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Generalized Myasthenia Gravis Identified During Evaluation for Streptococcal Pharyngitis: A Case Report

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Abstract

Myasthenia gravis is an autoimmune disorder of the neuromuscular junction characterized by fluctuating skeletal muscle weakness that worsens with exertion and improves with rest. Ocular manifestations, including ptosis and diplopia, are common early features, but generalized disease may also involve bulbar, limb, axial, or respiratory muscles. Because symptoms may be intermittent or occur alongside common acute illnesses, first-contact clinicians must avoid prematurely attributing fatigue or malaise to a concurrent infection when focal fatigable weakness is present.

We present a case of a 50-year-old woman with hypertension, obesity, postmenopausal bleeding, and 12 lb of unintentional weight loss over three months who presented to an outpatient clinic with upper respiratory symptoms and generalized fatigue. Rapid antigen testing was positive for group A Streptococcus, and antibiotic therapy with amoxicillin was initiated. During the same evaluation, she reported several weeks of progressive right eyelid drooping, intermittent diplopia, worsening fatigue later in the day, evening gait difficulty, and slurred speech that became more noticeable during prolonged conversations. Examination demonstrated right-sided ptosis, preserved pupillary responses, and ocular misalignment worsened by sustained upward gaze. The ice-pack test improved right margin reflex distance 1 from 1 mm to 4 mm after two minutes of cooling. Given the combination of ocular, bulbar, and limb fatigability, the presentation was classified as generalized myasthenia gravis, Myasthenia Gravis Foundation of America class IIb. Acetylcholine receptor binding antibody was elevated at 8.6 nmol/L, repetitive nerve stimulation demonstrated a 17% decrement in compound muscle action potential amplitude with 3-Hz stimulation of the right facial nerve recording from the nasalis, and chest computed tomography showed no thymoma. Pyridostigmine was initiated at 30 mg orally three times daily and later increased to 60 mg three times daily as tolerated, with improvement in ptosis and dysarthria at two-week follow-up.

This case highlights the importance of recognizing generalized myasthenia gravis when fatigable ptosis, diplopia, dysarthria, and end-of-day weakness coexist with a common acute infection. Streptococcal pharyngitis explained the patient's upper respiratory symptoms but did not explain the pattern of fluctuating ocular and bulbar weakness. Bedside fatigability testing, careful differential diagnosis, medication review, and urgent neurology referral are essential because infection can precipitate clinical worsening and myasthenic crisis.

Categories: Family/General Practice, Neurology
Keywords: diplopia, fatigability, ice-pack test, myasthenia gravis, neuromuscular junction, ocular myasthenia, ptosis, streptococcal pharyngitis

Introduction

Myasthenia gravis is a chronic autoimmune disorder of the neuromuscular junction characterized by fluctuating skeletal muscle weakness. The most common immunologic mechanism involves autoantibodies directed against the postsynaptic acetylcholine receptor, although antibodies against muscle-specific kinase, low-density lipoprotein receptor-related protein 4, and other neuromuscular junction targets may be implicated in selected patients [1,2]. Because neuromuscular transmission is impaired, symptoms characteristically worsen with repeated activity and improve with rest.

Myasthenia gravis is uncommon but clinically important because delayed recognition can lead to respiratory failure, aspiration, or myasthenic crisis. Epidemiologic studies demonstrate age- and sex-related patterns, with earlier-onset disease more common in women and later-onset disease increasingly recognized around and beyond the fifth decade of life [3,4]. A 50-year-old woman therefore falls near the clinically important transition between early- and late-onset groups, where clinicians must consider both autoimmune neuromuscular disease and age-appropriate malignancy evaluation when constitutional or gynecologic red flags are present.

