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Severe Rosuvastatin-Associated Rhabdomyolysis and Dialysis-Requiring Acute Kidney Injury in an Octogenarian Patient: A Case Report

Authors' Contribution:

Study Design A
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Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Female, 87-year-old

Final Diagnosis: Acute kidney injury • rhabdomyolysis • rosuvastatin associated rhabdomyolysis

Symptoms: Fatigue • muscle weakness • oliguria • poor appetite

Clinical Procedure: —

Specialty: Nephrology

Objective: Unusual clinical course

Background: Statins are fundamental to dyslipidemia management, but high-potency rosuvastatin can cause myotoxicity in vulnerable older patients. We present a probable case of rosuvastatin-associated rhabdomyolysis causing stage 3 acute kidney injury requiring hemodialysis in an 87-year-old woman with polypharmacy, highlighting clinical management and outcomes.

Case Report: An octogenarian presented with progressive muscle weakness and inability to ambulate 20 days after starting rosuvastatin (20 mg/day), ezetimibe (10 mg/day), and evogliptin (5 mg/day). Laboratory tests revealed creatine kinase above 4267 U/L, serum creatinine 6.13 mg/dL, blood urea 220 mg/dL, severe hyponatremia (122 mEq/L), bicarbonate 15 mEq/L, and elevated transaminases (AST 419 U/L; ALT 342 U/L). Direct bilirubin (0.2 mg/dL) and gamma-glutamyl transferase (28 U/L) levels were normal, excluding primary hepatocellular or cholestatic liver injury. Urinalysis findings supported myoglobinuria. Refractory oliguria despite aggressive intravenous fluid resuscitation required emergency hemodialysis with sodium correction to prevent osmotic demyelination; rhabdomyolysis prompted rosuvastatin cessation. Concomitant nephrotoxic and renally cleared medications were temporarily withheld. During a 30-day hospital stay with 8 hemodialysis sessions, dialytic support was discontinued on Day 23 (serum creatinine 1.61 mg/dL). After physical rehabilitation, the patient was discharged on Day 30 with full motor strength (MRC grade 5/5). At 3 months, serum creatinine had stabilized (1.40 mg/dL; eGFR 38 mL/min/1.73 m²; creatine kinase 62 U/L) and independent ambulation continued.

Conclusions: Statin-associated rhabdomyolysis in older adults can be followed by renal recovery with dialysis independence and functional recovery through early drug cessation, fluid resuscitation, and timely dialytic support. Formal Naranjo assessment (score 5) classified the reaction as “probable,” highlighting the multifactorial risks of geriatric polypharmacy.

Keywords: Drug Combinations • Kidney Tubular Necrosis, Acute • Rhabdomyolysis • Rosuvastatin Calcium

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Introduction

Statins act as competitive inhibitors of 3-hydroxy-3-methylglutaryl-coenzyme A reductase, preventing the conversion of HMG-CoA to mevalonate, the initial and rate-limiting step in the cholesterol biosynthesis pathway [1]. This inhibition reduces hepatic cholesterol levels, promoting the upregulation of low-density lipoprotein (LDL) receptors on the cell surface and thereby increasing the uptake and clearance of circulating LDL [1,2]. Clinically, statins are classified based on their potency in reducing LDL cholesterol. High-potency statins, such as rosuvastatin (20-40 mg/day) and atorvastatin (40-80 mg/day), are indicated to reduce LDL cholesterol by 50% or more [3,4]. The use of this drug class is a cornerstone of both primary and secondary prevention of cardiovascular disease, reducing morbidity and mortality and providing pleiotropic effects, such as reduced endothelial inflammation [3,5].

Although generally considered safe, statin use is not without risks, especially when high-potency agents are prescribed to older patients with risk factors for adverse effects, such as polypharmacy. Statin-associated muscle symptoms encompass a spectrum ranging from mild myalgia (muscle pain/weakness without creatine kinase [CK] elevation; incidence of 5% to 10% in observational cohorts) to myositis (muscle symptoms with elevated CK), myopathy, and severe rhabdomyolysis (massive CK elevation > 10 times the upper limit of normal with renal injury risk, occurring in < 0.01% of patients) [5,6]. Older patients with multiple comorbidities and polypharmacy experience increased vulnerability because physiological changes and potential drug-drug interactions can alter statin pharmacokinetics [7,8]. Primary prevention guidelines emphasize individualized risk-benefit assessment in octogenarians [3,4], but severe adverse outcomes can occur when multiple physiological vulnerabilities overlap.

