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CASE REPORT

OCULAR MYASTHENIA GRAVIS IN A YOUNG ADULT: A RARE CASE WITH FAVORABLE OUTCOME

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ABSTRACT

Myasthenia gravis (MG) is an autoimmune disorder characterized by fluctuating weakness and fatigability of skeletal muscles. Ocular myasthenia gravis (OMG) presents with symptoms confined to the ocular muscles. A 21-year-old male presented with a two-month history of bilateral ptosis, which improved in the morning or after a nap but worsened throughout the day. Ocular examination revealed bilateral ptosis with gaze restriction. Visual acuity, anterior segment, visual fields, slit-lamp, and fundus examinations were unremarkable. The ice pack and rest tests showed marked improvement, raising suspicion for MG. The acetylcholine receptor (AChR) antibody test was positive for AChR binding antibodies. The patient was initiated on pyridostigmine, an acetylcholinesterase inhibitor, and corticosteroids. Ptosis and ophthalmoplegia improved at the one-week follow-up and completely resolved after eight weeks. The hallmark features of MG are fluctuating, fatigable muscle weakness that worsens with activity and improves with rest. A thorough clinical examination, along with diagnostic tests such as electrophysiological studies and antibody tests, is essential for confirming the diagnosis. Ocular myasthenia gravis has a high likelihood of progressing to generalized myasthenia gravis (GMG) within two years. Early diagnosis and prompt treatment are critical to prevent or mitigate the risk of myasthenic crisis and long-term complications.

***Keywords**: Ocular myasthenia gravis, ptosis, acetylcholine receptor antibody*

INTRODUCTION

Myasthenia gravis (MG) is an autoimmune disorder affecting the postsynaptic nicotinic acetylcholine receptors at neuromuscular junctions of striated muscle. Ocular symptoms are the most common initial presentation.[1,2] MG can affect individuals of all ethnicities and ages but is often referred to as 'a disease of young women and elderly men,' with peak onset in women between ages 20–39 and in men between 50–70.[9] Epidemiological studies suggest a slightly higher prevalence among individuals of African descent, particularly in MuSK-positive cases. Despite numerous studies, incidence and prevalence rates vary widely, with recent estimates ranging from 1.5 to 17.9 per 100,000 population.[7,13]symptoms such as diplopia The diagnosis of MG is often based on ocula and ptosis, which occur in approximately two-thirds of patients. These symptoms typically fluctuate—improving with rest, sleep, or cold exposure, and worsening with sustained muscle activity due to fatigability. Among those with initial ocular involvement, up to 80% progress to generalized myasthenia gravis (GMG) within two years.[3,6] While cholinesterase inhibitors provide symptomatic relief, corticosteroids and immunosuppressants such as azathioprine are more effective for long-term control of ocular myasthenia gravis (OMG) and in preventing its progression to GMG.[6,11]

This study aims to evaluate the clinical progression and treatment efficacy in myasthenia gravis. The findings are intended to support improved clinical management and optimize patient outcomes.

CASE REPORT

A 21-year-old man presented with bilateral eyelid ptosis persisting for over two months. The ptosis improved in the morning or after napping and worsened by evening (Fig. 1).

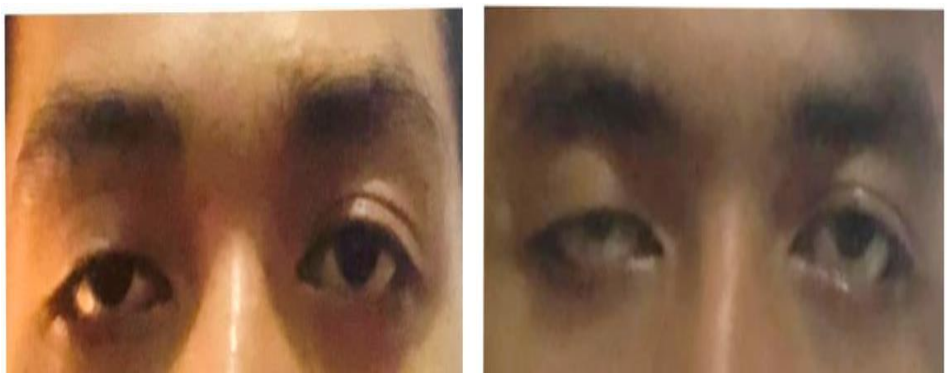

Figure 1. Clinical photograph of the patient at the initial visit, showing bilateral ptosis upon waking in the morning and worsening in the evening

The patient had intermittent double vision, which disappeared when one eye was closed. No other symptoms or relevant medical history were noted. General and neurological exams were normal. Vision was 5/5 in both eyes, eye pressure was 13 mmHg, and Hirschberg test showed straight eye alignment.

