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ATORVASTATIN-INDUCED MYOPATHY WITH ELEVATED CREATINE KINASE: A CASE REPORT

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ABSTRACT **Background:** Statins are widely prescribed for hyperlipidemia and cardiovascular risk reduction, but their use may be limited by muscular adverse effects collectively termed statin-associated muscle symptoms (SAMS). These range from benign myalgias to severe myopathy and rare, life-threatening rhabdomyolysis. Recognition of statin-induced myopathy is critical for timely intervention. **Case Report:** We report the case of a 65-year-old male with coronary artery disease and hypertension, on long-term atorvastatin (80 mg) therapy, who presented with progressive muscle weakness, myalgia, and easy fatigability. Laboratory evaluation revealed elevated serum creatine kinase levels. A Naranjo causality assessment score of 6 indicated a probable association with atorvastatin. The drug was discontinued, and the patient received supportive therapy including hydration, steroids, and physiotherapy. Symptoms gradually improved, with marked decline in creatine kinase levels, and the patient regained mobility at discharge. **Conclusion:** This case highlights a classical presentation of atorvastatin-induced myopathy and emphasizes the importance of early recognition, risk factor assessment, and prompt discontinuation to prevent serious complications

KEYWORDS : Statins, Myopathy, Atorvastatin, Creatine Kinase, Muscle weakness, Corticosteroid Therapy

INTRODUCTION

Statins (HMG-CoA reductase inhibitors) remain cornerstone agents in the management of hyperlipidemia and the prevention of cardiovascular events. Although generally well tolerated, muscular adverse effects—collectively referred to as statin-associated muscle symptoms (SAMS)—pose a significant clinical challenge. These manifestations range from mild myalgia to severe conditions such as myopathy and, rarely, the life-threatening rhabdomyolysis.[1] Myopathy is among the most frequently reported adverse effects of statin therapy, with an estimated incidence of nearly 15%. [2] Several risk factors increase susceptibility, including high statin doses, advanced age, polypharmacy, and comorbidities such as diabetes and hypothyroidism. [3] Differentiating benign muscle pain without biochemical abnormalities from true statin-induced myopathy is critical, since the latter warrants immediate discontinuation of therapy and may require active medical intervention.

Here, we report a case of a 65-year-old male with coronary artery disease and hypertension who developed atorvastatin-induced myopathy, confirmed by elevated creatine kinase levels and clinical myalgia, which resolved after drug withdrawal. This case underscores the importance of vigilance and pharmacovigilance in routine statin therapy,especially among high-risk patients.

Case Report

A 65-year-old male with a past medical history of coronary artery disease (CAD) for the past 5 years and hypertension for the past 8 years presented to the outpatient department with chief complaints of progressive muscle weakness and pain in lower extremities bilaterally and to a lesser extent the upper Extremities (unable to walk more than 20 m) for the past 3 weeks, associated with easy fatigability and difficulty in climbing stairs. There was no history of recent trauma, fever, alcohol use, or new drug intake. He had been taking atorvastatin 80 mg once nightly for 5 years. Other medications included Enalpril 2.5mg, Amlodipine 5mg, Clopidogrel 75mg, Pantoprazole 40mg and Aspirin 150mg.

On Physical examination, the patient had reduced muscle power symmetrically in both upper legs (scored 4 out of 5) without any weakening in the upper limbs (strength scored 5 out of 5). There were no fasciculations, swelling of the affected muscles, or skin rash. Reflexes were universally weak. There were no sensory or visual deficits. Cardiovascular, respiratory and abdominal examination had no abnormailities. Heart rate (75 b.p.m.), blood pressure (130/80 mmHg), oxygen saturation (95 %), and respiratory rate (22breaths per minute) were normal. Laboratory investigations (Table 1) revealed that serum Creatine Kinase was elevated.

Based on the patient's long history of Atorvastatin use, myalgias with muscle weakness along with elevated creatine kinase, Atorvastatin induced Myopathy was considered.

Causality assessment using the Naranjo Adverse Drug Reaction Probability Scale gave a score of 6, indicating a probable adverse drug reaction (Table 2).

The patient was admitted and Atorvastatin was discontinued. The patient was advised complete rest and adequate hydration. The patient was started with IV methylprednisolone 40 mg every 8 hours for 3 days. Supportive therapy with paracetamol 500 mg was initiated for symptomatic relief of myalgia. Serum creatine kinase levels were monitored, which gradually declined over two weeks. IV steroids was switched to oral prednisolone. The steroid was tapered gradually as the patient improved symptomatically. He received intensive physiotherapy. Muscle weakness slowly improved and the patien was able stand on his own and walk about 10 m at the time of discharge.

