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Case Report

# HMG-CoA Reductase Autoantibody-Mediated Myositis: A Case Report

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## Abstract

Statin-associated muscle symptoms are a common adverse effect of statin therapy; however, not all cases of muscle pain necessarily require discontinuation of treatment. We report the case of an elderly male patient who was admitted with unexplained physical weakness due to rhabdomyolysis. Despite cessation of statin therapy, no clinical improvement was observed. Radiological imaging suggested myositis of the lower-extremity muscles, and serological testing demonstrated the presence of HMG-CoA reductase antibodies, confirming the diagnosis of HMG-CoA reductase autoantibody-mediated myositis. Treatment with intravenous immunoglobulins was administered, resulting in a marked reduction of creatine kinase levels and improvement in physical condition. In cases of persistent or unexplained rhabdomyolysis despite discontinuation of statin therapy, autoimmune myositis should be considered as an important differential diagnosis. When confirmed, immunosuppressive therapy with intravenous immunoglobulins or glucocorticoids is the treatment of choice.

**Keywords: HMG-CoA reductase autoantibody; Autoimmune myopathy; Myositis; Statin; Rhabdomyolysis**

## Abbreviations

HMG-CoA: 3-Hydroxy-3-Methylglutaryl Coenzyme A; CK: Creatine Kinase; BMI: Body Mass Index; MRI: Magnetic Resonance Imaging; STIR: Short T1 Inversion Recovery; Anti-Jo-1/His-tRNA: Anti-Histidyl-Transfer Ribonucleic Acid; PM-Scl: Polymyositis/Scleroderma; Ig: Immunoglobulin; SINAM: Statin-Induced Necrotizing Autoimmune Myopathy; SRM: Statin-Related Muscle Toxicity

## Introduction

Statin medications have become a cornerstone in the prevention and management of cardiovascular disease by effectively lowering low-density lipoprotein cholesterol and reducing the risk of myocardial infarction and stroke. However, despite these clear benefits, a significant proportion of patients report muscle-related symptoms during statin therapy, ranging from mild myalgia to more severe myopathy. These statin-associated muscle symptoms present a clinical challenge, as they can compromise adherence, prompting patients to discontinue treatment prematurely. Furthermore, distinguishing statin-related symptoms from other causes of myalgia-such as hypothyroidism, vitamin d deficiency, or autoimmune processes can be difficult.

## Case Presentation

A 76-year-old male patient was admitted to the hospital due to undefined physical exhaustion, weakness, and gait disturbance. He reported difficulties walking as well as muscle pain and fatigue in the lower extremities, which had been present for approximately six months. Brain and nervous system function had been evaluated two months earlier in an outpatient neurological department, without any significant findings.

His past medical history included type 2 diabetes mellitus, paroxysmal atrial fibrillation, previous cerebral infarction, prior carotid endarterectomy due to left internal carotid artery stenosis, and multiple surgeries for a coccygeal fistula. His home medications consisted of carvedilol, rivaroxaban, atorvastatin, metformin, dapagliflozin, oral semaglutide (as part of a dementia study), and insulin glargine. Occupational history revealed previous exposure to asbestos. On physical examination, he appeared in slightly reduced general physical condition and was overweight (BMI 28 kg/m$^2$). The physical examination was unremarkable except for an abnormal vibration test, consistent with known diabetic polyneuropathy.

Neurological examination showed hypoesthesia on the soles of the feet, while superficial sensation was otherwise intact. Deep tendon reflexes in the arms and legs were moderately brisk and symmetrical, although the Achilles tendon reflex could not be reliably elicited bilaterally. The Babinski reflex was negative on both sides. Vibration sense (pallesthesia) at the malleoli was rated 5/8 bilaterally.

Laboratory tests revealed no signs of infection; however, CK was markedly elevated at 4222 U/l, and myoglobin was elevated at 2984 µg/l. After discontinuation of the statin therapy, the patient reported slight improvement in proximal leg weakness, accompanied with a mild decrease in CK levels (down to 2377 U/l).