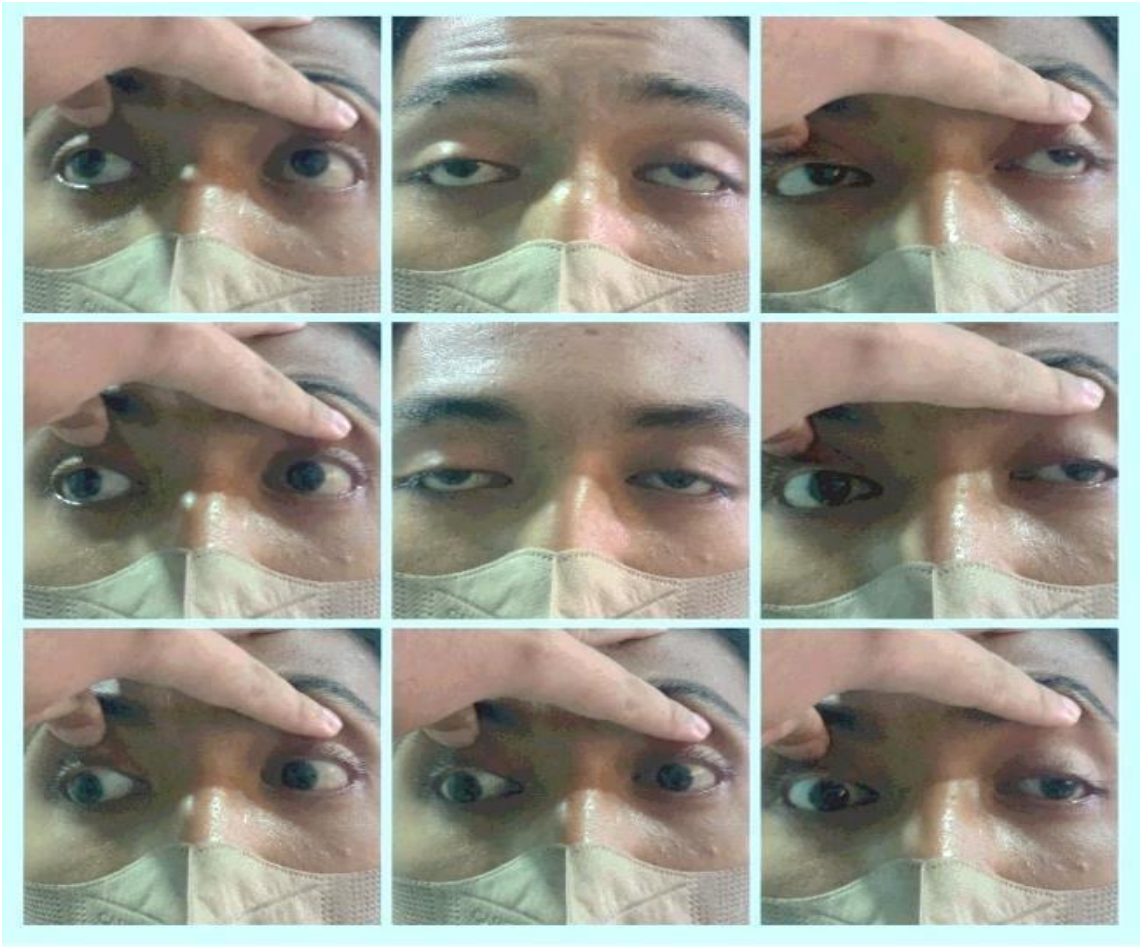

Figure. 2 Ocular motility showed marked restriction in the right eye: -4 in medial and inferomedial gazes, and -3 in inferior, inferolateral, lateral, superolateral, superior, and superomedial directions. The left eye exhibited -2 limitation in superior, superolateral, lateral, and inferolateral gazes, and -3 in inferior, inferomedial, medial, and superomedial directions

The right eye demonstrated ptosis with a margin reflex distance 1 (MRD1) of -1 mm, interpalpebral fissure (IPF) width of 6 mm, and levator function (LF) of 8 mm. The left eye also showed ptosis with MRD1 of 0 mm, IPF width of 6 mm, and LF of 9 mm (Fig. 3).