Table 1 Laboratory Investigations

Parameter	Value	Normal Range
Creatine Kinase	700 U/L	<170 U/L
Hb	13.3 g/dL	12-16g/dL
Platelet count	179000	150000-400000
MCV	75fL	80-100fL
Random Sugar	180mg/dL	<200mg/dL
Blood urea	30mg/dL	10-50mg/dL
Serum creatinine	1.0mg/dL	0.6-1.3mg/dL
SGOT	20U/L	0-35U/L
SGPT	22U/L	0-35U/L
Serum sodium	140mmol/L	135-145mmol/L
Serum potassium	3.9mmol/L	3.5-5mmol/L

Table 2 Naranjo Adverse Drug Reaction Probability Scale For Atorvastatin Induced Myopathy

Question	Yes (Score)	No (Score)	Do Not Know (Score)	Patient's Score

1. Are there previous conclusive reports on this reaction?	+1	0	0	+1
2. Did the adverse event appear after the suspected drug was given?	+2	-1	0	+2
3. Did the adverse reaction improve when the drug was discontinued?	+1	0	0	+1
4. Did the adverse reaction reappear when the drug was re-administered?	+2	-1	0	0 (not done)
5. Are there alternative causes that could have caused the reaction?	-1	+2	0	+2
6. Did the reaction reappear when a placebo was given?	-1	+1	0	0 (not done)
7. Was the drug detected in blood (or other fluids) in toxic concentration	+1	0	0	0 (not done)
8. Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	+1	0	0	0 (not applicable)
9. Did the patient have a similar reaction to the same or similar drug in the past?	+1	0	0	0 (no history)
10. Was the adverse event confirmed by objective evidence? (e.g., ↑CK)	+1	0	0	+1
Total Score				6

DISCUSSION

Statin-associated muscle symptoms (SAMS) span a spectrum from self-limited myalgias to myopathy and, rarely, rhabdomyolysis, and remain a leading reason for non-adherence or discontinuation of therapy. Recognizing this spectrum is essential to balance cardiovascular benefit against harm. The European Atherosclerosis Society (EAS) consensus statement emphasizes structured assessment of symptom pattern, temporal relationship to statin exposure, and objective testing (e.g., creatine kinase, CK) to guide management[4]

The clinical features in our patient—proximal muscle weakness, marked CK elevation, temporal association with atorvastatin 80 mg, and resolution after drug withdrawal—are typical of statin-induced myopathy and mirror prior case reports. For example, Brunda et al. (2023) reported a statin-associated myopathy case in which symptoms and laboratory abnormalities were temporally linked to statin use and improved after discontinuation, underscoring the usual clinical course and management considerations.[2] Similarly, De Cock et al. (2018) described a case of statin-induced muscle toxicity emphasizing the need for early recognition and dechallenge to prevent progression.[5] Together, these reports and our case highlight a reproducible pattern seen across settings.

The pathophysiology of statin myotoxicity is multifactorial. Proposed mechanisms include alterations in the mevalonate pathway (affecting downstream isoprenoids), mitochondrial dysfunction and impaired energy production, coenzyme Q10 depletion, and—in rare cases—autoimmune necrotizing myopathy that persists despite statin cessation and may require immunosuppression. Distinguishing a toxic myopathy (expected to improve after stopping the drug) from immune-mediated forms (which may worsen or persist) has implications for prognosis and further therapy. [6]

Multiple factors increase susceptibility to SAMS: higher statin doses, advanced age, comorbidities (such as diabetes and hypothyroidism), polypharmacy and interacting drugs, and genetic predisposition (e.g., SLCO1B1 variants). In our patient, advanced age and a relatively high atorvastatin dose were likely contributory. Recognising and modifying reversible risk factors (drug interactions, untreated hypothyroidism, recent vigorous exercise) is an important first step in management[7]

From a pharmacy practice and pharmacovigilance standpoint, systematic documentation (temporal course, CK trends), application of a causality scale (e.g., Naranjo), reporting to national pharmacovigilance programs, and clear patient counseling about symptom recognition are pivotal. Such steps support safer long-term lipid management while preserving the proven cardiovascular benefits of statins for high-risk patients.

This case illustrates a typical presentation of atorvastatin-related myopathy, the importance of prompt discontinuation and follow-up, and practical alternatives for lipid management in patients who cannot tolerate standard statin therapy

CONCLUSION

Statin-induced myopathy, though uncommon, remains a clinically significant adverse effect that requires vigilance. Early identification through careful symptom assessment, creatine kinase monitoring, and recognition of risk factors can prevent progression to severe complications. Prompt drug withdrawal, appropriate supportive measures, and pharmacovigilance reporting are essential steps in management. This case emphasizes the need for individualized therapy and patient education to ensure safe and effective long-term use of statins.

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