**Case report**

**Rhabdomyolysis Triggered by Statin Use: A Case Report**

**Abstract**

Rhabdomyolysis is a serious condition marked by the rapid breakdown of skeletal muscle, leading to the release of intracellular contents that can cause complications such as acute kidney injury (AKI). Statins are a known, though rare, cause of rhabdomyolysis. We report a case of severe statin-induced rhabdomyolysis in a male patient presenting with AKI and markedly elevated creatine phosphokinase levels. Despite supportive treatment with IV fluids and bicarbonate, he required hemodialysis. Following multiple sessions and continued care, his renal function and symptoms gradually improved. This case highlights the importance of early recognition and intervention in statin-associated rhabdomyolysis to prevent irreversible renal damage.

**Introduction**

Rhabdomyolysis is a complicated medical disorder in which injured or damaged skeletal muscle dissolves quickly. Intracellular muscle components such as creatine kinase (CK), myoglobin, lactate dehydrogenase, aldolase, and electrolytes are directly released into the circulation and extracellular space as a result of this skeletal muscle integrity violation which in turn causes life threatening complications such as acute renal failure (ARF), disseminated intravascular coagulation and electrolyte imbalances. One such cause of this condition is the use of statins [1]. We report a case of rhabdomyolysis in a patient caused by the consumption of Statins.

**Case presentation**

A male patient was admitted to the ward, with initial laboratory investigations revealing creatinine 13.3 mg/dL, eGFR 3.8 mL/min/1.73 m², urea 373 mg/dL, potassium 3.2 mmol/L, albumin 2.9 g/dL, and bicarbonate 16.9 mmol/L. He was started on intravenous (IV) fluids and sodium bicarbonate 50 mL every 8 hours for metabolic acidosis and presumed acute kidney injury (AKI).

On day 2, repeat investigations showed markedly elevated creatine phosphokinase (CPK) at 26,449 U/L, worsening creatinine 14.6 mg/dL, urea 412 mg/dL, and persistent low bicarbonate 16.7 mmol/L. Despite aggressive IV hydration, diuretics, and sodium bicarbonate infusion, the patient's renal function and urine output remained unchanged. Consequently, a right femoral catheter was placed, and emergency hemodialysis was initiated in the intensive care unit (ICU).

The initial impression was severe AKI secondary to rhabdomyolysis, possibly triggered by rosuvastatin 40 mg once daily. Empiric antibiotics with piperacillin-tazobactam were started, and internal medicine was consulted for glycemic control. A 2D echocardiogram was also performed, which showed the patient has increased left ventricular (LV) septal and posterior wall thickness with moderate diastolic dysfunction (reversible restrictive pattern), evidenced by a reduced E/A ratio of 0.5 and an elevated E/E' ratio of 15.2. The left atrium is dilated. The mitral valve is thickened and heavily calcified, particularly the posterior leaflet, with impaired mobility and a minimally reduced valve area; transmitral peak/mean gradient is 8/3 mmHg. There is mild mitral regurgitation (grade I/IV) and mild disease of the calcific mitral subvalvular apparatus. The aortic bioprosthesis shows mild post-operative stenosis with a peak/mean gradient of 32/19 mmHg. Additionally, there is mild functional tricuspid regurgitation and mild pulmonary hypertension with an estimated peak/mean pulmonary artery pressure of 42/30 mmHg. Over the course of the hospital stay, the patient underwent four hemodialysis sessions, in addition to continued IV hydration. Gradual improvement was observed in renal function, CPK levels, liver enzymes, and clinical symptoms, including muscle weakness, dyspnea, and lower limb pain. The patient’s urine output improved, and he was referred to physiotherapy for active and passive rehabilitation.