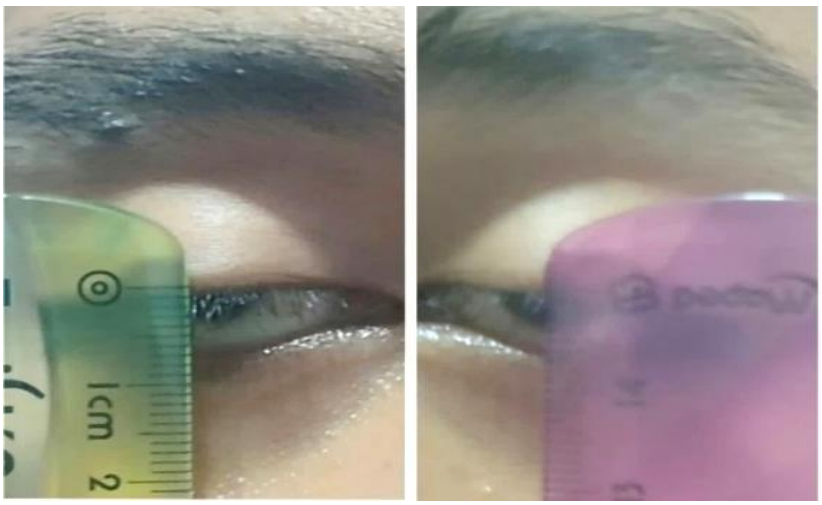

Figure 3. Ptosis examination of the patient, showing bilateral eyelid drooping

Further evaluations included the sleep test, ice-pack test, and Cogan's lid twitch (CLT). Following 30 minutes of rest (Fig. 4A), the right interpalpebral fissure (IPF) improved from 6 mm to 8 mm (MRD1: 2 mm), and the left IPF increased from 6 mm to 9 mm (MRD1: 2 mm), indicating a positive sleep test response.

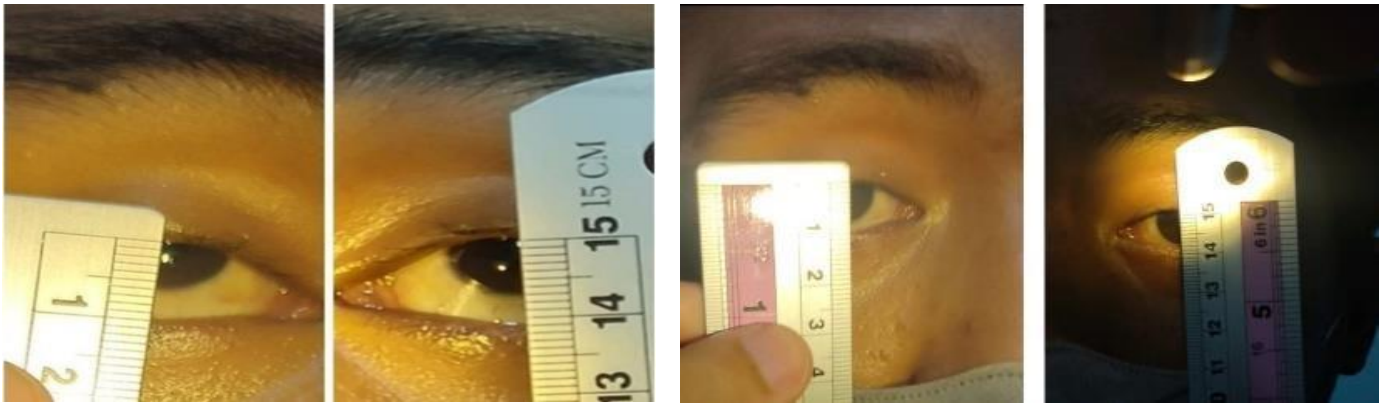

Figure 4. (A) Improvement in ptosis observed after 30 minutes of rest. **(B)** Improvement in eyelid position following a 2-minute ice-pack test

The ice-pack test result (Fig. 4B) demonstrated improvement in ptosis. Following a 2-minute application, the right interpalpebral fissure (IPF) increased from 6 mm to 10 mm (MRD1: 2 mm), and the left IPF also improved from 6 mm to 10 mm (MRD1: 2 mm).

