Author’s response to reviews

Title: Statin associated Myasthenia Gravis: A case report.

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Author’s response to reviews: see over
Dr Kurukumbi

We thank Dr Kurukumbi for his helpful comments.

We have attempted to redraft the case report in a more narrative style as suggested.

Regarding the specific points raised:

1. Simvastatin was discontinued after 4 weeks.
2. The lipid profile on admission showed a cholesterol of 6.1 mmol/l, LDL 3.8, HDL 1.0, TG 2.8 – all non-fasting.
3. Simvastatin had been taken for 4 weeks two months prior to admission but stopped because of myalgia, weakness, and an elevated CK of 2599 (normal < 200). This all settled on Simvastatin withdrawal.
4. The Atorvastatin was started 4 days after admission (because of the incorrect putative diagnosis of brainstem stroke). The CK was normal prior to starting the Atorvastatin but climbed after initiation. It was stopped after two weeks.
5. The MRI was ordered but cancelled when the diagnosis became apparent!
6. The dysarthria and dysphagia were fatiguable rather than diurnal.
7. CT chest was done and no evidence of thymoma was found.
8. After a prolonged period of ventilatory support the patient was weaned, tracheostomy was closed, and hospital discharge followed. He is at home on tapering immunosuppression, independent in activities of daily living and enjoying gardening again!
Statin associated weakness in Myasthenia Gravis: A case presentation.

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Dr Juel

We thank Dr Juel for his helpful comments.

We agree with Dr Juel that it is not possible to prove a direct effect on the myasthenic neuromuscular transmission defect by the Atorvastatin in this case. This is at least in part because there clearly was evidence prior to the onset of the myasthenia of statin-induced myopathy due to Simvastatin and therefore it seems irrefutable that the Atorvastatin caused a similar statin myopathy when commenced after the onset of myasthenia.

As such to demonstrate, in addition to the myotoxic component of the Atorvastatin, an additional detrimental effect on an already defective neuromuscular junction would have been a formidable challenge.

Our aim in presenting this case was primarily to report our observation that a myasthenic patient misdiagnosed with a brainstem stroke and prescribed statins became weaker and started to improve only when both his myasthenia and statin myopathy were recognised and treated appropriately. Secondly, we wished to alert clinicians to what at the very least is a functional interaction between myasthenia gravis and statins – both of which can make a patient weak. Finally we invite others to consider the possible mechanisms whereby statins, myasthenia, and indeed other autoimmune diseases may interact at a more complex level.

We are very grateful for Dr Juel’s comments and have adjusted the text to clarify these points.