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Case Report

Statin-Induced Necrotizing Myopathy: A Case Report of Severe Muscle Weakness and Recovery

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Abstract:
Statin-induced necrotizing myopathy (SINM) is an infrequent yet severe complication associated with the use of statin medications, which are commonly prescribed to manage hyperlipidemia and reduce the risk of cardiovascular events. We reported a case of a 50-year-old homemaker with an eight-month history of unstable angina, who subsequently underwent coronary stent placement and initiated aspirin/atorvastatin therapy. Eight to ten days following the onset of lower limb pain and weakness, the patient experienced a sudden decline in upper limb strength. Notably, there was no history of trauma, sensory loss, incontinence, respiratory symptoms, fever, or relevant comorbidities. Clinical examination revealed severely reduced muscle power (0/5) in the lower limbs and moderate weakness (1/5) in the upper limbs, along with absent deep tendon reflexes bilaterally. This presentation is consistent with statin-induced necrotizing myopathy (SINM), a rare but serious side effect of statin therapy, which requires prompt recognition and management to mitigate potential complications.
Keywords: Statin-induced necrotizing myopathy, muscle weakness, cardiovascular risk factors

Introduction:

Statin-induced necrotizing myopathy (SINM) is a rare but potentially debilitating side effect of statin therapy, characterized by severe muscle weakness and muscle damage. Statins, widely prescribed for managing hyperlipidemia and preventing cardiovascular events, have been associated with various muscle-related adverse effects, with SINM being one of the most severe and least common. Our case report presents the clinical presentation of a patient who experienced a profound decline in muscle strength and function due to SINM, emphasizing the importance of recognizing and promptly managing this condition. Our report not only discusses the patient's challenging course of illness but also sheds light on their remarkable recovery, highlighting the potential for successful rehabilitation and improved quality of life for individuals affected by SINM. Understanding this condition is crucial for healthcare professionals to effectively monitor and manage patients on statin therapy and ensure the safest and most effective treatment strategies.

Case Presentation:

Herewith we reported, a case of 50-year-old female presented with an eight-month history of unstable angina, during which she underwent coronary angiography and received a stent in the left anterior descending artery. She was subsequently initiated on aspirin (75mg) and atorvastatin (20mg) once daily. However, within 8-10 days, she reported experiencing pain and weakness in both her lower limbs, followed by a sudden decline in upper limb strength over 3-4 days. The patient had no prior history of trauma, sensory loss, incontinence, respiratory symptoms, fever, or relevant medical conditions, such as tuberculosis, asthma, or diabetes mellitus. Additionally, the patient had no history of smoking, tobacco use, or alcohol consumption.

Six months prior to the onset of her symptoms, the patient underwent coronary angiography, which revealed single-vessel disease in the left anterior descending artery (LAD) necessitating stenting. Following the procedure, she was prescribed ticagrelor (90mg) and rosuvastatin (40mg) daily.

Upon examination, the patient's blood pressure was 130/90 mmHg, with a pulse rate of 90 beats per

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minute. Her SpO2 was 98% on room air, and the respiratory rate was 15 breaths per minute. Physical examination revealed no pallor, cyanosis, clubbing, icterus, lymphadenopathy, or edema. The patient was awake, alert, and oriented during neurological assessment, with normal cranial nerve function and higher mental faculties. Muscle tone was within the normal range, and there was no muscle wasting. Notably, muscle power in both lower limbs was severely diminished (0/5), while power in both upper limbs was moderately weakened (1/5). Deep tendon reflexes were absent in both upper and lower limbs. Sensory system examination yielded normal findings, with no meningeal symptoms or cerebellar signs.

Investigations:

Haemogram	
HB	12.5 g/dl (11.6 – 15.0 g/dl)
TLC	12,700/mic lt (4000-10000/mic lt)
Platelets	3,34,000/mic lt (150000-410000/mic lt)
Electrolytes	
Sodium Potassium	131 mmol/Lt (136 to 145 mmol/Lt) 5.8 mmol/Lt (3.5-5.1 mmol/Lt)
Phosphorus	>9 mg/dl (2.6-4.7 mg/dl)
Calcium	7.9 mg/dl (8.6-10.2 mg/dl)
Magnesium	3.2 mg/dl (1.8-2.4 mg/dl)
LFT	
Bilirubin	Total-0.59 mg/dl (0.22 – 1.20 mg/dl) Conjugated-0.27 mg/dl (upto 0.5 mg/dl)
SGOT[AST]	325 U/L (8 to 43 U/Lt)
SGPT[ALT]	362U/L (7 to 45 U/Lt)
ALP	83U/L (35 to 104 U/Lt)
RFT	
Urea	273 mg/dl (17-49 mg/dl)
Creatinine	11.25 mg/dl (0.6 to 1.2 mg/dl)
S. Uric acid	14.2 mg/dl (3.7 to 8.00 mg/dl)
S. Proteins	Total-6.5 mg/dl (6.4-8.3 g/dl), Albumin-3.1 g/dl (3.5-5.2 g/dl)
CPK-Total (Creatine phosphokinase total)	15,034 U/L (Normal value in females- 26 to 192 U/L)
PT-INR	13.3 Sec; INR-1.14
HIV/HBsAG/HCV	Non Reactive
ANA (IF) AND ANA BLOT	Negative
TFT	Normal
ESR	87 mm/hr (<30 mm/hr)
CRP	27.70 mg/L (<0.3 mg/dl)
CSF examination	WNL
Cultures-Blood, Urine and CSF	No Growth
Urine	R/M; Proteins-trace, UPCR- 2.63 (moderate proteinuria 1.10-3.00) Urine for myoglobin-positive