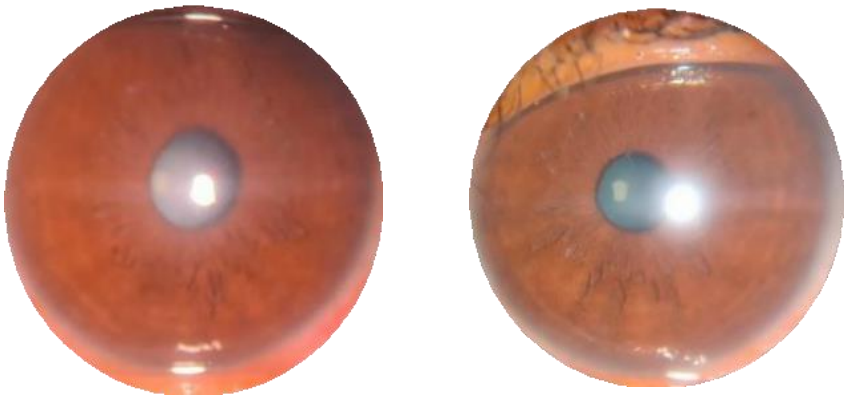

Figure 5. Anterior segment examination of both eyes, within normal limits

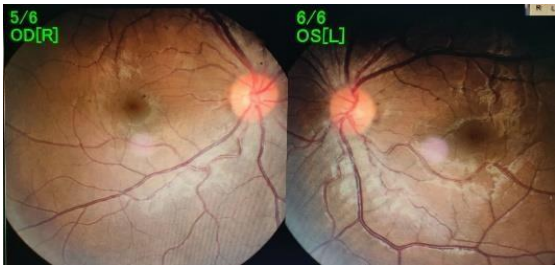

Figure 6. Funduscopic photograph within normal limit

Slit-lamp biomicroscopy of the anterior segment was unremarkable in both eyes (Fig. 5). Pupils were round, 3 mm in diameter, with positive light reflexes and no relative afferent pupillary defect. Posterior segment examination was also within normal limits in both eyes (Fig. 6).

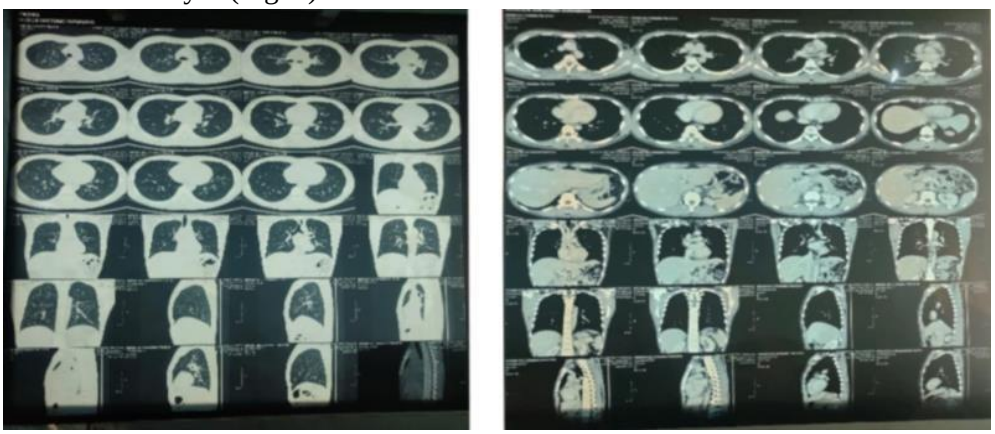

Figure 7. Thoracic CT scan revealed no evidence of thymic enlargement

Ancillary tests showed a 39% decrement on RNS-EMG of the left orbicularis oculi. Thoracic CT (Fig. 7) revealed no thymic enlargement. AChR antibody was positive at 3.93 (normal <0.4).

Table 1. Blood laboratory result

Parameters	Result	Reference Value
Hb	14.6	13,3 – 16,6 g/dL
WBC	6.15 x 10^3	3,37 – 10 (x10^3 / µL)
PLT	266 x 10^3	150 – 450 (x10^3 / µL)
Neut	45.4	39,8 – 70,5 %
Lymph	44.7	23,1 – 49,9 %
GDP	81	100 – 126 mg / dL
GD2JPP	90	< 140 mg / dL
HbA1C	4.9	< 5,7 %
SGOT / SGPT	19 / 15	0 -50 U / L
BUN / SK	12.4 / 1.1	7 -18 mg/dL / 0,6-1,3 mg/dL
K / Na / Cl	4.1 / 139 / 103	3,5-5,1 / 136-145 / 98 -107 mmol/L
Cholesterol	145	0 - 200 mg/dL
TG	101	30 – 150 mg/dL
HDL	45	40 – 60 mg/dL
LDL	87	0 – 99 mg/dL
PPT / APTT	10.5 / 30.3	9-12 / 23-33 seconds
BSR	25	0-15 mm/h

* Blood laboratory result revealed increased of blood sediment rate.

After one week of treatment, the ptosis improved (Fig.8). Both upper eyelidscould open wider and double vision only occur during evening. The limited motility of both eyes were also improved (Fig.9).