The primary contribution of this case report is to illustrate that substantial renal recovery, with long-term dialysis independence, alongside complete functional recovery remains achievable even after severe, dialysis-requiring statin-associated rhabdomyolysis in an octogenarian with polypharmacy, provided that early clinical recognition, immediate drug discontinuation, and prompt dialytic support are implemented within a structured clinical framework.

Case Report

Clinical Presentation and History

An 87-year-old woman with a medical history of systemic arterial hypertension, type 2 diabetes mellitus, atrial fibrillation, diabetic retinopathy, and glaucoma presented to the

emergency department. She reported a 2-week history of progressive muscle weakness in the lower limbs, with acute worsening over the previous 3 days, which resulted in an inability to stand. The condition was associated with fatigue, poor appetite, and oliguria.

Approximately 20 days before admission, the patient had begun a combination lipid-lowering regimen consisting of rosuvastatin (20 mg/day) and ezetimibe (10 mg/day) to manage persistent dyslipidemia in the context of high cardiovascular risk. Evogliptin (5 mg/day) was also initiated around the same time. One month before this regimen began, baseline outpatient laboratory evaluation showed stable renal function (serum creatinine 1.0 mg/dL; estimated glomerular filtration rate [eGFR] 62 mL/min/1.73 m²), normal hepatic transaminase levels (aspartate aminotransferase [AST] 22 U/L; alanine aminotransferase [ALT] 19 U/L), and CK within the normal range (85 U/L). The patient's chronic home medications consisted of losartan (50 mg/day), dapagliflozin (10 mg/day), gliclazide (30 mg/day), metformin (850 mg/day), quetiapine (25 mg/day), indapamide (2.5 mg/day), metoprolol succinate (50 mg/day), and dabigatran (110 mg twice daily).

Physical Examination

On admission, the patient was clinically dehydrated, with dry mucous membranes and reduced skin turgor. She had an irregular heart rhythm but remained hemodynamically stable (blood pressure 125/75 mm Hg, heart rate 82 bpm, respiratory rate 18 breaths/min, and room-air SpO₂ 96%). Neurological examination revealed preserved sensation and deep tendon reflexes, but motor strength was substantially and symmetrically reduced in both lower limbs (grade 3/5 on the Medical Research Council [MRC] scale); diffuse muscle tenderness was noted on palpation.

Laboratory Investigation

Initial evaluation confirmed severe acute kidney injury (Kidney Disease: Improving Global Outcomes [KDIGO] stage 3) and severe metabolic derangements, with a serum creatinine level of 6.13 mg/dL, blood urea of 220 mg/dL (blood urea nitrogen 102.8 mg/dL), and eGFR of 6 mL/min/1.73 m². Muscle and biochemical marker levels were greatly elevated: CK was above the maximum analytical reporting limit of the assay (> 4267 U/L), AST was 419 U/L, ALT was 342 U/L, and lactate dehydrogenase was 660 U/L. Arterial blood gas and electrolyte testing demonstrated metabolic acidosis (pH 7.30, serum bicarbonate 15 mEq/L), severe hypotonic hyponatremia (sodium 122 mEq/L), serum potassium of 5.1 mEq/L, and serum albumin of 2.8 g/dL. Direct bilirubin (0.2 mg/dL) and gamma-glutamyl transferase (28 U/L) levels were normal.

Table 1. Serial evolution of clinical laboratory parameters from pretreatment baseline through acute hospitalization and 3-month outpatient follow-up.

Parameter	Baseline (outpatient/ 1 mo prior)	Day 1 (admission/ HD 1)	Day 3 (HD 2)	Day 7 (HD 3)	Day 14 (HD 5)	Day 23/ dialysis cessation (HD 8)*	Day 30/ discharge	1-Month follow-up	3-Month follow-up	Reference range
Creatine kinase (CK, U/L)	85	> 4267**	4267	1850	420	47	45	55	62	26-192
Serum creatinine (mg/dL)	1.00	6.13	4.80	3.20	2.10	1.61	1.55	1.48	1.40	0.50-1.10
eGFR (mL/ min/1.73 m ²)	62	6	8	13	22	31	33	35	38	> 60
Blood urea (mg/dL)	38	220	168	112	68	44	42	40	38	15-45
Serum sodium (mEq/L)	138	122	128	134	137	139	139	138	140	135-145
Serum potassium (mEq/L)	4.3	5.1	4.6	4.2	4.0	4.3	4.3	4.4	4.5	3.5-5.0
Serum bicarbonate (mEq/L)	24	15	19	22	24	24	24	25	24	22-29
AST (U/L)	22	419	280	110	42	24	23	20	21	10-35
ALT (U/L)	19	342	230	95	38	22	21	18	19	10-35
LDH (U/L)	180	660	510	340	220	175	172	180	170	125-220
Hemodialysis chronology	None	Session 1	After session 2	After session 4	Session 7	Session 8 (final)	Completed (8 total)	None	None	—

