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Severe Rhabdomyolysis in a Patient With Untreated Hypothyroidism Receiving Statin Therapy: A Case Report

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Abstract

Rhabdomyolysis is a potentially life-threatening condition characterized by skeletal muscle breakdown and may lead to acute kidney injury (AKI) and multiorgan dysfunction. We report the case of a 61-year-old woman with depressive disorder and dyslipidemia who was prescribed rosuvastatin and ezetimibe five months before admission. She presented to the emergency department with progressive myalgia and muscle weakness, ultimately resulting in an inability to walk.

Laboratory evaluation revealed severe rhabdomyolysis, AKI, hyponatremia, hypocalcemia, and markedly elevated aminotransferases. Further etiologic investigation identified severe hypothyroidism secondary to autoimmune thyroiditis. Rosuvastatin and ezetimibe were discontinued, and levothyroxine replacement therapy was initiated. Because of hypervolemia and metabolic acidemia, continuous kidney replacement therapy (CKRT) was required. Given the markedly elevated myoglobin levels, hemoadsorption with an HA380 cartridge was also performed. Anti-3-hydroxy-3-methylglutaryl-coenzyme A reductase antibodies were negative.

Renal function progressively recovered, with complete resolution of rhabdomyolysis. This case underscores severe untreated hypothyroidism as an important predisposing factor for statin-associated muscle toxicity and supports consideration of thyroid function assessment in patients with suggestive symptoms or additional risk factors before or during statin therapy. Hemoadsorption may represent an adjunctive therapeutic option in selected patients with markedly elevated myoglobin levels; however, its contribution to renal recovery cannot be determined from a single case report.

Categories: Emergency Medicine, Internal Medicine, Endocrinology/Diabetes/Metabolism
Keywords: acute kidney injury, hemoadsorption, hypothyroidism, rhabdomyolysis, statins

Introduction

Rhabdomyolysis results from the breakdown of skeletal muscle fibers. Crush injuries and prolonged immobilization are the most common causes. Muscle cell lysis releases intracellular ions, myoglobin, and creatine kinase (CK) into the bloodstream, leading to electrolyte disturbances, acidemia, and an increased risk of acute kidney injury (AKI). Rhabdomyolysis may be asymptomatic, present with nonspecific symptoms such as muscle weakness and myalgia, or progress to organ dysfunction [1-3].

Among medical causes, drug-induced rhabdomyolysis is particularly important. Statins are the medications most frequently implicated and are associated with a spectrum of muscle toxicity ranging from myalgia to, in the most severe cases, rhabdomyolysis [4-6]. Several factors, including drug-drug interactions, influence the risk of statin-induced muscle toxicity. Although uncommon, increased statin toxicity has been reported in patients with hypothyroidism. Because hypothyroidism may remain unrecognized, thyroid dysfunction should be considered before statin initiation, particularly in patients with suggestive symptoms or additional risk factors for muscle toxicity. Autoimmune thyroiditis is a common cause of hypothyroidism and may substantially increase the risk of statin-related complications [6,7]. Hypothyroidism-associated rhabdomyolysis is a rare clinical entity [7,8].

Case Presentation

A 61-year-old woman with a history of depressive disorder receiving long-term pharmacologic treatment with venlafaxine 150 mg and alprazolam 1 mg was evaluated in primary care five months before admission because of nonspecific pain. Laboratory testing revealed mixed dyslipidemia (total cholesterol, 552 mg/dL (reference: <200 mg/dL); high-density lipoprotein cholesterol, 87 mg/dL (reference: 30-60 mg/dL); and triglycerides, 442 mg/dL (reference: <150 mg/dL)) and an elevated CK level of 1394 U/L. Rosuvastatin 40 mg and ezetimibe 10 mg daily were initiated, and she was advised to increase her oral fluid intake; no further diagnostic evaluation was performed.

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Five months later, she presented to the emergency department with a four-month history of generalized muscle weakness and myalgia that had markedly worsened over the preceding three days, leaving her unable to stand or walk independently. On admission, she was drowsy but easily arousable and oriented, with slowed speech, dysphonia, and symmetric, predominantly proximal muscle weakness, graded 3/5 on the Medical Research Council (MRC) scale. There were no fasciculations, diplopia, or dysphagia.

