

Statin induced myopathy presenting as quadriparesis: a case report

Introduction

Statins act by inhibiting endogenous cholesterol production by competitive inhibition of HMG-CoA reductase, the enzyme that catalyzes the rate limiting step in cholesterol synthesis.¹ They are used widely to treat dyslipidemia for treatment of atherosclerosis. Its role in reducing mortality from atherosclerotic cardiovascular diseases has been well established.² The use of statin is associated with a wide range of muscle toxicity, which includes myalgia, myositis, myopathy and rhabdomyolysis; most of which resolves after its discontinuation.³

Case report

A 47 year old female with no known comorbidities presented to our centre with the complaints of muscle pain and weakness of bilateral lower limb which progressed gradually to involve upper limb and the whole body in duration of two weeks. On further questioning, she revealed the history of intake of statins since last 2 weeks for raised cholesterol levels in routine checkup.

On physical examination, she had reduced muscle power in all four limbs (RUL: 3/5, LUL: 3/5, RLL: 2/5, LLL: 2/5). Rest of the general and systemic examination was unremarkable. Laboratory workup showed significantly raised creatinine phosphokinase (CPK): 6570 U/L. Antinuclear antibody (ANA)-ELISA and rheumatoid factor (RA) factor was negative. Cerebrospinal fluid (CSF) analysis was done which revealed no abnormal findings. Nerve conduction study showed features of early axonal motor neuropathy affecting lower and upper limbs. Normal sensory nerve conduction study; features consistent with possibility of motor neuropathy. The clinical diagnosis of Statin induced myopathy was made based on the association between the exposure to statin and her symptoms. Additionally, there was no evidence to favor alternate diagnosis such as connective tissue disorder, CNS infections, malignancy, and active viral infection which supported the diagnosis of Statin induced Myopathy.

The patient was admitted to the Intensive Care Unit for five days. Statins were stopped since the time of hospital admission and pulse steroid (Inj. Methylprednisolone) therapy was started. Tablet Prednisolone was started after completing 5 days of pulse steroid therapy. Gabapentin and Methylcobalamin were added for neuropathic pain. After the discontinuation of statins and being managed in the Intensive Care Unit for 5 days all her symptoms subsided and she was discharged.

Discussion

Although statin are usually safe and generally well tolerated by most people, muscular side effects are commonly encountered by its users. Different mechanisms have been involved in the mechanism of statin toxicity such as: mitochondrial dysfunction, oxidative stress and impaired mevalonate mechanism.^{4,6}

Different spectrum of muscle disorders are associated with use of statins ranging from myalgia, myositis, rhabdomyolysis to asymptomatic rise in creatinine kinase.⁷ These patients present with varying symptoms such as fatigue, muscle ache, muscle tenderness,

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nocturnal cramping and tendon pain. Another entity is Immune Mediated Necrotizing Myopathy (IMNM), an autoimmune myopathy in which patients present with progressive proximal muscle weakness in both limbs.⁸ Our patient had weakness of all four limbs as a manifestation of muscular toxicity owing to use of statin which is quite an uncommon presentation. Statin induced quadriparesis is very rarely reported.

Creatinine kinase (CK) level is one of the initial investigations to assess muscle damage and its severity.⁴ Our patient also had significantly raised creatinine kinase level which gradually decreased as her symptoms improve. Other investigations which could be helpful are electromyography and muscle biopsy. Unfortunately, our patient did not consent for these investigations.

Conclusion

Statins are being widely prescribed all over the world, it is important for all to know and recognize these adverse effects. It is thus important to be able to distinguish between benign muscle pain and serious myopathies caused by the use of statins. In most cases the levels of creatine kinase decrease gradually with discontinuation of the drug and all symptoms are reversed over time.

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