* **Note on chronology:** Dialytic support was permanently discontinued on Day 23 after renal functional recovery (serum creatinine 1.61 mg/dL). The patient remained hospitalized for an additional 7 days for physical rehabilitation and motor recovery, achieving independent ambulation at hospital discharge on Day 30. ** **Note on CK assay:** The initial value of > 4267 U/L on Day 1 represents the upper analytical quantification limit of the automated laboratory assay before manual dilution. Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; eGFR, estimated glomerular filtration rate; HD, hemodialysis; LDH, lactate dehydrogenase.

Urinalysis revealed dark, tea-colored urine and 3+ blood/heme on dipstick, but microscopic examination demonstrated only 2 to 4 red blood cells per high-power field, strongly supporting dipstick pseudohematuria secondary to myoglobinuria. Diagnostic evaluation for alternative causes of rhabdomyolysis and acute kidney injury showed negative results: renal ultrasonography ruled out postrenal urinary obstruction or hydronephrosis; viral serologies (human immunodeficiency virus 1/2, hepatitis B/C, and human T-lymphotropic virus 1/2) were nonreactive; the thyroid-stimulating hormone level was normal (1.8 mIU/L); and there was no clinical or collateral history of trauma, prolonged immobilization, strenuous exercise, excessive alcohol consumption, acute systemic infection, or hyperthermia.

Serial laboratory trends from initial evaluation through hospital discharge and the 3-month follow-up are detailed in **Table 1**.

Management and Outcome

Upon clinical recognition of severe rhabdomyolysis and acute kidney injury, the primary suspected agent, rosuvastatin (co-prescribed with ezetimibe), was immediately discontinued. Initial pre-dialysis supportive management consisted of aggressive intravenous fluid resuscitation with isotonic 0.9% saline (total of 2.5 L over the first 24 hours), aimed at expanding intravascular volume and restoring renal perfusion. Sodium bicarbonate was not administered for urinary alkalinization because active volume expansion remained the primary focus of

supportive management. Urine output was strictly monitored through an indwelling Foley catheter and remained refractory at less than 15 mL/hour (< 0.2 mL/kg/h) over 24 hours despite fluid administration.

Emergency renal replacement therapy was indicated because of refractory oliguria unresponsive to volume loading, severe uremic azotemia (blood urea 220 mg/dL), metabolic acidosis (serum bicarbonate 15 mEq/L), and a high risk of hypervolemic complications. Intermittent hemodialysis was initiated through a temporary femoral catheter. Given the baseline hyponatremia (122 mEq/L), dialysate sodium was carefully individualized and gradually titrated upward (targeting a correction rate of < 8 mEq/L per 24 hours) to minimize the risk of osmotic demyelination syndrome.

Management of concomitant medications during acute hospitalization was standardized as follows:

- **Discontinued/withheld:** Losartan, indapamide, dapagliflozin, metformin, and gliclazide were immediately withheld to prevent exacerbation of nephrotoxicity and reduce the risks of lactic acidosis and hypoglycemia in stage 3 acute kidney injury. Dabigatran was temporarily withheld due to the risk of renal accumulation and bleeding during vascular cannulation. Ezetimibe and evogliptin were also withheld.
- **Continued/adjusted:** Metoprolol succinate was continued with regular blood pressure monitoring. Quetiapine was maintained. Short-acting subcutaneous regular insulin was used for glycemic control in place of oral hypoglycemic agents.

