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CASE REPORT

The Havoc of Statins: A Case of Statin-Induced Rhabdomyolysis Following Rescue PCI to the Right Coronary Artery.

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Abstract

Background: Rhabdomyolysis is a potentially life-threatening condition characterized by skeletal muscle injury leading to the release of intracellular components such as creatine kinase (CK), myoglobin, and electrolytes into the systemic circulation. Statins remain one of the most common pharmacologic causes, particularly when precipitating factors such as high-dose therapy, sepsis, dehydration, or shock coexist. Early recognition and prompt intervention are crucial to avoid complications such as acute kidney injury (AKI), metabolic derangements, and arrhythmias.

Case Presentation: A 56-year-old woman with poorly controlled diabetes and hypertension presented with acute inferior wall myocardial infarction (IWMI). She received fibrinolysis followed by rescue percutaneous coronary intervention (PCI) to the right coronary artery (RCA). Post-procedure, she developed atrial fibrillation, hemodynamic instability, and sepsis, for which she received high-dose statin therapy as part of post-MI management. Within 48 hours, she developed dark urine, rising creatinine, markedly elevated CK levels (6,458 U/L), and declining urine output. Laboratory evaluation confirmed rhabdomyolysis, likely precipitated by statins in the context of sepsis and shock. Statins were discontinued, aggressive IV hydration was initiated, and antibiotics were started for hospital-acquired infection.

Results: Using the McMahon Score, the patient had a high-risk score of 7, indicating a significant likelihood of requiring dialysis. With early recognition, meticulous fluid resuscitation, hemodynamic support, and infection control, her renal function gradually improved without requiring renal replacement therapy. By discharge, her hemodynamics stabilized, urine output normalized, and creatinine levels began trending downward. At the one-week follow-up, renal function had completely recovered, cardiac rhythm normalized, and she remained clinically stable.

Conclusion: This case highlights the importance of recognizing statin-induced rhabdomyolysis in high-risk cardiovascular patients, particularly when interacting factors such as sepsis, dehydration, and hemodynamic compromise coexist. Careful monitoring of renal function and CK levels, early discontinuation of statins, and prompt supportive therapy remain critical to preventing morbidity and facilitating full recovery.

Keywords

Statins, Rhabdomyolysis, Acute Kidney Injury, Rescue Percutaneous Coronary Intervention, Myocardial Infarction, Sepsis, Mcmahon Score.

Introduction

Rhabdomyolysis is a potentially life-threatening clinical syndrome characterized by the breakdown of skeletal muscle fibers and the subsequent release of intracellular contents such as creatine kinase (CK), myoglobin, and electrolytes into the systemic circulation[1–3]. This biochemical cascade can lead to acute kidney injury (AKI), electrolyte disturbances, coagulopathy, and severe cardiac arrhythmias if not recognized promptly[4,5]. Although trauma, exertional stress, infections, and metabolic disorders are well-recognized causes, drug-induced etiologies particularly statin-associated muscle toxicity represent an important contributor in hospitalized patients[6–9]. Statins may trigger muscle injury through mitochondrial dysfunction, depletion of coenzyme Q10, and disruption of sarcolemmal stability[10,11]. Their risk is amplified in the presence of sepsis, dehydration, hemodynamic instability, renal impairment, or interacting medications[8,12]. This report describes a case of severe statin-induced rhabdomyolysis following rescue percutaneous coronary intervention (PCI) in a high-risk cardiac patient, highlighting the importance of early diagnosis, risk stratification, and aggressive management to prevent irreversible renal injury.

Case Presentation

A 56-year-old woman with poorly controlled diabetes mellitus and hypertension presented with acute retrosternal chest pain radiating to the jaw, accompanied by diaphoresis and shortness of breath. ECG confirmed an inferior wall ST-elevation myocardial infarction (IWMI), and she received fibrinolysis with streptokinase, resulting in partial ST-segment resolution. Ongoing chest discomfort warranted angiographic evaluation, which revealed multivessel coronary artery disease with significant stenosis of the right coronary artery (RCA). Rescue PCI was performed with deployment of two drug-eluting stents. Post-procedure, she developed atrial fibrillation and thromboembolic occlusion of the nodal branch, requiring amiodarone therapy. Over the next 48 hours, she experienced hemodynamic instability, fever, generalized myalgia, and progressively decreasing urine output with tea-colored discoloration. Her past medical history, recent high-dose statin initiation, and evolving clinical picture raised concern for muscle injury and renal compromise.