The clinical presentation is variable. Ocular symptoms such as ptosis and diplopia are common early manifestations, but weakness may generalize to involve bulbar, limb, axial, and respiratory muscles [1,2]. Bulbar symptoms, including dysarthria, dysphagia, chewing fatigue, or voice change with prolonged speaking, are particularly important because they suggest disease beyond isolated ocular myasthenia and increase the urgency of neurologic evaluation.

Patients with myasthenia gravis may first present to first-contact clinicians with unrelated or competing complaints, including musculoskeletal symptoms, constitutional symptoms, or acute infection [5]. Concurrent infection may worsen fatigue, lower the safety margin of neuromuscular transmission, and precipitate clinical deterioration or myasthenic crisis [6,7]. Diagnosis is supported by clinical examination, bedside fatigability testing, serologic testing, electrophysiologic confirmation, and chest imaging to assess for thymoma or thymic hyperplasia [2,8,9]. Bedside maneuvers such as the ice-pack test and sustained upward gaze testing may provide early clues when confirmatory testing is not immediately available [10].

The clinical significance of this case lies in the diagnostic challenge of recognizing a potentially serious neuromuscular disorder during an encounter for a confirmed common infection. Although acute infections frequently explain constitutional symptoms and generalized fatigue, they do not necessarily account for concurrent focal or activity-dependent weakness. This distinction is particularly relevant to primary care and other first-contact settings, where premature diagnostic closure may delay recognition of neuromuscular disease. By emphasizing the importance of identifying symptom patterns that remain unexplained by an established diagnosis, this case illustrates a practical approach to clinical reasoning that may facilitate earlier recognition, appropriate neurologic evaluation, and prevention of potentially life-threatening complications.

Case Presentation

In May 2025, a 50-year-old woman with a medical history of hypertension, obesity, and postmenopausal bleeding presented to an outpatient clinic with unintentional weight loss, generalized fatigue, and upper respiratory symptoms. Rapid antigen testing was positive for group A Streptococcus, and antibiotic therapy was initiated with amoxicillin 500 mg orally twice daily for 10 days. During the same evaluation, she reported several weeks of progressive right eyelid drooping, intermittent double vision, fatigue that worsened as the day progressed, difficulty with eye movements, trouble walking in the evening, and slurred speech that became more noticeable during prolonged conversations. She denied dyspnea, dysphagia, choking, headache, neck pain, jaw claudication, acute visual loss, sensory loss, autonomic symptoms, or fixed limb weakness.

On physical examination, the patient exhibited right-sided ptosis. Pupils were equal, round, and reactive to light, with no anisocoria, helping reduce concern for compressive third-nerve palsy or Horner syndrome. Extraocular examination showed intermittent right hypertropia and variable esotropia, worse with sustained upward gaze and right lateral gaze. Sustained upward gaze for 60 seconds provoked worsening ocular misalignment and fatigability. The ice-pack test was performed for two minutes and improved right margin reflex distance 1 from 1 mm to 4 mm, corresponding to a 3-mm improvement. Speech became nasal and mildly dysarthric after counting aloud to 50, without choking or overt aspiration. Proximal limb strength was initially 5/5, but right deltoid strength fatigued to 4+/5 after repeated arm abduction. Single-breath count was 35, oxygen saturation was 98% on room air, and there was no accessory muscle use. Sensation, coordination, reflexes, and gait were otherwise intact.

The presentation was notable because the positive rapid streptococcal antigen test explained the acute upper respiratory symptoms but did not explain the several-week history of fluctuating ptosis, intermittent diplopia, fatigable dysarthria, and end-of-day functional worsening. Given ocular, bulbar, and limb symptoms, the presentation was clinically consistent with generalized myasthenia gravis rather than isolated ocular myasthenia. Based on fatigable dysarthria and evening gait difficulty without documented respiratory failure, the clinical severity was classified as Myasthenia Gravis Foundation of America class IIb. The diagnostic approach to suspected myasthenia gravis is summarized in Figure *1*.