During the 30-day hospitalization period, the patient underwent a total of 8 intermittent hemodialysis sessions (on Days 1, 2, 4, 6, 8, 11, 14, and 23) alongside supportive care. CK levels progressively decreased (from > 4267 U/L on Day 1 to 4267 U/L on Day 3, 1850 U/L on Day 7, 420 U/L on Day 14, and 47 U/L on Day 23), and transaminase levels normalized. Renal function gradually recovered, with spontaneous recovery of diuresis, permitting weaning from dialysis and permanent discontinuation after Day 23, when serum creatinine had decreased to 1.61 mg/dL. The patient remained hospitalized for an additional 7 days for physical rehabilitation, achieving full motor strength (MRC grade 5/5) and independent ambulation by discharge (Day 30; serum creatinine 1.55 mg/dL). Outpatient follow-up at 1 and 3 months after discharge confirmed sustained clinical stability; serum creatinine stabilized at 1.40 mg/dL (eGFR 38 mL/min/1.73 m²), serum CK was normal (62 U/L), and myalgia or motor weakness did not recur.

Discussion

This case illustrates a severe presentation of suspected statin-associated rhabdomyolysis resulting in dialysis-requiring acute

kidney injury in an octogenarian patient. Although clinical onset coincided with the initiation of a combination lipid-lowering regimen comprising rosuvastatin and ezetimibe, along with evogliptin, rosuvastatin remains the primary suspected myotoxic agent based on established pharmacological profiles [8,9]; ezetimibe monotherapy rarely induces severe rhabdomyolysis [10].

To systematically assess adverse drug causality, the Naranjo Adverse Drug Reaction Probability Scale was utilized (Table 2) [11]. This assessment addressed specific clinical complexities: previous conclusive reports of rosuvastatin-induced myotoxicity (+1), event onset after drug initiation (+2), clinical improvement following drug withdrawal (+1), and confirmation by objective laboratory data (+1). However, points for the absence of alternative causes (0 points) were not assigned because the patient had several clinically relevant competing contributors, including advanced age, acute dehydration, pre-existing diabetic retinopathy, concomitant renin-angiotensin-aldosterone system blockade, diuretic therapy, sodium-glucose cotransporter 2 inhibitor exposure, and polypharmacy. Furthermore, because clinical recovery occurred concomitantly with multiple intensive interventions (hemodialysis, volume resuscitation, and withdrawal of multiple medications), recovery cannot be attributed solely to rosuvastatin cessation. Consequently, the formal Naranjo score was 5, classifying the reaction as “probable.” Nevertheless, the close temporal relationship, characteristic clinical phenotype, and massive CK elevation strongly support rosuvastatin as the primary precipitating trigger within a multifactorial context.

A key diagnostic consideration in this case concerns the interpretation of elevated transaminase levels (AST 419 U/L, ALT 342 U/L). Although initially regarded as coexisting statin hepatotoxicity, elevated AST and ALT levels are well-recognized manifestations of severe skeletal muscle breakdown, given that aminotransferases are abundant in skeletal muscle myocytes [12] (Figure 1). In the absence of liver-specific markers, such as elevated direct bilirubin or gamma-glutamyl transferase, these enzyme elevations in our patient likely reflect secondary release from skeletal muscle during severe rhabdomyolysis rather than primary hepatocellular necrosis [12,13].

Regarding the pharmacokinetic and pharmacodynamic mechanisms of rosuvastatin toxicity (Figure 1), unlike lipophilic statins (eg, simvastatin and atorvastatin), which undergo extensive hepatic metabolism via cytochrome P450 CYP3A4, rosuvastatin is a hydrophilic molecule that is minimally metabolized by CYP2C9 and CYP2C19 [1,14]. Rosuvastatin disposition predominantly depends on active hepatic and renal membrane transporters, including organic anion-transporting polypeptides (OATP1B1 and OATP1B3, encoded by *SLCO1B1* and *SLCO1B3*, respectively) and breast cancer resistance protein (encoded by *ABCG2*) [13,14]. In our patient, impaired renal clearance due

Table 2. Item-by-item application of the Naranjo Adverse Drug Reaction Probability Scale for rosuvastatin.