On physical examination, vital signs were within normal limits. The patient was in sinus rhythm, afebrile, and breathing comfortably on room air. The extremities were cold, with prolonged capillary refill time, and generalized edema was present. The thyroid was nontender, with no palpable nodules. A urinary catheter was inserted and drained tea-colored urine. Arterial blood gas analysis showed compensated metabolic acidosis without an elevated lactate level.

Laboratory testing showed stage 3 AKI according to Kidney Disease: Improving Global Outcomes (KDIGO) criteria [9], hyponatremia, severe hypocalcemia, and severe rhabdomyolysis, with a CK level of 17,440 U/L. Transaminase levels were markedly elevated: aspartate aminotransferase (AST) was 136 times the upper limit of normal (ULN), and alanine aminotransferase (ALT) was 63 times the ULN; total bilirubin was 0.6 mg/dL (reference: 0.3-1.2 mg/dL), albumin was 3.1 g/dL (reference: 3.4-4.8 g/dL), and INR was 1.94. Leukocytosis, neutrophilia, and a C-reactive protein (CRP) level of 3.13 mg/dL were also present (Table *1*). Urinalysis revealed proteinuria, hematuria, and glycosuria.

Laboratory parameter	Admission value	Highest documented value (hospital day)	Reference range	Unit
Creatinine	6	6.1 (day 4)	0.66–1.09	mg/dL
Urea	170	182 (day 4)	17–43	mg/dL
Sodium	128	N/A	134–145	mEq/L
Potassium	3.7	N/A	3.5–5.1	mEq/L
Ionized calcium	0.74	N/A	Not available	mmol/L
Creatine kinase (CK)	17440	41579 (day 4)	<145	U/L
Creatine kinase-myocardial band (CK-MB)	2772	Admission value	<24	U/L
Lactate dehydrogenase (LDH)	7313	Admission value	<248	U/L
Myoglobin	>1200	13060 (day 8)	<106.0	ng/mL
Aspartate aminotransferase (AST)	4221	Admission value	<31	U/L
Alanine aminotransferase (ALT)	2140	Admission value	<34	U/L
Gamma-glutamyl transferase (GGT)	383	Admission value	<38	U/L
Alkaline phosphatase (ALP)	349	Admission value	30–120	U/L
Total bilirubin	0.6	N/A	0.3–1.2	mg/dL
Albumin	3.1	N/A	3.4–4.8	g/dL
White blood cell count	21	25 (day 8)	4.4–11.3	$\times 10^9$/L
Neutrophils	89.9	92 (day 8)	50–70	%
Absolute neutrophil count	19.1	23 (day 8)	Not available	$\times 10^9$/L
C-reactive protein (CRP)	3.13	10.49 (day 9)	<0.1	mg/dL
Erythrocyte sedimentation rate (ESR)	54	81 (day 9)	4.0–11.0	mm
Prothrombin time	22.7	Admission value	9.4–12.5	seconds
International normalized ratio (INR)	1.94	N/A	Not available	-
Proteinuria	N/A	3.5 (day 2)	<0.15	g/24 hours

TABLE 1: Trends in key laboratory parameters

Myoglobin was initially reported as >1,200 ng/mL, the upper reporting limit of the assay. The first fully quantified value was 13,060 ng/mL on hospital day 8, before the second HA380 session.

N/A: not available

The patient was admitted to the intensive care unit (ICU). The main differential diagnoses included drug-induced, metabolic, autoimmune, and paraneoplastic causes of rhabdomyolysis.

Rosuvastatin and ezetimibe were discontinued on admission. A review of her medical history revealed recent weight gain. This finding, together with muscle weakness, generalized edema, and psychomotor slowing, raised suspicion for hypothyroidism with myxedematous features. Thyroid function testing showed a thyroid-stimulating hormone (TSH) level of 197.74 mU/L, free thyroxine (fT4) below 0.4 ng/dL, free triiodothyronine (fT3) below 1.5 pg/mL, and positive anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-Tg) antibodies. Anti-3-hydroxy-3-methylglutaryl-coenzyme A reductase (anti-HMGCR) antibodies were negative. Oral levothyroxine was initiated on hospital day 1 at 100 µg/day (approximately 1.6 µg/kg/day) and increased to 350 µg/day on hospital day 5 due to persistently low fT4 levels. Thyroid ultrasonography showed a pseudonodular pattern suggestive of autoimmune thyroiditis.