Diagnostic Assessment

Laboratory evaluation demonstrated markedly elevated CK levels of 6,458 U/L (ULN 168 U/L), serum creatinine of 7.7 mg/dL, and urea of 118 mg/dL, consistent with severe rhabdomyolysis and evolving AKI. Additional findings included leukocytosis, elevated ESR, deranged liver function tests, and features of sepsis. Urine output dropped to 20 mL/hour, and dark-brown urine strongly suggested myoglobinuria. ECG monitoring revealed recurrent atrial fibrillation followed by a transient 2:1 atrioventricular block. Echocardiography showed preserved ejection fraction (55%) with regional wall motion abnormalities of the basal septum and basal inferior wall. Using the McMahon Score, a validated risk prediction tool for dialysis and mortality in rhabdomyolysis, the patient scored 7, placing her in a high-risk category with a significant likelihood of requiring renal replacement therapy[7,13,14]. The combination of high-dose statin exposure, sepsis, hemodynamic compromise, and markedly elevated CK supported a diagnosis of statin-induced rhabdomyolysis precipitated by critical illness.

Therapeutic Intervention

Management focused on rapid reversal of precipitating factors and prevention of further renal injury. Statins and all potentially nephrotoxic medications were immediately discontinued. Aggressive intravenous hydration was initiated and titrated according to central venous pressure and urine output in order to mitigate myoglobin-related tubular injury[10,12]. Broad-spectrum antibiotics were administered for suspected hospital-acquired lower respiratory tract infection. Hemodynamic instability required dual inotropic support, and amiodarone infusion continued for rate and rhythm control. Nephrology and cardiology teams collaborated to closely monitor renal function, electrolytes, CK trends, and urine output. Although her McMahon score suggested

significant risk, early resuscitation and supportive management prevented the need for dialysis.

Follow-up and Outcomes

Over the next several days, the patient demonstrated steady improvement. Her urine output gradually normalized, creatinine decreased to 3.2 mg/dL, and urea trended down to 90 mg/dL by the time of discharge. Myalgia and generalized weakness resolved, and her hemodynamic status stabilized without further rhythm disturbances. One-week follow-up demonstrated complete renal recovery with creatinine at 0.9 mg/dL and urea at 36 mg/dL. Repeat ECG showed normalized PR interval and stable sinus rhythm, while echocardiography confirmed preserved left ventricular systolic function with unchanged regional hypokinesis. She remained clinically stable and was continued on dual antiplatelet therapy and ezetimibe instead of statins.

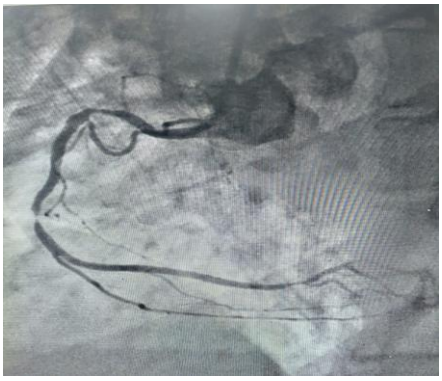

Figure 1: Pre-PCI LAO view of RCA engaged with JR4 catheter.

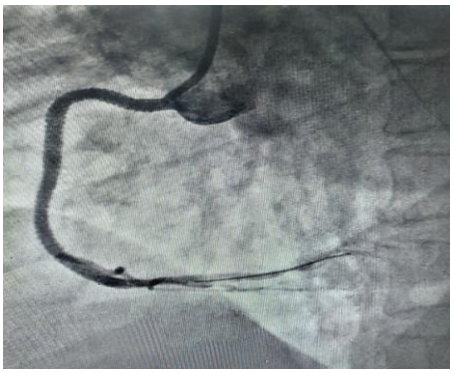

Figure 2: Post-PCI LAO view of RCA engaged with JR4 catheter showing occlusion of AV nodal branch.

