International Journal of Health Sciences and Research
[Indexed, Peer-Reviewed Journal]
Volume 15; Issue: 10; October 2025
Website: www.ijhsr.org
Case Report *ISSN: 2249-9571*

Statin-Induced Necrotizing Autoimmune Myositis (SINAM)- Rare Chronic Progressive Disease in an Elderly- First Reported Case in Bahrain

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DOI: https://doi.org/10.52403/ijhsr.20251018

ABSTRACT

Statins are commonly used for controlling lipid profile; however, it has spectrum of side effects that ranged from very mild myalgia to a serious fatal necrotizing myositis. An entity known as statin induce necrotizing autoimmune myositis (SINAM) is characterized by proximal muscle weakness and elevated creatinine kinase.
If not treated, it can lead to rhabdomyolysis, thus managing it in early stages is crucial. Nevertheless, steroids remain the milestone of treatment. In this study, we describe a case of 74-year-old patient who was on daily statin and developed right thigh pain and bilateral progressive proximal lower limb weakness for several months until the point where he became almost bedridden. Labs of this patient showed high CK level and positive HMG-CoA enzyme, his MRI showed show abnormal high T2 signal intensity of pelvic muscles with reduced muscle bulk and fatty infiltration. Statin was discontinued and patient received steroieds and IVIG and was monitored until discharge.

Keywords: Statins, Myopathy, necrotizing autoimmune myositis, vasculitis, rhabdomyolysis

INTRODUCTION

The usefulness of statins therapy in preventing primary and secondary atherosclerotic cardiovascular diseases is well supported by many studies, [(1)] hence it is one of the most commonly prescribed medication in healthcare practice. [(2)] However, patient's compliance might be suboptimal due to its side effects which varies from mild muscle ache to life-threatening rhabdomyolysis. [(3)]
Statin induce necrotizing immune-mediated myopathy (SINAM) is another complication that is very rare and might be missed in many occasions. [(4)] Thus, early recognition of statin side-effects is the foremost important step.

Here, we report a case of SINAM to withdraw readers' attention to treatable potential- life threatening condition.

CASE PRESENTATION

A 74-year-old gentleman with a background history of type 2 diabetes mellitus, hypertension and dyslipidemia since 2016, taking biphasic insulin as part 26 international unit twice per day, vildagliptin 50mg along with metformin 1000mg twice per day and atorvastatin 40mg once per day. Had a previous history of bilateral total knee

replacement secondary to advanced osteoarthritis in 2014-2015, and he is ambulatory with walking aid. In 2023, he was referred from a private hospital with a history of right thigh pain and bilateral progressive proximal lower limb weakness for several months. He became almost bed bound mobilized around with wheelchair. He denied history of pain in his knees nor injury nor any history of fall or a trauma. His pain was new to him. His musculoskeletal examination revealed (upper limb power 2/5 proximally bilateral and lower power was 4/5 distally bilateral). Patient had no acute tenderness on palpation, muscle tone was normal and sensation on both side was intact, both ankle and knee reflexes were absent. He was advised for admission for further work up. His blood tests showed:

Table 1: Laboratory findings.

Hematology		**Reference Range**
WBC	4.38 X10^9/L	3.6-9.6
HB	11.4 g/dl	12-14.5
Platelet	239X10^9/L	150-400
Biochemistry		
Urea	3.8 mmol/L	3.2-8.2
Creatinine	55 MUmol/L	55-96
ALK	82U/L	46-116
ALT	64 U/L	<41
GGT	<6 U/L	<73
Glucose Fasting	3.8 mmol/L	3.9-5.6
HBA1C	42 mmol/mol	29-42
Special Coagulation		
Anti-thrombin 3 Assay	85%	79-112
Protein C	108.7%	70-140
Protein S	60.2%	60-130
Lupus Anticoagulant		
LA1 (Screening test)	32.5 s	31-44
LA2 (confirmation test)	31.3 s	30-38
LA1:LA2 Ratio	1.04 s	0.8-1.2
comments	LA not detected	
Urine RM	Unremarkable	
Hematology		
ESR	8 mm/hour	<20
Immunology/ Serology		
CRP	0.17 mg/L	0-3
ANA	-ve	
Rheumatoid factor test	9.69 IU/ml	<14
c-ANCA Ab (IF)	-ve	
p-ANCA	-ve	
Anti-smooth muscle antibody	-ve	

Initial laboratory findings showed markedly increased creatinine kinase in private hospital reaching up to 5870 IU (N: 171). When came to our hospital it was 2307 IU. See below table 2 for the trend of the documented CK throughout his life. In addition, Hydroxymethylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors level came up positive.