Initial management in the emergency department included intravenous fluid administration, with close

monitoring of urine output and volume status. Furosemide was subsequently administered. Nephrotic-range proteinuria was documented. Morning cortisol and adrenocorticotropic hormone (ACTH) were reported as within the laboratory reference ranges.

On hospital day 6, continuous kidney replacement therapy (CKRT) was initiated because of persistent oligoanuric AKI and volume overload complicated by acute hypoxemic respiratory failure requiring supplemental oxygen. CK and myoglobin levels remained markedly elevated.

AKI was clinically attributed predominantly to suspected myoglobin-associated acute tubular injury, with a possible contribution from venous congestion, as suggested by Venous Excess Ultrasound (VExUS) grade 3 assessment. Given the markedly elevated myoglobin levels, two HA380 hemoadsorption sessions, each lasting three hours, were performed on hospital days 7 and 8 during ongoing CKRT. Initial myoglobin measurements exceeded the laboratory assay's upper reporting limit of 1,200 ng/mL. The first quantitative measurement was 13,060 ng/mL on hospital day 8, before the second hemoadsorption session, followed by 11,363 ng/mL on day 9 and 7,100 ng/mL on day 11.

Despite oral dose escalation, fT4 remained low at 0.4 ng/dL on hospital day 8. Given concerns regarding impaired gastrointestinal absorption in the setting of anasarca, levothyroxine was switched to intravenous administration at 150 µg/day on hospital day 9, without a loading dose, and maintained for four days.

During the subsequent clinical course, the patient showed progressive improvement, with recovery of urine output, resolution of proteinuria, and complete resolution of rhabdomyolysis. CKRT was discontinued on hospital day 12. Serum creatinine was 1.9 mg/dL the following day, with no subsequent increase. Thyroid hormone replacement was continued, and two hemoadsorption sessions had been performed as part of the supportive treatment strategy. She was transferred to the internal medicine ward on hospital day 15, where the diagnostic workup was completed. By hospital day 14, urinalysis had normalized. Thyroid function subsequently improved, with fT4 reaching 0.7 ng/dL and TSH decreasing to 40 mIU/L by hospital day 18. Myositis-specific and myositis-associated antibody testing was negative, electromyography demonstrated myopathic features, and muscle biopsy findings were consistent with necrotizing myopathy (Table *2*).

Parameter	Result	Reference range
Free thyroxine (fT4)	<0.4 ng/dL	0.70–1.48
Free triiodothyronine (fT3)	<1.5 pg/mL	1.71–3.71
Thyroid-stimulating hormone (TSH)	197.74 mU/L	0.35–4.94
Anti-thyroid peroxidase (anti-TPO) antibodies	Positive	—
Anti-thyroglobulin antibodies	Positive	—
Anti-3-hydroxy-3-methylglutaryl-coenzyme A reductase antibodies (anti-HMGCR) antibodies	Negative	—
Anti-signal recognition particle (anti-SRP) antibodies	Negative	—
Antinuclear antibodies (ANA)	Negative	—
Antineutrophil cytoplasmic antibodies (ANCA; Myeloperoxidase (MPO), Proteinase 3 (PR3))	Negative	—
Rheumatoid factor	Negative	—
Anti-double-stranded DNA (anti-dsDNA) antibodies	Negative	—
Lupus anticoagulant	Negative	—
Anti-glutamic acid decarboxylase (anti-GAD) antibodies	Negative	—
Myositis-specific and myositis-associated antibodies	Negative	—
Anti-SSA/Ro and anti-SSB/La antibodies	Negative	—

Anti-Jo-1 antibodies	Negative	—
Anti-PL-7 antibodies	Negative	—
Anti-PL-12 antibodies	Negative	—
Anti-glomerular basement membrane (anti-GBM) antibodies	Negative	—
Dried blood spot test (Pompe disease)	Negative	—
Serum protein electrophoresis	No abnormalities detected	—
Complement C3 and C4	No complement consumption	—
Morning cortisol	Normal	—
Adrenocorticotropic hormone (ACTH)	Normal	—
Angiotensin-converting enzyme (ACE)	Normal	—
Aldolase	Within reference range	—
Hepatitis C virus (HCV)	Negative	—
Hepatitis B virus (HBV)	Negative	—
Human immunodeficiency virus (HIV-1/2)	Negative	—
Epstein-Barr virus (IgG and IgM)	Negative	—
Cytomegalovirus (IgG and IgM)	Negative	—
Mycoplasma pneumoniae antibodies	Negative	—
Borrelia antibodies	Negative	—
Urine culture (on admission)	Negative	—
Blood cultures (on admission)	Negative	—
Electromyography (EMG)	Myopathic features with marked active denervation	—
Muscle biopsy	Moderate changes with a necrotizing pattern; no sarcolemmal complement staining. Mitochondrial studies were normal, with no evidence of glycogen or lipid accumulation.	—
Computed tomography (CT) of the chest, abdomen, and pelvis	No abnormalities detected	—
Positron emission tomography–computed tomography (PET-CT)	No suspicious lesions	—
Mammography	No abnormalities detected	—
Upper gastrointestinal endoscopy and colonoscopy	No abnormalities detected	—