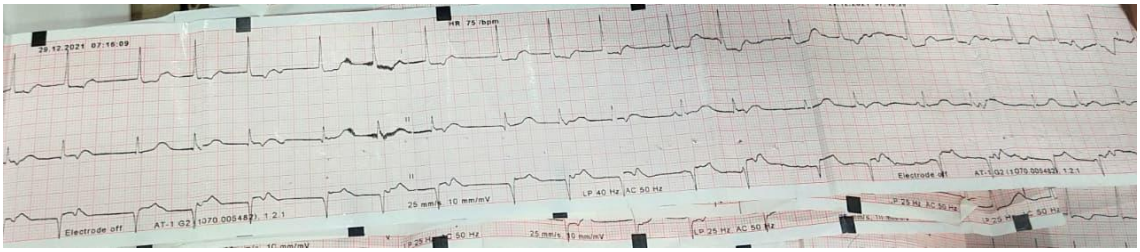

Figure 3: Rhythm strip (showing leads I, II, III) taken after the Injection of Cordarone in the ICU, which showed 2:1 AV block.

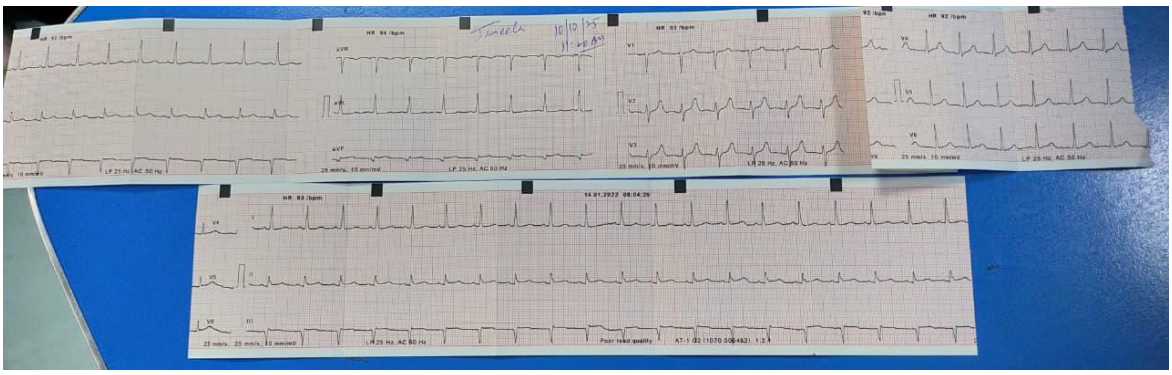

Figure 4: Complete 12 Lead ECG and Rhythm strip (showing leads I, II, III) done on a week follow-up of this patient.

Table 1: McMahon Score – Variables and Points.

Parameter	Criteria	Points
Age	>50 years	2
Sex	Female	1
Initial creatinine (mg/dL)	1.4–2.2	1
	>2.2	3
Initial calcium (mg/dL)	<7.5	2
Initial phosphate (mg/dL)	>4.5	3
Initial bicarbonate (mEq/L)	<19	2
Initial CK (U/L)	>40,000	2
Etiology	Seizure, syncope, exercise, prolonged immobilization	2

Discussion

Rhabdomyolysis is a serious clinical condition with diverse etiologies, but drug-induced forms particularly statin-associated muscle injury represent a major cause in hospitalized and high-risk cardiac patients[8,15,16]. Statins can cause muscle damage through mitochondrial disruption, impaired oxidative phosphorylation, and destabilization of muscle membranes, with risk heightened by sepsis, dehydration, hypotension, renal impairment, and drug interactions[9–11]. In this case, the combination of critical illness, high-dose statin therapy, ongoing inflammation, and hemodynamic stress created conditions that precipitated severe rhabdomyolysis. The markedly elevated CK level, dark urine, acute rise in creatinine, and rapid clinical decline were hallmark features described in the literature[2,5,13]. Early application of the McMahon Score was valuable in identifying the risk of dialysis and guiding critical care management[7]. Timely fluid resuscitation, cessation of statins, sepsis control, and vigilant renal monitoring were essential to preserving kidney function and preventing progression to multiorgan failure. This case underscores the high index of suspicion required when statins are prescribed in unstable cardiac patients and highlights the importance of individualized risk assessment.