USG Abdomen and pelvis- WNL (Within normal limit)
MRI Whole spine screening with dedicated cervical spine- WNL
MRI Brain - WNL

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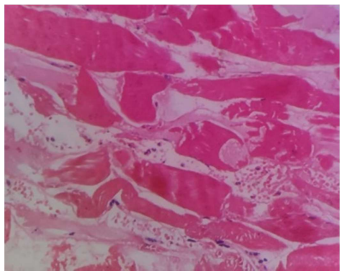
Nerve conduction study-WNL

Electromyography is done in the following muscles (left tibialis anterior, right vastus lateralis, and right biceps brachii) showed spontaneous activity in the form of positive, sharp waves and fibrillations and small and Polyphasic Motor Unit Action Potentials (MUAP) were noted in the tested muscles.
Early and complete recruitment was noted in all tested muscles, suggesting a myogenic pattern.
Muscle biopsy showed necrosis in individuals as well as small groups of muscle fibers along with loss of striations and surrounded by macrophages and occasional foci of lymphocytes.
There was no Fibrinoid necrosis, perivascular atrophy, or inclusion bodies. Regeneration o muscle fibers were seen, but there was no significant fibrosis.



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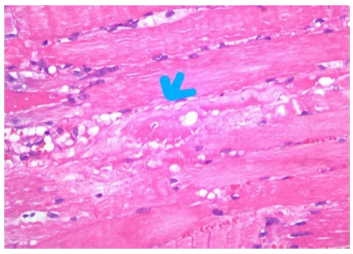

40 X magnification

Figure – Prominent myonecrosis is present with a mild interstitial inflammatory infiltrate.
In light of statin-induced muscle wasting, increased CPK-Total, negative ANA Blot and IF, normal NCV Study, inflammatory muscle disease on EMG, and necrotizing myopathy on muscle biopsy
Statin-induced necrotizing myopathy (SINAM) was determined to be the cause.

Following the cessation of statin therapy, the patient's treatment regimen was adapted to address Statin-Induced Necrotizing Myopathy (SINM). She was administered Tab. Wysolone at a dosage of 1 mg/kg, which was gradually tapered over four weeks. Concurrently, hemodialysis with alternate-day plasmapheresis was initiated to manage her condition effectively. Continuation of physiotherapy was essential to aid her recovery. Encouragingly, after four weeks of this comprehensive treatment plan, significant improvements in muscle weakness were observed. By the end of six weeks, the patient had made substantial progress, regaining her ability to walk unassisted and independently perform daily activities, including rising from a seated position.

Discussion:

The clinical presentation of this patient aligns with statin-induced necrotizing myopathy (SINM), a rare but severe side effect associated with statin therapy. SINM is characterized by progressive muscle weakness and muscle damage, often necessitating immediate discontinuation of statin treatment. In this case, the patient's initiation of aspirin/atorvastatin therapy coincided with the onset of her myopathic symptoms. It is essential for healthcare providers to promptly recognize and manage SINM to minimize complications and optimize patient care.

In the context of the presented clinical and investigative findings, the diagnosis of Statin-Induced Necrotizing Myopathy (SINM) becomes

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apparent. The elevated creatine phosphokinase (CPK-Total), characteristic myogenic electromyographic patterns, and muscle biopsy results displaying myonecrosis and inflammatory infiltrate align with SINM. Importantly, the negative autoimmune markers, normal nerve conduction studies, and absence of other systemic diseases support this conclusion. SINM is a rare but severe side effect of statin therapy, underscoring the necessity for vigilant monitoring and timely recognition in patients receiving statins. This case highlights the significance of considering SINM in patients presenting with unexplained muscle weakness and underscores the importance of prompt management to mitigate complications. This case highlights the potential for successful rehabilitation in individuals with SINM when prompt and appropriate interventions are employed, emphasizing the importance of recognizing and managing this rare but debilitating condition.

Conclusion:

This case report underscores the importance of considering SINM as a potential complication of statin therapy, particularly in patients with preexisting cardiovascular risk factors. Early diagnosis and prompt intervention are crucial in managing SINM and improving patient outcomes. Healthcare professionals should be vigilant in monitoring and assessing patients for adverse effects when prescribing statin medications.

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