**Case Details**

A 24-year-old gentleman presented with bilateral eye ptosis, fatigable generalized weakness, and dyspnea on exertion 3 years back. Ice-pack test was positive, repetitive nerve stimulation showed a decremental response, and the anti-acetylcholine (Ach) receptor antibody titer was 10.32 nmol/L (normal: <0.50 nmol/L). Anti-MuSK antibody was not tested. He was diagnosed with generalized myasthenia gravis (MG) and started on pyridostigmine and prednisolone, resulting in clinical improvement. Computed tomography of the chest showed no thymic abnormality. Unfortunately, he had used the prescribed medicines only for a year before defaulting and came to us with a recurrence of symptoms. On examination, he had bilateral external ophthalmoparesis, ptosis [Video 1], and fatigable weakness, but no diplopia. Thyroid function tests were normal. His ptosis and weakness improved with pyridostigmine and prednisolone, but the ophthalmoparesis did not. The magnetic resonance imaging of the orbits showed severe thinning of all the extraocular muscles [Figure 1].

**Discussion**

Extraocular muscle atrophy in MG was considered rare, with only a few case reports describing this feature until recently.[1-3] However, three recent studies have revealed that fat replacement and atrophy are not so uncommon in these muscles.[4-6] MG related to anti-MuSK antibodies is known to cause atrophy of the tongue and facial muscles. However, extraocular involvement was not extensively reported.[1] Moreover, the atrophy of these muscles was considered rare in those with anti-Ach receptor antibodies.[1-3] We described a case of anti-Ach receptor antibody-positive MG with severe atrophy of all the extraocular muscles.

Lueangaram *et al.*[4] showed a statistically significant difference in the muscle volumes of an MG group compared to a control group. Moreover, the atrophy of extraocular muscles in MG was more in chronic cases, although it occurred early in some. A similar result was reported by Velonakis *et al.*[6] who included 19 patients with either generalized or ocular MG, mostly with anti-Ach receptor antibody positivity. All the patients included in this study had a chronic disease, and 14 of them were on steroids for a long duration. This group reported muscle atrophy and fat replacement in approximately one-third and one-fourth of the MG patients, respectively. In contrast, another study done by Keene *et al.*[5] found increased mean muscle volume in 11 MG patients (10 of them had anti-Ach antibodies), though there was also an increase in fat fractions when compared with healthy controls. However, 45% of the patients in this study had a disease duration of fewer than 3 months, which might explain the results. Thus, there appears to be early inflammation in extraocular muscles in MG, resulting in edema, which might lead to atrophy later on.

The pathophysiology of muscle atrophy in such cases is not fully understood. One possible mechanism is the persistent inflammation leading to the damage of motor endplates, resulting in the denervation and fibrosis of muscles.[1,4,6] The ocular motility might not improve with treatment in such instances.[3] Our patient also had a persistent limitation of the extraocular movements, which was unresponsive to the treatment. Therefore, it is important to consider the possibility of the atrophy of extraocular muscles if there is no clinical improvement after adequate treatment, which may prevent unwarranted escalation of immunotherapy.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initial will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

**Conflicts of interest**

There are no conflicts of interest.
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