Conclusion

Statin-induced rhabdomyolysis, while uncommon, can rapidly progress to life-threatening complications particularly in patients with sepsis, hemodynamic instability, or renal vulnerability. Early recognition of characteristic symptoms such

as myalgia, dark urine, and rising creatinine, along with prompt discontinuation of statins and aggressive fluid resuscitation, is critical to ensuring renal recovery. This case reinforces the need to carefully evaluate high-risk cardiovascular patients before initiating or escalating statin therapy and emphasizes the value of interdisciplinary management in preventing dialysis and improving survival.

Learning Points

- Statin-induced rhabdomyolysis should be suspected in post-MI or post-PCI patients presenting with dark urine, rising creatinine, or severe myalgia.
- The McMahon Score is a valuable tool for early prediction of dialysis risk in rhabdomyolysis.
- Sepsis, dehydration, hypotension, and high-dose statin therapy significantly increase the risk of muscle toxicity.
- Early discontinuation of statins and aggressive IV hydration are the cornerstones of preventing AKI.
- Interdisciplinary care greatly improves outcomes in severe rhabdomyolysis.

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References

1) Torres PA, Helmstetter JA, Kaye AM, Kaye AD. Rhabdomyolysis: Pathogenesis, Diagnosis, and Treatment. Ochsner J. 2015;15(1):58–69.
2) Khan FY. Rhabdomyolysis: A review of the literature. Neth J Med. 2009;67(9):272–283.
3) Melli G, Chaudhry V, Cornblath DR. Rhabdomyolysis: An Evaluation of 475 Hospitalized Patients. Medicine (Baltimore). 2005;84(6):377–85.
4) Sauret JM, Marinides G, Wang GK. Rhabdomyolysis. Am Fam Physician. 2002;65(5):907–912.
5) Huerta-Alardín AL, Varon J, Marik PE. Bench-to-bedside review: Rhabdomyolysis — an overview for clinicians. Crit Care. 2005;9(2):158–169.
6) Stahl K, Rastelli E, Schoser B. A systematic review on the definition of rhabdomyolysis. J Neurol. 2020;267(4):877–882.
7) McMahon GM, Zeng X, Waikar SS. A risk prediction score for kidney failure or mortality in rhabdomyolysis. JAMA Intern Med. 2013;173(19):1821–1828.
8) Omar MA, Wilson JP. FDA adverse event reports on statin-associated rhabdomyolysis. Ann Pharmacother. 2002;36(2):288–295.
9) Sathasivam S. Statin induced myotoxicity. Eur J Intern Med. 2012;23(4):317–324.
10) Rosenson RS, Baker SK, Jacobson TA, Kopecky SL, Parker BA. An Assessment by the Statin Muscle Safety Task Force: 2014 Update. J Clin Lipidol. 2014;8(3 Suppl):S58–71.
11) Pasternak RC, Smith SC Jr, Bairey-Merz CN, Grundy SM, Cleeman JI, Lenfant C. ACC/AHA/NHLBI Clinical Advisory on the Use and Safety of Statins. Circulation. 2002;106(8):1024–1028.
12) Bagley WH, Yang H, Shah KH. Rhabdomyolysis. Intern Emerg Med. 2007;2(3):210–218.
13) Clarkson PM, Hubal MJ. Exercise-induced muscle damage in humans. Am J Phys Med Rehabil. 2002;81(11 Suppl):S52–69.
14) Zimmerman JL, Shen MC. Rhabdomyolysis. Chest. 2013;144(3):1058–1065.
15) Bosch X, Poch E, Grau JM. Rhabdomyolysis and acute kidney injury. N Engl J Med. 2009;361(1):62–72.
16) Warren JD, Blumbergs PC, Thompson PD. Rhabdomyolysis: A review. Muscle Nerve. 2002;25(3):332–347.