Duplex sonography ruled out any significant peripheral arterial occlusive disease. Echocardiography showed good left ventricular systolic function, without regional wall motion abnormalities or significant valvular disease.

MRI of the thigh revealed findings consistent with bilateral myositis. The adductors, biceps femoris, semimembranosus - and on

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the left side - the distal portion of the gluteus maximus, along with the bilateral gluteus minimums, and left gluteus Medius showed increased signal intensity in the STIR sequences. This signal enhancement persisted after intravenous contrast administration.

Based on the clinical, laboratory and radiological findings a statin-induced myopathy was suspected.

Serological testing for specific autoantibodies, including anti-JO-1/His-tRNA synthetase, PM-Scl, and HMG-CoA reductase antibodies, was performed. The HMG-CoA reductase antibody level was elevated at 66 U/ml (normal <20 U/ml), confirming the diagnosis of HMG-CoA reductase antibody-mediated myositis triggered by statin therapy. At this stage, no biopsy was performed.

Intravenous immunoglobulins were administered over a 5-day period, with a total dose of 150 g. Treatment was initially started with Opti globin®, but due to intolerance (hypertensive crisis and chills), therapy was switched to Ig Vena®. During treatment, the patient reported moderate improvement in muscle weakness. Laboratory follow-up on day four after initiation of therapy showed a reduction in CK levels from 2134 U/l on the first treatment day to 1328 U/l.

## Discussion

Statins influence lipid metabolism by reducing endogenous cholesterol synthesis in the liver through inhibition of the enzyme 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase [1]. Some patients develop muscle symptoms while taking statins [2], although the causal relationship is not always clear [3]. Symptoms may include myalgia, muscle cramps, limb pain, or non-specific musculoskeletal discomfort [2]. Mild myalgia occurs in approximately 5% to 10% of patients, whereas complications such as rhabdomyolysis, and in particular, Statin-Induced Necrotizing Autoimmune Myopathy (SINAM), are rare [3]. There is no substantial variation in the relative effects of different statins [2]. The detection of autoantibodies against HMG-CoA reductase supports the diagnosis of statin-induced autoimmune myopathy. Statin-Related Muscle Toxicity (SRM) is classified into seven grades [4], with SRM 0 representing asymptomatic CK elevation and SRM 6 denoting the most severe form of autoimmune necrotizing myositis, characterized by positive HMG-CoA reductase autoantibodies, corresponding biopsy findings, and no significant improvement in symptoms after discontinuation of statin therapy.

When statin-induced symptoms such as muscle pain occur, switching drug class or discontinuing statin therapy should be considered [3]. If CK levels are elevated, statin therapy should be discontinued. In cases of suspected Statin-Induced Necrotizing Autoimmune Myopathy (SINAM) - characterized by proximal muscle weakness, markedly elevated CK, persistence of symptoms, and lack of CK reduction after discontinuation testing for HMG-CoA reductase autoantibodies is recommended. These antibodies are present in approximately 94% of SINAM patients. Muscle biopsy in such cases typically shows myonecrosis. SINAM is associated with genetic variants in the HLA gene, particularly HLA-DR11 and the DRB1*11:01 allele. Early recognition, diagnosis, and immunosuppressive treatment of statin-induced necrotizing autoimmune myopathy are essential to prevent progression to severe and often irreversible muscle weakness [2,3].

## Conclusion

In patients who develop muscle weakness or muscle pain during statin therapy, statin-induced myositis or myopathy should be considered as a differential diagnosis and investigated accordingly. If no clinical or laboratory improvement is observed after discontinuation of the statin, further diagnostic evaluation should include MRI imaging, testing for HMG-CoA reductase autoantibodies and, if necessary, muscle biopsy of the affected region.

Therapeutically, immunosuppressive treatment options include oral steroids, methotrexate, Intravenous Immunoglobulins (IVIG), and rituximab [5,6]. Among these, IVIG should be considered a first-line therapy [6].

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