streptococcus pyogenes, Staphylococcus aureus*), electrolyte or endocrine derangements (hypokalemia, hypophosphatemia, hypothyroidism, diabetic ketoacidosis), dysautonomic syndromes (malignant hyperthermia, heat stroke, neuroleptic malignant

syndrome), and inherited metabolic or structural myopathies [2,3]. Our patient denied trauma, exertion, heat exposure, illicit substance use, prescription medications, recent illness, irregular sleeping positions, and any personal or family history of similar episodes, and he reported only moderate social alcohol intake. The history was therefore unrevealing, leaving the etiology of this presentation unexplained at the time of initial evaluation. Additionally, the initial CK of 255,720 U/L markedly exceeded values typically reported in large rhabdomyolysis case series, where presenting levels average roughly 30,000 IU/L and maximums generally fall below 110,000 IU/L [8,9].

Several diagnostic findings highlighted the severity of this patient's presentation and assisted in narrowing the differential diagnosis. The polycythemia found during the ED course likely represented significant hemoconcentration from massive third-spacing of intravascular fluid into damaged muscle compartments [10]. Its magnitude served as an indicator of the volume of muscle involved and predicted the aggressive resuscitation requirement [11]. The high anion-gap metabolic acidosis and marked lactate elevation could not be attributed to shock or hypoxemic respiratory failure and instead favored regional muscle ischemia with rhabdomyolysis as the source of lactate production [12]. Additionally, the electrolyte derangements and elevated transaminases are characteristic of myocyte lysis, and the aspartate aminotransferase (AST)-predominant pattern is likely muscle-derived rather than hepatic in this setting [10,11,13]. The patient's abdominal pain improved with the management of rhabdomyolysis and compartment syndrome, so this decreased concern for additional abdominal pathology. Vascular and cross-sectional imaging demonstrated bilateral changes consistent with edema and proximal right arterial occlusion that likely occurred as a secondary consequence of muscle necrosis. Elevated intracompartmental pressure compromises tissue perfusion and can obliterate arterial flow without a thromboembolic source [14]. Negative bilateral venous duplex ultrasounds excluded extensive DVT, and radiographs excluded occult fracture [14]. Taken together, these findings in combination with the patient's pain out of proportion and tense compartments narrowed a broad differential to rhabdomyolysis with bilateral compartment syndrome and reframed the arterial and hematologic abnormalities as likely downstream consequences rather than primary causes. Although tense compartments are a common finding in compartment syndrome, their absence is not a reliable method of ruling out this diagnosis [5].

The length of the 26-day hospital course was attributable primarily to renal failure and to the wound burden of multiple fasciotomies. Despite early aggressive intervention, the patient progressed to oliguric AKI, ultimately requiring intermittent hemodialysis, but he did experience renal recovery approximately one month after initial presentation. This complication is not unexpected at this level of severity [15]. Wound management required staged fasciotomy closures over three weeks, and the persistent swelling illustrates the prolonged edema that follows ischemia. Surgical outcomes of compartment syndrome are associated with high morbidity and frequent reoperation [9]. A recurrent diagnostic challenge involved distinguishing the reactive neutrophilic leukocytosis and fever of large open wounds from true infection. Negative blood cultures, negative MRSA screening, and no operative evidence of necrotizing fasciitis led to the ultimate discontinuation of antibiotics once all sites were closed and fever resolved. New-onset mild LV systolic dysfunction with severely reduced RV function (EF 41-45%) and intermittent tachycardia added further complications. Its relationship to the marked CK-MB elevation versus a stress- or volume-mediated cardiomyopathy remains unresolved, but repeat echocardiography in an outpatient setting has been recommended.

This case also illustrates how language access impacts diagnostic certainty. The patient's preferred language was Chuj, a Guatemalan dialect that shares some similarities with Spanish but also differs greatly. Access to professional interpreters demonstrably improves outcomes, supports equitable care, and reduces error [16]. Here, Chuj interpretation was intermittently unavailable, and communication frequently proceeded in Spanish, the patient's second language. While this was an appropriate and consented accommodation, history-taking conducted in a non-primary language carries a risk of incomplete disclosure. This is particularly impactful for this patient's history, which required specific detail about the quantity and timing of alcohol intake, unwitnessed loss of consciousness, sleep position, and substance use. The urine drug screen was positive only for substances administered prior to collection, and the serum ethanol level was 76 mg/dL. The patient repeatedly denied any ingestion other than a few alcoholic beverages. Combined sedative and alcohol exposure is the classic setting for prolonged obtundation that permits sustained compression of dependent limbs [9,14]. The case should be read as one in which polysubstance exposure is unlikely but cannot be entirely excluded.

We consider unwitnessed positional rhabdomyolysis in the setting of ethanol exposure as a possible underlying explanation, but this could not be confirmed through the patient's history. This hypothesis accounts for the onset recognized on awakening, bilateral lower extremity involvement with right-sided predominance suggesting a dependent sleeping position, and the toxicologic findings. Alcohol's direct myotoxicity would act synergistically with prolonged compression to cause ischemic necrosis [10]. Another theory that merits consideration includes an unrecognized genetic condition that predisposes patients to rhabdomyolysis [8,17,18]. These conditions involve disorders of glycogen and lipid metabolism, mitochondrial myopathies, muscular dystrophies, and disorders of excitation-contraction coupling such as RYR1-related disease [8,19]. Kruijt et al. introduced the RHABDO acronym as a clinical tool to distinguish patients likely harboring genetic susceptibility from those with acquired triggers [20]. Historical and clinical evidence that suggests genetic susceptibility is highlighted in this acronym and includes recurrent episodes,

high CK levels exceeding 50 times the upper limit of normal, accustomed exercise intensity that cannot explain severe breakdown, blood CK levels persisting for greater than eight weeks, drugs or medications that are absent, and other family history [20]. This patient lacked prior episodes of rhabdomyolysis, possessed a CK level greater than 50 times the upper limit of normal, had no significant increases in exercise activity, had normalization of CK levels within eight weeks, denied drug or medication use, and denied similar family history. In this patient, the absence of prior episodes, family history, or exertional triggers does not exclude a genetic etiology, as many causative variants present with a first episode in adulthood. Further investigation with genetic testing would likely prove beneficial for this patient.

Conclusions

Atraumatic rhabdomyolysis complicated by compartment syndrome without a definitively confirmed cause represents a rare but clinically significant presentation. The co-occurrence of these two conditions in the absence of trauma underscores the importance of maintaining a high suspicion for compartment syndrome in any patient with severe rhabdomyolysis, regardless of the mechanism. Early recognition and aggressive management of both rhabdomyolysis and compartment syndrome remain critical to preventing irreversible complications, including AKI, limb loss, and death. When no acquired etiology is identified, a systematic evaluation for inherited myopathy is warranted, as many previously idiopathic cases are now being attributed to specific genetic defects. Outpatient priorities for this patient include repeat echocardiography, serial assessment for late renal recovery, and referral for neuromuscular genetic evaluation, with all such discussions conducted through professional interpretation in the patient's preferred language.

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: Emma Whitson, Sean Trostel

Acquisition, analysis, or interpretation of data: Emma Whitson, Abby Whitt, Kathryn West, Sean Trostel

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