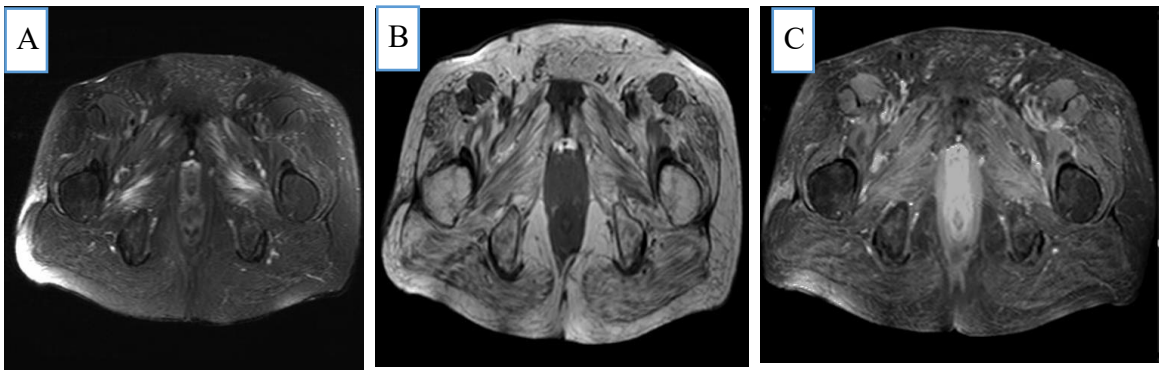
Table 2: CK trends of patient.

Test	**Result**	**Result Date**
CREATINE KINASE	297	07/7/23
CREATINE KINASE	562	26/02/23
CREATINE KINASE	807	10/02/23
CREATINE KINASE	727	09/02/23

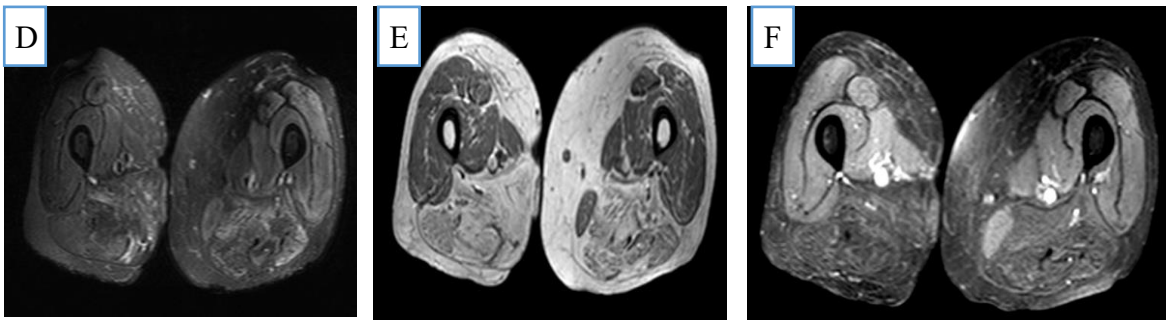
CREATINE KINASE	1224	08/02/23
CREATINE KINASE	1920	07/02/23
CREATINE KINASE	2307	06/02/23
CREATINE KINASE	2398	05/02/23
CREATINE KINASE	14631	03/08/19
CREATINE KINASE	13593	03/08/19
CREATINE KINASE	3758	09/03/17
CREATINE KINASE	4506	06/03/17
CREATINE KINASE	5612	05/03/17
CREATINE KINASE	3935	01/03/17
CREATINE KINASE	4905	28/02/17
CREATINE KINASE	5267	28/02/17
CREATINE KINASE	5703	28/02/17
CREATINE KINASE	6524	27/02/17
CREATINE KINASE	142	02/03/16

Chest x-ray was unremarkable.

MRI femurs revealed diffuse areas of intramuscular signal abnormality and homogenous post contrast enhancement involving the majority of the bilateral pelvic and thigh groups, more pronounced at piriformis, obturator internus and adductor muscles bilaterally and left hamstring muscles. No signs of myonecrosis. Normal marrow signal. No focal bony lesion. Small joint effusion bilaterally.



MRI images obtained in a 74 years old male patient, Image A: Illustrate axial T2-weighted with fat saturation and ImageB: shows axial T1- weighted MR images which show abnormal high T2 signal intensity of pelvic muscles with reduced muscle bulk and fatty infiltration, image C: Demonstrate T1 axial with fat saturation and contrast adminstration shows homogenous post contrast enhancement of affected muscles.