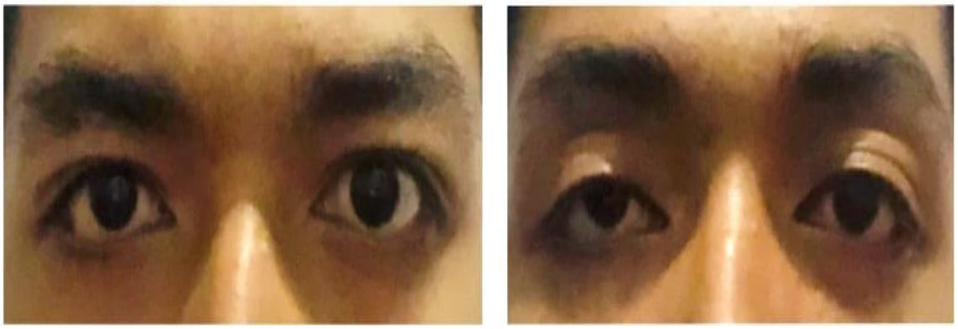

Figure 8. Clinical picture after one-week treatment

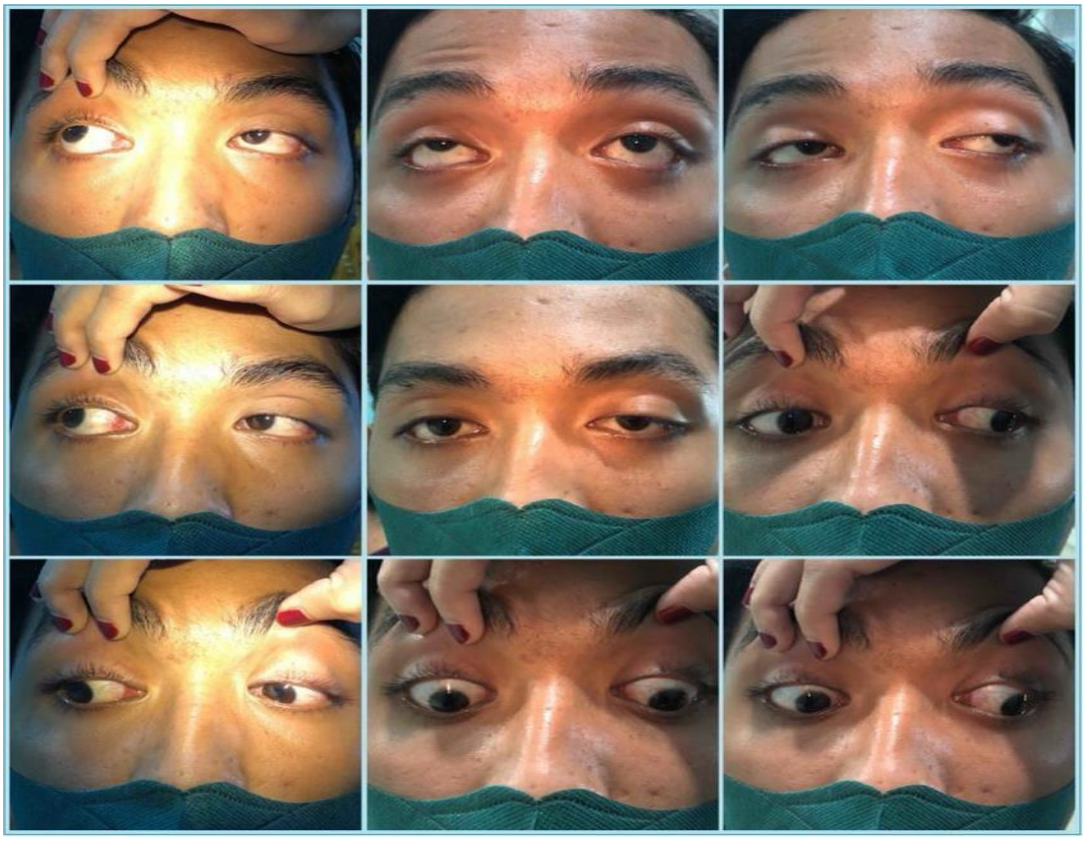

Figure 9. Clinical picture after one week of treatment, showing significant improvement of ocular motility

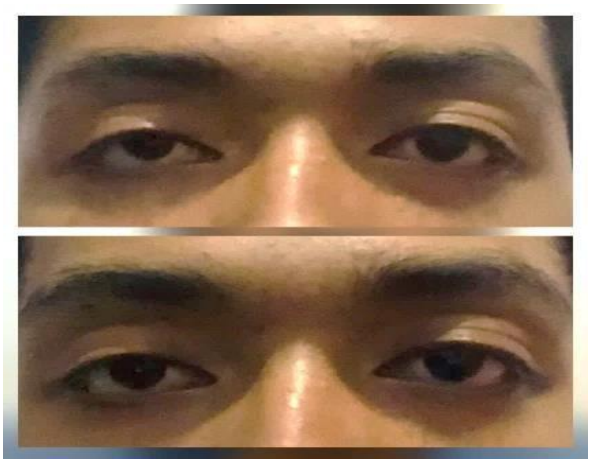

Figure 10. Clinical picture after four weeks of treatment, showing further improvement in ptosis