His platelet count remained below 100,000/µL, likely due to heparin-induced thrombocytopenia (HIT). He received IV albumin for three days due to hypoalbuminemia and third spacing, and IV iron supplementation was given for three days due to iron deficiency. Erythropoietin therapy was considered due to persistent anemia (Hb 9.1 g/dL)

**Discussion**

Rhabdomyolysis is a severe breakdown of skeletal muscle tissue that results in the release of intracellular muscle components into the bloodstream and muscular necrosis. In severe cases, it can result in significant enzyme elevations, electrolyte imbalance, and acute renal injury in 10–50% of patients. It is characterized by high serum creatine kinase (CK), muscle pain, and myoglobinuria. Trauma and crush syndrome, vascular ischaemia, toxins, infections and sepsis, salt and water metabolic disorders, and various medications are among the several factors that can cause acquired rhabdomyolysis [2]. Since rhabdomyolysis is often oligo-symptomatic or asymptomatic, its actual incidence is unknown. In the US, approximately 26,000 rhabdomyolysis cases are recorded each year [3].

Although the cause of a particular incidence of rhabdomyolysis is frequently understood, it is less evident how the different insults that can result in this disease finally produce muscle damage and necrosis. Regardless of the original insult, either direct myocyte damage or a breakdown in the energy supply within the muscle cells are the last steps that lead to rhabdomyolysis [4].

Lipophilic statins such as atorvastatin, simvastatin, fluvastatin, and pitavastatin are associated with a higher risk of muscle-related side effects, including myopathy and rhabdomyolysis, compared to hydrophilic statins like pravastatin and rosuvastatin. This is because lipophilic statins can more easily cross cell membranes, leading to greater accumulation inside muscle cells and increased toxicity [5].

Rhabdomyolysis results from injury to skeletal muscle cells, leading to membrane instability, energy depletion, and calcium overload. Various triggers, including statins, can impair mitochondrial function by depleting coenzyme Q10 and disrupting ATP production. ATP depletion inhibits key membrane pumps such as Na⁺/K⁺ ATPase and Ca²⁺ ATPase, leading to intracellular sodium and calcium accumulation. The increased intracellular calcium activates proteolytic enzymes like phospholipase A2, which degrade cell membrane components, further exacerbating calcium influx and structural breakdown. This cascade disrupts mitochondrial integrity, induces oxidative stress, and ultimately leads to muscle fiber necrosis and apoptosis. The release of intracellular contents into circulation can trigger systemic inflammation and complications such as acute kidney injury [3][5].

An increased creatinine kinase (CK) is typically used to confirm the diagnosis of rhabdomyolysis, though a comprehensive medical history and physical evidence may raise suspicions. Rhabdomyolysis has been diagnosed using a variety of criteria, but the most widely accepted one is that a CK >1000, or five times the upper limit of normal, is indicative of rhabdomyolysis. After an injury, CK usually rises for the first 12 to 24 hours before starting to fall. For prognostication of renal impairment, serial CK testing at 12-hour intervals until it starts to decline is helpful. Urine microscopy usually shows tubular casts but no blood; however, dipstick urinalysis may provide positive results for blood [6].

Acute kidney injury (AKI) prevention is the main goal of rhabdomyolysis treatment. You should stop taking any nephrotoxic medications. According to British guidelines, adults and children who have been diagnosed with rhabdomyolysis-related AKI risk and who are not volume overloaded should get rapid intravenous volume expansion to reach a high urine flow rate. Additionally, early fluid resuscitation is recommended over late fluid resuscitation in the latest Danish guidelines for preventing rhabdomyolysis-induced AKI. The fundamental idea is to use fluid supplementation to maintain a sufficient urine output [3].

**Conclusion**

This case underscores the serious risks associated with statin use, particularly the potential to trigger rhabdomyolysis and acute kidney injury requiring dialysis. Timely identification, immediate withdrawal of the statin, and early supportive care, especially with aggressive hydration and renal support, are key to improving patient outcomes. Even commonly used statins like rosuvastatin, which are generally considered safer, can lead to severe complications in susceptible individuals. Healthcare providers should remain vigilant for signs of rhabdomyolysis in patients presenting with muscle symptoms or unexplained renal dysfunction, especially those on statin therapy.

 **References:**

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