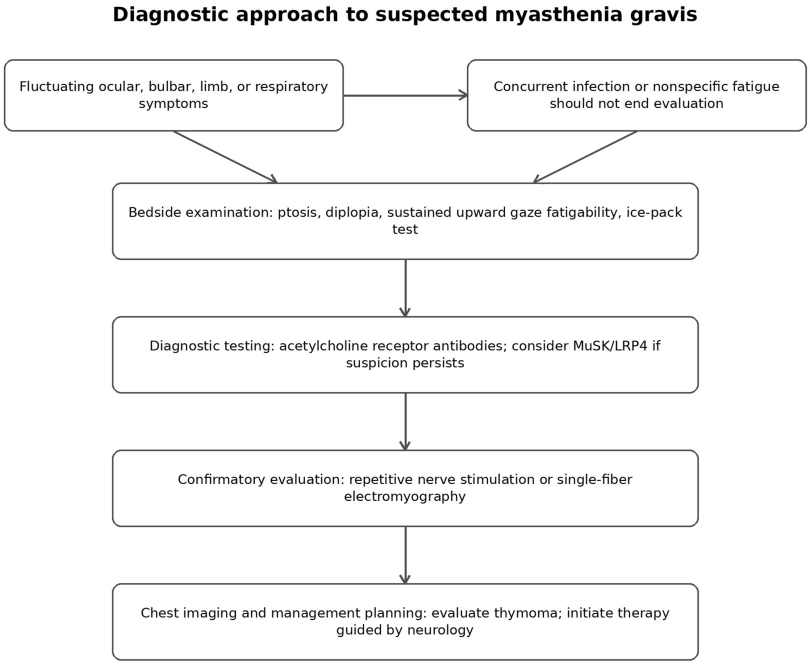


FIGURE 1: Diagnostic approach to suspected myasthenia gravis.

This original diagnostic algorithm summarizes the clinical pathway for suspected myasthenia gravis, beginning with fluctuating ocular, bulbar, limb, or respiratory symptoms; proceeding through bedside maneuvers such as the ice-pack test and sustained upward gaze testing; and continuing with antibody testing, electrophysiology, chest imaging, and management planning.

Laboratory and serologic evaluation demonstrated normal blood counts, normal thyroid function, normal creatine kinase, mildly elevated inflammatory markers in the setting of acute streptococcal pharyngitis, and positive acetylcholine receptor antibodies (Table *1*). Acetylcholine receptor binding antibody was elevated at 8.6 nmol/L, acetylcholine receptor blocking antibody was 42% inhibition, acetylcholine receptor modulating antibody was 68%, muscle-specific kinase antibody was negative, and low-density lipoprotein receptor-related protein 4 antibody was not detected. Repetitive nerve stimulation demonstrated a 17% decrement in compound muscle action potential amplitude with 3-Hz repetitive nerve stimulation of the right facial nerve recording from nasalis, consistent with impaired neuromuscular transmission. Single-fiber electromyography was not performed because acetylcholine receptor antibody positivity and repetitive nerve stimulation confirmed the diagnosis. Chest computed tomography demonstrated no thymoma or anterior mediastinal mass, with only mild age-related thymic involution.

Test	Result	Reference Range	Interpretation
White blood cell count	9.8 × 10^3/μL	4.0-11.0 × 10^3/μL	Within normal limits
Hemoglobin	12.7 g/dL	12.0-16.0 g/dL	Within normal limits
Platelet count	286 × 10^3/μL	150-450 × 10^3/μL	Within normal limits
Thyroid-stimulating hormone	1.7 μIU/mL	0.4-4.5 μIU/mL	Within normal limits
Free thyroxine	1.1 ng/dL	0.8-1.8 ng/dL	Within normal limits
Creatine kinase	74 U/L	30-200 U/L	Within normal limits
Erythrocyte sedimentation rate	22 mm/hr	0-20 mm/hr	Mildly elevated
C-reactive protein	1.8 mg/dL	<0.5 mg/dL	Elevated, interpreted in the setting of acute streptococcal pharyngitis
Acetylcholine receptor binding antibody	8.6 nmol/L	<0.5 nmol/L	Positive
Acetylcholine receptor blocking antibody	42% inhibition	<15% inhibition	Positive
Acetylcholine receptor modulating antibody	68%	<32%	Positive
Muscle-specific kinase antibody	Negative	Negative	Not detected
Low-density lipoprotein receptor-related protein 4 antibody	Not detected	Negative	Not detected

TABLE 1: Laboratory and serologic evaluation.

