Reviewer's report

Title: Statin associated Myasthenia Gravis: A case report.

Version: 3 Date: 6 November 2008

Reviewer: Vern Juel

Which of the following best describes what type of case report this is?: Unreported or unusual side effects or adverse interactions involving medications

Has the case been reported coherently?: Yes

Is the case report authentic?: Yes

Is the case report ethical?: Yes

Is there any missing information that you think must be added before publication?: Yes

Is this case worth reporting?: No

Is the case report persuasive?: No

Does the case report have explanatory value?: No

Does the case report have diagnostic value?: No

Will the case report make a difference to clinical practice?: No

Is the anonymity of the patient protected?: Yes

Comments to authors:

This is an interesting case of a patient with autoimmune myasthenia gravis and cholesterol-lowering agent myopathy occurring in parallel. Although both of these disorders producing skeletal muscle weakness were coincident, there is not convincing evidence that the cholesterol-lowering agent exposure caused or necessarily exacerbated the neuromuscular junctional disorder.

The initial exposure to simvastatin was associated with classic symptoms of cholesterol lowering agent myopathy with myalgias and proximal muscle weakness that resolved upon withdrawal of the medication. Painless dysarthria and dysphagia apparently began three months later as the initial symptoms of myasthenia gravis. Subsequent exposure to atorvastatin was associated with CK elevation that resolved upon cessation, and nearly parallel treatment with
prednisolone was followed by increased weakness in a distribution likely owing to myasthenia gravis (e.g. including extraocular muscles) that suggests a corticosteroid-related myasthenic exacerbation. Although the authors attribute the overall clinical improvement to the cessation of atorvastatin, the timing of improvement is also compatible with a response of the myasthenia gravis to corticosteroids and to IVIG infusions.

The case reported by Cartwright et al (Neurology 2004;63:2188) presents a more convincing case for HMG-CoA reductase inhibitors exacerbating myasthenia gravis (instead of causing a parallel cholesterol-lowering agent myopathy) due to the lack of associated CK elevation, the absence of electromyographic findings of myopathy, and clear demonstration of abnormal neuromuscular transmission with repetitive nerve stimulation studies.

Quality of written English: Acceptable

Declaration of competing interests:

I declare that I have no competing interests.