Figures D, E, F: Shows MRI images that we obtained for same patient at thigh level with same sequences shows similar MRI abnormality involve thigh muscles with homogenous post contrast enhacment and no myonecrosis.

Nerve conduction study was done and showed normal F study and normal motor conduction study of peroneal and posterior tibial nerves' and, normal sensory conduction study of superficial peroneal and sural sensory nerves.

The statin was immediately stopped once his CK report came and he was started on IV hydration and given methylprednisolone 1000mg once daily IV for 3 days then started on oral prednisone 1mg/kg with two doses of IVIG 7000 mg two weeks apart. He was kept in hospital for daily monitoring of is renal profile, patient was discharged home after 6 days with close follow up at the clinic.

DISCUSSION

Statins are one of the most effective lipid lowering agents. They work by inhibiting HMG-CoA reductase, which is considered as a critical rate-limiting step in cholesterol synthesis. There are various types of statins, however it is well-established that statins effects on lipid profile is a dose-dependent. Although statins have numerous benefits, side effects such as elevated liver enzymes and musculoskeletal manifestations are well recognized.[5] The prevalence of statin myopathy is estimated to be 6-10%. [(6)]

Despite the fact that exact mechanism of myopathy is not fully understood, it was suggested that it reduces the serum coenzyme q10 levels which in turn may result in mitochondrial dysfunction. [(7)]

And it is assumed that patients taking high potent statins, or higher doses are more susceptible to statin toxicity, and it was seen in one of the studies that atorvastatin are more likely to developed statin induce myopathy in comparison to simvastatin and rosuvastatin. [(8)]

A recent meta-analysis that pooled more than 4 million patients highlighted that statin intolerance prevalence is relatively low.[9] Recently there is an increasing global attention regarding rare reported adverse effects of statins such as disabling conditions and potentially fatal complications like SINAM and rhabdomyolysis. [(10)]

One of the largest studies showed that the prevalence of rhabdomyolysis estimated to be 1 per 10,000 patients. [(11)] On the other hand, SINAM has autoimmune nature where most patients with SINAM found to have positive anti-HMGCR antibodies and it is hypothesized that exposure to statin therapy, upregulate HMGCR in genetically susceptible patients. [(12)]

Also, it is suggested that the interaction between statin and HMG-CoA reductase might lead to structural modifications within the protein, generating epitopes that induce immune reaction. [(13)]

Patients with SINAM and statin-induce myalgia commonly shares similar symptoms, as they typically present with symmetrical progressive proximal muscle weakness, however the myalgia in SINAM usually last longer after statin cessation, as well creatinine kinase level usually markedly elevated and persist for longer duration post statin discontinuation. [(14)]

In these patients, muscle biopsy characterized by myofiber necrosis and regeneration, with no lymphocytic infiltration. In addition, the presence of major histocompatibility complex (MHC) class 1 is usually characteristic. [(15)]

The management of patients with statin therapy with anti-HMGCR myopathies, encompass discontinuation of this class of medications and initiation of immunosuppressive therapy. Steroids is the drug of choice, with possible addition of steroids sparing agents in refectory cases such as intravenous immunoglobulin (IVIG). [(16)]

In our case, we found that our patient was has been taking atorvastatin for many years prior his presentation, and this was also seen in some studies that highlighted delayed onset of SINAM. [(17)] As well he had positive anti-HMGCR antibodies which can be diagnostic in suspicious clinical picture with the absence of other factors for myopathy.

Fortunately, he did not develop acute kidney injury despite the factor that he had persistent myoglobinemia un-noticed for many years prior to his subacute presentation and responded very well with good hydration, high doses of steroids and IVIG.

CONCLUSION

Maintaining high index of suspicions of statin induce myonecrosis is potential, especially when patient complains of myalgia. Also, monitoring CK level in elderly with multiple co-morbidities may early detect and prevent progression to rhabdomyolysis. The mainstream of management is typically steroids.

Declaration by Authors
Ethical Approval: Not applicable
Acknowledgement: None
Source of Funding: None
Conflict of Interest: The authors declare no conflict of interest.

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How to cite this article: Maryam Adam Ali, Fatima Haji, Ahmed Alnaggar Statin-induced necrotizing autoimmune myositis (SINAM)-rare chronic progressive disease in an elderly-first reported case in Bahrain. *Int J Health Sci Res.* 2025; 15(10):171-175. DOI: *https://doi.org/10.52403/ijhsr.20251018*

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