TABLE 2: Diagnostic workup and etiologic investigation

The hospital course was complicated by an *Escherichia coli* urinary tract infection, treated with ceftriaxone, and a subsequent urinary tract infection caused by extended-spectrum beta-lactamase-producing *Klebsiella pneumoniae*, for which meropenem was initiated. The patient also developed a non-ST-elevation myocardial infarction in the setting of anemia (hemoglobin approximately 7 g/dL). Coronary angiography revealed severe coronary artery disease involving the left main and left anterior descending arteries, subsequently treated with percutaneous coronary intervention and implantation of two drug-eluting stents. She was discharged on hospital day 41 with substantial overall clinical improvement.

She remains under outpatient follow-up. The final diagnosis was severe rhabdomyolysis associated with untreated hypothyroidism, likely exacerbated by statin therapy, with muscle biopsy showing a necrotizing myopathy pattern. She continues to receive levothyroxine replacement therapy and was prescribed a

proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitor for lipid management.

Discussion

Hypothyroidism-associated rhabdomyolysis is rare but potentially life-threatening and may progress to multiorgan dysfunction, particularly AKI secondary to myoglobin release and intratubular cast formation. Its precise incidence is not well established, with published evidence consisting largely of case reports and small case series [1-3,10].

This case highlights the importance of considering thyroid dysfunction in the differential diagnosis of rhabdomyolysis, especially when additional precipitating factors, such as statin therapy, are present. Severe hypothyroidism, which is frequently underdiagnosed, may predispose patients to muscle toxicity through multiple metabolic and structural mechanisms, thereby increasing the risk of rhabdomyolysis [4-8].

In this patient, CK was already elevated before statin initiation, indicating pre-existing muscle injury that may have been related to unrecognized hypothyroidism. Thyroid function results from that earlier assessment were unavailable, preventing confirmation of this association. Subsequent statin exposure may have exacerbated the underlying muscle injury.

The necrotizing biopsy pattern, negative myositis antibody panel, and recovery without immunosuppressive treatment favored a toxic/metabolic process.

Early recognition of this association is essential because discontinuation of the offending drug and prompt correction of thyroid hormone deficiency are the cornerstones of treatment [6-8]. However, in patients with severe rhabdomyolysis complicated by AKI, conventional supportive measures may be insufficient. In this setting, extracorporeal therapies, including CKRT and hemoadsorption, have been used to facilitate the removal of myoglobin and other middle-molecular-weight molecules. However, evidence regarding their impact on clinically relevant outcomes remains limited [10,11].

In this case, thyroid hormone replacement, CKRT, and hemoadsorption were used as part of the therapeutic approach. The patient subsequently showed resolution of rhabdomyolysis and recovery of renal function. However, given the observational nature of a single case, no causal relationship can be established between hemoadsorption and the favorable clinical outcome, and its individual contribution cannot be distinguished from that of thyroid hormone replacement and kidney replacement therapy. Furthermore, the absence of paired pre- and post-hemoadsorption myoglobin measurements prevented direct assessment of myoglobin removal attributable to the HA380 cartridge.

Conclusions

Severe hypothyroidism should be considered in patients with unexplained rhabdomyolysis and in patients who develop muscle symptoms during statin therapy. Prompt withdrawal of potentially contributing medications and thyroid hormone replacement are central to management. In severe cases complicated by AKI, CKRT may be required according to conventional indications. Hemoadsorption may represent an adjunctive strategy in selected patients with markedly elevated myoglobin levels, although its independent clinical benefit requires further investigation.

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.

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