The right eye demonstrated a -2 limitation in all directions of gaze, while the left eye showed a -1 limitation. Examination of the right eye revealed an MRD1 of 1 mm, interpalpebral fissure width of 8 mm, and levator function of 9 mm. The left eye showed an MRD1 of 2 mm, IPF width of 9 mm, and LF of 10 mm. Thyroid function tests were within normal limits (Table 2).

Table 2. Thyoid function examination

Parameters	Result
fT4	1.19
Total T3	1.3
C3/C4	113/27
TSH	2.86

***** *Thyoid function examination within normal limit ocular motility*

The right eye demonstrated a -2 limitation in all directions of gaze, while the left eye showed a -1 limitation. Examination of the right eye revealed an MRD1 of 1 mm, interpalpebral fissure width of 8 mm, and levator function of 9 mm. The left eye showed an MRD1 of 2 mm, IPF width of 9 mm, and LF of 10 mm. Thyroid function tests were within normal limits (Table 2).

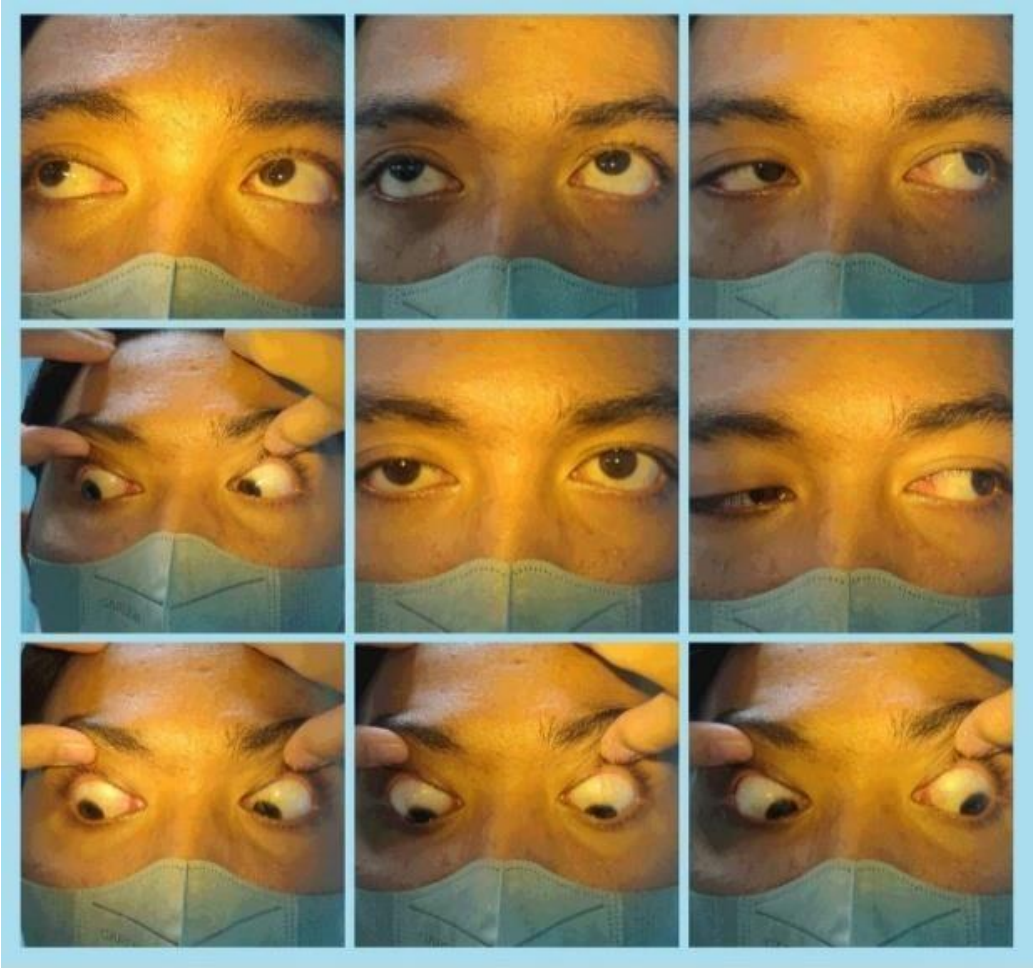

Figure 11. After 4 weeks of treatment, right eye had -1 limitation in lateral gaze; left eye had -1 in medial gaze

At the 4-week follow-up, ptosis had improved significantly (Fig. 10), with both upper eyelids opening normally and no reported diplopia. Ocular motility also showed improvement (Fig. 11), with the right eye demonstrating a -1 limitation in lateral gaze and the left eye a -1 limitation in medial gaze. Examination of the right eye revealed an MRD1 of 2 mm, interpalpebral fissure (IPF) width of 9 mm, and levator function (LF) of 13 mm. The left eye showed an MRD1 of 2 mm, IPF width of 10 mm, and LF of 14 mm.