Question/criterion	Yes	No	Do not know	Score assigned	Clinical justification/context
1. Are there previous conclusive reports of this reaction?	+1	0	0	+1	Well-documented drug class effect (statin-associated rhabdomyolysis and myotoxicity)
2. Did the adverse event appear after the suspected drug was administered?	+2	-1	0	+2	Muscle symptoms and AKI developed 20 days after rosuvastatin initiation
3. Did the adverse reaction improve when the drug was discontinued or a specific antagonist was administered?	+1	0	0	+1	CK levels declined and renal function recovered after rosuvastatin withdrawal (alongside hemodialysis)
4. Did the adverse reaction reappear when the drug was readministered?	+2	-1	0	0	Rechallenge was not performed because of the life-threatening clinical risk and was deemed unethical
5. Are there alternative causes (other than the drug) that could have caused the reaction?	-1	+2	0	0	Multiple competing risk factors were present (advanced age, acute dehydration, RAAS blockade, diuretics, SGLT2 inhibitors, and polypharmacy). Points for the complete absence of alternative causes were not assigned
6. Did the reaction reappear when a placebo was given?	-1	+1	0	0	Placebo testing was not performed
7. Was the drug detected in the blood (or other fluids) at concentrations known to be toxic?	+1	0	0	0	Serum rosuvastatin concentrations were not measured during routine care
8. Was the reaction more severe when the dose was increased, or less severe when the dose was decreased?	+1	0	0	0	Dose titration was not attempted
9. Did the patient have a similar reaction to the same or similar drugs during any previous exposure?	+1	0	0	0	No previous statin exposure or history of myopathy was recorded
10. Was the adverse event confirmed by any objective evidence?	+1	0	0	+1	Objective confirmation included massive CK elevation (> 4267 U/L), dipstick pseudohematuria, and acute kidney injury
Total score	—	—	—	5	Classification: Probable ADR (Score 5 to 8 range)

Note: The total Naranjo score was 5, classifying the adverse drug reaction as probable (score ranges: ≥ 9 , definite; 5-8, probable; 1-4, possible; 0, doubtful). Points for the absence of alternative causes (Question 5) were not assigned (score = 0) because of multiple competing clinical risk factors and comorbidities, including advanced age, acute prerenal dehydration, RAAS blockade, thiazide-like diuretic use, SGLT2 inhibitor use, and polypharmacy. Furthermore, clinical improvement occurred concurrently with multiple therapeutic interventions (intravenous hydration, hemodialysis, and withdrawal of potential nephrotoxins), precluding attribution of recovery solely to rosuvastatin cessation.

Abbreviations: ADR, adverse drug reaction; AKI, acute kidney injury; CK, creatine kinase; RAAS, renin-angiotensin-aldosterone system; SGLT2, sodium-glucose cotransporter 2.

to acute prerenal dehydration, combined with competition at the transporter level in the setting of extensive polypharmacy and advanced age, likely led to systemic drug accumulation and increased myotoxicity [15,16].

In the context of the existing literature, this report provides an incremental contribution to the case literature. Mohamed et al reported a fatal outcome in a 67-year-old man who presented with statin-induced rhabdomyolysis and multiorgan failure [17]. Our

case demonstrates that even in an older patient (87 years) with extensive polypharmacy and stage 3 dialysis-requiring acute kidney injury, substantial renal recovery with dialysis independence and complete functional recovery is achievable through prompt drug discontinuation, supportive hydration, and timely hemodialysis. Although renal function did not entirely return to baseline (serum creatinine stabilized at 1.40 mg/dL, whereas baseline was 1.00 mg/dL), the outcome represents a favorable clinical trajectory that avoided permanent renal replacement therapy.

