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Case report

Effect of statin therapy on undiagnosed case of Myasthenia gravis

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Abstract

Myasthenia gravis is a rare autoimmune disease. It is undiagnosed most of the time in the older age groups; we report a case of 55 years old male presented with bilateral ptosis, diplopia and muscle weakness mainly in the evening after taking statin therapy. He was not previously diagnosed with myasthenia gravis.

Keywords: myasthenia gravis**,** autoimmune disorder**,** statin therapy.

1. Introduction

Myasthenia gravis is an autoimmune disorder mainly caused by autoantibodies to the muscle acetylcholine receptors (AChRs) at the neuromuscular junction. Loss of these receptors leads to a defect in neuromuscular transmission with muscle weakness and fatigue[1].
The estimated prevalence of myasthenia gravis is approximately 20 cases per 100,000 populations, with the disease affecting twice as many women as men. However, in older age group, men are affected more often and the disease is often misdiagnosed[2].

Many factors influence cholinergic transmission, including drugs, temperature and emotional state. The adverse effects of many medications may provoke exacerbations[3]. Statin may cause worsening of myasthenia gravis without regard to type of myasthenia or brand of statin. Worsening of weakness can occur independent of myalgic syndrome and usually involves oculobulbar symptoms within 1-6 weeks of the initiation of statin treatment[4].

2. Case Report

A 60 years old hypertensive and diabetic male presented in emergency department of Capital Development Authority Hospital, Islamabad, Pakistan on December 29th 2013 with the chief complaint of bilateral ptosis with diplopia, which was progressive in nature; muscle weakness and fatigueability mainly in the evening. He was having difficulty in swallowing. He visited general practitioner two weeks ago for high fasting lipid profile for which he was prescribed simvastatin. After taking simvastatin he developed all the symptoms mentioned above. His past medical, surgical, family, socioeconomic, and allergic history were unremarkable. He was on ACE inhibitors and Glimepiride for the last one year. His pulse rate was 80/min, blood pressure 120/70, respiratory rate 14/min, bilateral ptosis and pupils were reactive to light. Systemic examination was unremarkable.

Specific Laboratory studies revealed raised Acetylcholinesterase antibodies titre. Electromyography (EMG) test was positive which showed muscle fatigueability, decremental response in trapezius muscle; sensory and motor system was intact. Tensilon test was positive.
Other laboratory investigations include blood complete picture, liver function test, renal function test, blood sugar random, serum aldolase level, x-ray chest, computed tomography chest and brain were within normal limits. Ultrasound showed fatty liver. Serum cholesterol and low density lipid were on higher side.

The diagnosis of myasthenia gravis was made which was unmasked by statin therapy. Statin therapy was stopped and patient was started on Pyridostigmine Bromide (acetylcholineesterase inhibitor). He was monitored for his symptoms and his condition was improved within one week.

Fig 1: Bilateral ptosis and facial muscle weakness

(Picture was taken after consent)

3. Discussion

Myasthenia gravis is an autoimmune *channelopathies*. There is no known causative pathogen that could account for myasthenia. There is a slight genetic predisposition, particularly HLA type; upto 75% of the patient have an abnormality of the thymus, 10% have thymoma[5]. Drug induced myasthenic syndrome are characterized by progressive and typically symmetric muscle weakness. The most common manifestations are ptosis, diplopia, dysphagia, dysarthria as well as weakness of limbs and respiratory muscles. The clinical pattern varies with different drugs; and not all of the symptoms are present in individual cases[6]. In our case, patient presented with bilateral ptosis and fatigueability. Signs and Symptoms of the myasthenic syndrome can appear days to months after initiation of the offensive drug.[7] In our case symptoms appeared after two weeks of initiation of therapy.

Statin therapy does not cause myasthenia gravis but it aggravates the symptoms of the disease.[4,8] In our case patient did not have any symptoms of myasthenia but after taking statin, his silent and undiagnosed disease appeared with severe form. In literature very less data is available regarding statin associated myasthenia gravis and its exacerbations. [9,10] One possible mechanism of statins on myasthenia gravis patients is believed to be mediated by immune system. [4]

Myasthenia gravis can be a difficult diagnosis as the symptoms can be subtle and have to be distinguished from both normal variant and neurological disorder. Many different types of investigations are available. Electromyography (EMG) is most sensitive test and Acetylcholine receptor antibodies test has 80-96% sensitivity.[11] We ruled out all other causes which precipitate myasthenia gravis and made diagnosis of myasthenia gravis which was unmasked by statin therapy. Myasthenia gravis in older age group is not associated with thymoma. It is mainly associated with autoimmune disease.[12] Myaesthenia gravis is one of the most treatable neurological disorders. Pharmacological therapy includes anticholinestrase medication and immunosuppressive agents. Other treatment options are plasmapheresis and thymectomy.[3,13]

4. Conclusion

Aim of this case report is to raise awareness among clinicians about the effect of stating on myasthenia gravis. Physicians should have high index of suspicion about Myasthenia Gravis in any patient presenting with fatigue and weakness after taking statin therapy. In older age group, myasthenia gravis should be differentiated from other central nervous system lesions.

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