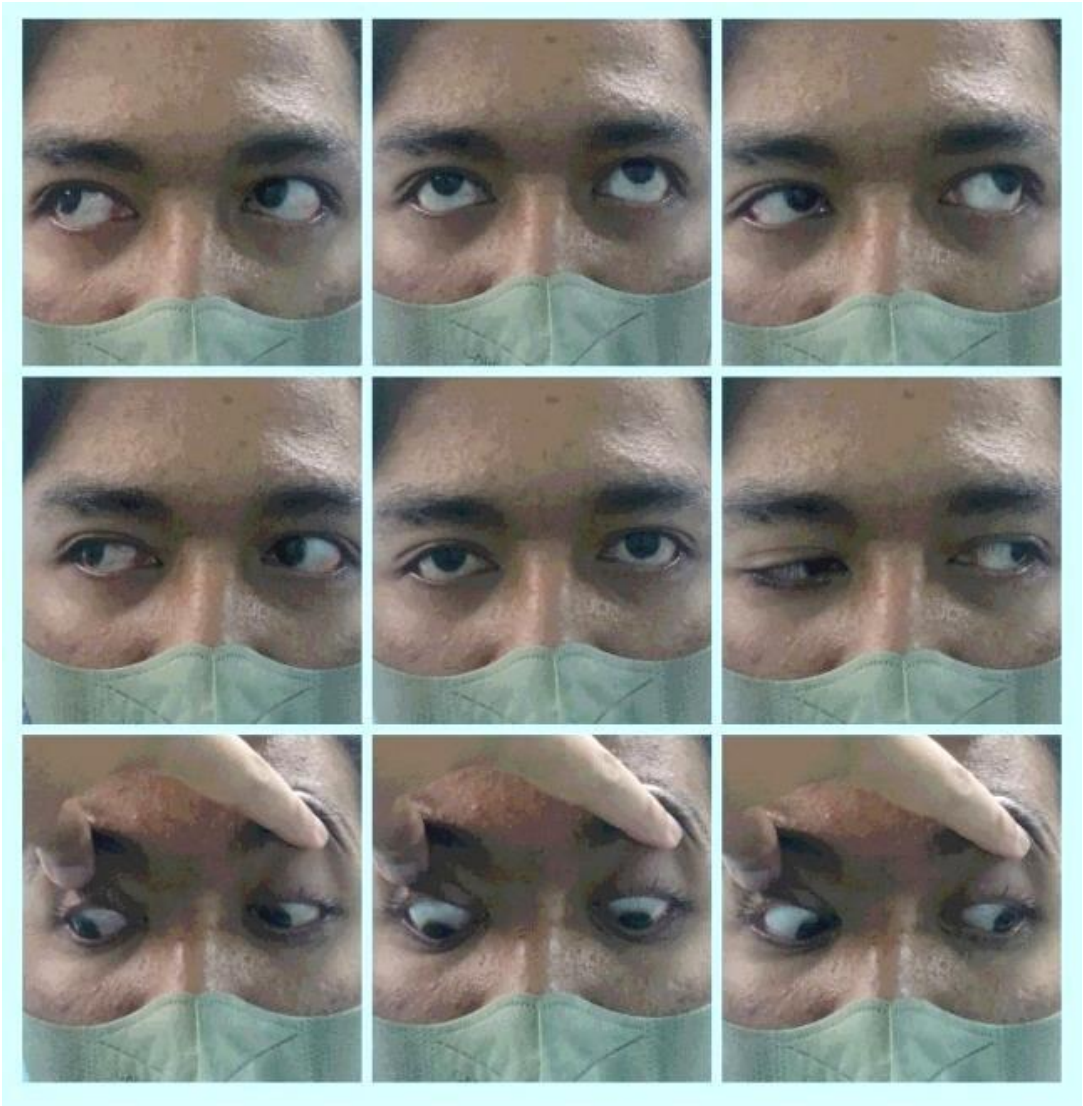

Figure 12. Clinical picture after eight weeks of treatment, no evidence of ptosis and no limitations observed in any gaze direction

Due to the satisfactory response, Methylprednisolone was tapered to 32 mg once daily, while Pyridostigmine 60 mg three times daily and neurotropic agents twice daily were continued. At the 8-week follow-up, the patient reported no ptosis or diplopia. Examination revealed MRD1 of 3 mm, interpalpebral fissure (IPF) width of 10 mm, and levator function (LF) of 14 mm in both eyes. Extraocular movements were full in all directions without limitation (Fig. 12).