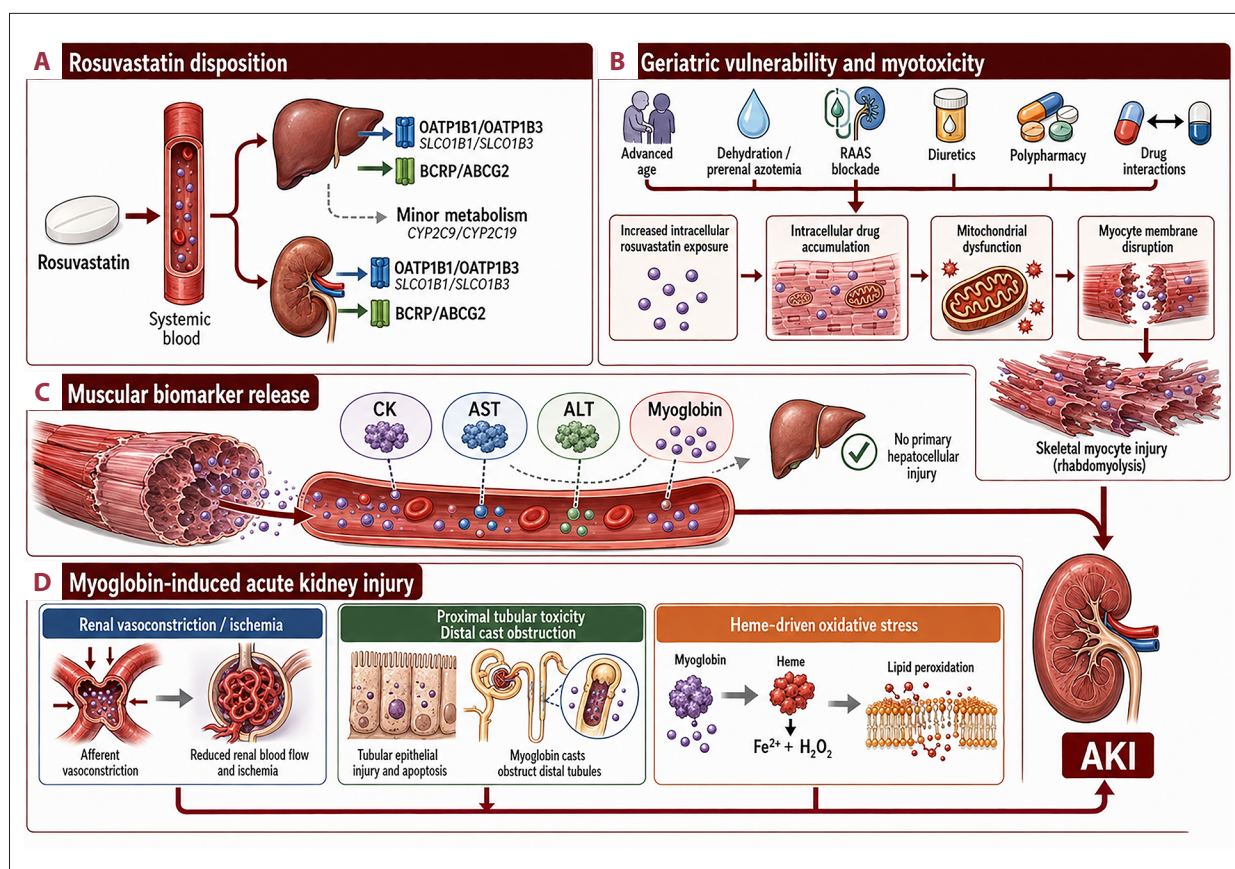


Figure 1. Integrated pathophysiological mechanisms of rosuvastatin disposition, rhabdomyolysis, muscular transaminase release, and myoglobin-induced acute kidney injury. Schematic representation illustrating: **(A)** pharmacokinetic determinants of rosuvastatin disposition, highlighting its primary reliance on hepatic and renal uptake and efflux transporters—organic anion-transporting polypeptides OATP1B1/OATP1B3, encoded by *SLCO1B1/SLCO1B3*, and breast cancer resistance protein (BCRP), encoded by *ABCG2*—alongside minor metabolism mediated by CYP2C9 and CYP2C19; **(B)** the interplay of geriatric vulnerability factors—including advanced age, acute dehydration and prerenal azotemia, renin-angiotensin-aldosterone system (RAAS) blockade, diuretic use, polypharmacy, and potential drug-drug interactions—that promotes increased intracellular rosuvastatin exposure, mitochondrial dysfunction, skeletal myocyte membrane disruption, and rhabdomyolysis; **(C)** the non-hepatic origin of aminotransferase elevations, demonstrating the release of creatine kinase (CK), aspartate aminotransferase (AST), alanine aminotransferase (ALT), and myoglobin from damaged skeletal myocytes into the systemic circulation without primary hepatocellular injury; and **(D)** the 3 mechanisms underlying myoglobin-induced acute kidney injury (AKI): renal vasoconstriction and ischemia, direct proximal tubular epithelial toxicity with distal myoglobin cast obstruction, and heme-driven oxidative stress and lipid peroxidation.

Conclusions

This report demonstrates that severe, dialysis-requiring statin-associated rhabdomyolysis can occur in vulnerable older patients with polypharmacy; early diagnosis, immediate drug cessation, and structured dialytic support can lead to substantial renal recovery, with sustained dialysis independence, alongside complete functional recovery. Because this observation is derived from a single patient with multiple baseline vulnerabilities and overlapping risk factors, causal attribution remains probable rather than definitively established. Clinical decisions regarding dyslipidemia management in older patients should emphasize careful individualization, dose

titration, routine medication review, and prompt investigation of acute muscle weakness.

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Institution Where Work Was Done

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Informed Consent

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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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