DISCUSSION

Myasthenia gravis is an autoimmune disorder characterized by fluctuating and fatiguing muscle weakness. Although it is typically a systemic disease, approximately 50% of patients initially present with ocular signs and symptoms. In some cases, ptosis or diplopia may be the sole presenting features. Myasthenia gravis can be classified into ocular, bulbar, or generalized forms, with each subtype potentially involving cranial nerves originating from the lower brainstem. Notably, in up to 90% of cases, ocular myasthenia gravis progresses to the generalized form within the first two years following the onset of ocular symptoms[1,13].

The pathophysiology of myasthenia gravis involves autoantibodies that reduce the number of functional nicotinic acetylcholine receptors (AChRs) at the neuromuscular junction. This autoimmune response, primarily mediated by humoral mechanisms, leads to receptor blockade, accelerated receptor degradation, and complement-mediated destruction. The resulting impairment in neuromuscular transmission ultimately causes muscle weakness and paralysis[1,5].

Diplopia and ptosis are common in myasthenia gravis, often appearing during the disease course. Up to 80% of patients with ocular onset develop generalized symptoms within two years. A Mayo Clinic study found that 51% presented with ocular symptoms, and over half progressed to generalized disease. Bulbar involvement may cause dysarthria, dysphagia, and facial weakness. Neck flexion weakness is more common than extension, and about 10% may develop head drop, especially older male patients.

The hallmark features of myasthenia gravis are fluctuating weakness and fatigability. Ptosis, either unilateral or bilateral, is the most common clinical sign. It often begins in one eye and may later involve the other, typically presenting asymmetrically. Ptosis may worsen following sustained upgaze, a finding known as the 'lid fatigability test.' Another associated sign is Cogan’s lid twitch, although it is not specific to ocular myasthenia gravis.

Orbicularis oculi weakness can be assessed by asking the patient to forcibly close the eyes while the examiner attempts to open them; the gradual separation of the eyelids, revealing the sclera, is referred to as the 'peek sign.' Extraocular muscles are particularly vulnerable due to their lower density of acetylcholine receptors, making them more susceptible to receptor loss and immune-mediated damage[1,13].

In patients with evident clinical signs, the diagnosis of myasthenia gravis can be supported by the sleep test, ice-pack test, or edrophonium chloride test. The sleep test is a safe and simple alternative, with symptom improvement after 30 minutes of eye closure strongly suggesting myasthenia gravis [1,2,3,4].

The ice-pack test is a simple, low-cost, and risk-free bedside tool for differentiating myasthenia gravis from other conditions. In this case, it yielded a positive result. A recent study on myasthenic diplopia reported a sensitivity of 76.9% and specificity of 98.3% for a 5-minute application, with no false positives observed[21]. Electromyography (EMG) is useful in confirming myasthenia gravis, especially in seronegative cases. Repetitive nerve stimulation (RNS), a component of EMG, assesses changes in motor unit potentials following repeated stimulation of facial or proximal nerves. In MG, a characteristic decrement of >10% in compound muscle action potential amplitude is typically observed by the fourth or fifth response. While RNS has high specificity (~90%) for ocular MG, its sensitivity remains modest (18–35%)[2,6,7,8].

Anticholinesterase medication is the first-line treatment for myasthenia gravis, with pyridostigmine commonly administered at 60 mg every 2 to 4 hours in adults. While generally well tolerated, only about 50% of patients with ocular symptoms respond adequately, with ptosis showing better improvement than diplopia[6,13].

With current therapies, most myasthenia gravis patients have a near-normal life expectancy. Myasthenic crisis mortality has dropped to around 4.5%. Morbidity mainly stems from drug side effects and aspiration due to muscle weakness[26].

CONCLUSION

Ocular myasthenia gravis (OMG) progresses to the generalized form in approximately 90% of cases within the first two years after symptom onset. A thorough neurological examination and detailed clinical history are essential for establishing the diagnosis. Bedside tests, electrophysiological studies, and serological antibody testing serve as supportive diagnostic tools. Early recognition and timely initiation of treatment are crucial to managing the disease and may help prevent or reduce the risk of myasthenic crisis.

DECLARATIONS

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no conflict of interest.

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