Laboratory evaluation demonstrated normal blood counts, normal thyroid function, normal creatine kinase, mildly elevated inflammatory markers in the setting of acute streptococcal pharyngitis, and positive acetylcholine receptor antibodies supporting acetylcholine receptor antibody-positive myasthenia gravis. Reference ranges may vary slightly by laboratory. μL: microliter; g/dL: grams per deciliter; μIU/mL: micro-international units per milliliter; ng/dL: nanograms per deciliter; U/L: units per liter; mm/hr: millimeters per hour; mg/dL: milligrams per deciliter; nmol/L: nanomoles per liter.

The differential diagnosis included multiple neuro-ophthalmologic, vascular, autoimmune, infectious, and paraneoplastic disorders (Table *2*). Third-nerve palsy was considered because ptosis and diplopia may reflect an oculomotor lesion, particularly when pupillary involvement suggests a compressive aneurysm. Horner syndrome from cervical artery dissection was considered because partial ptosis with miosis can mimic ocular myasthenia, especially when headache or neck pain is present. Brainstem or vertebrobasilar ischemia was considered because hypertension, diplopia, dysarthria, and gait difficulty can localize to the posterior circulation. Giant cell arteritis was considered because the patient was aged 50 years and had diplopia, fatigue, and weight loss; jaw claudication was distinguished from myasthenic chewing fatigue by history. Thyroid eye disease, Lambert-Eaton myasthenic syndrome, chronic progressive external ophthalmoplegia, Miller Fisher syndrome, and botulism were also considered. The fluctuating pattern, worsening with sustained effort, positive ice-pack test, antibody result, and electrophysiologic findings supported generalized myasthenia gravis.

Diagnostic consideration	Reason considered	Findings supporting or arguing against in this case
Generalized myasthenia gravis	Fluctuating ptosis, diplopia, dysarthria, limb fatigability, positive ice-pack test	Supported by fatigability, bedside response, positive acetylcholine receptor antibodies, repetitive nerve stimulation decrement, and bulbar symptoms
Compressive third-nerve palsy	Ptosis and diplopia may reflect oculomotor nerve involvement; pupillary involvement raises concern for aneurysm	Pupils were equal and reactive, and symptoms were fatigable rather than fixed
Horner syndrome/cervical artery dissection	Partial ptosis with miosis may mimic ocular myasthenia, especially with headache or neck pain	No anisocoria, miosis, anhidrosis, headache, or neck pain was present
Brainstem or vertebrobasilar ischemia	Hypertension, diplopia, dysarthria, and gait difficulty may reflect posterior circulation disease	No fixed focal neurologic deficit was present, and symptoms fluctuated with fatigue rather than occurring abruptly
Giant cell arteritis	Age >50, diplopia, weight loss, fatigue; jaw claudication may mimic chewing fatigue	No jaw claudication, scalp tenderness, acute visual loss, or temporal artery tenderness was reported; erythrocyte sedimentation rate was only mildly elevated in the setting of infection
Thyroid eye disease	Diplopia and ocular misalignment may occur with orbital disease	Thyroid function was normal, and there was no proptosis, lid retraction, or restrictive ophthalmopathy
Lambert-Eaton myasthenic syndrome	Proximal weakness and malignancy association, particularly with constitutional symptoms	Reflexes were preserved, there were no autonomic symptoms, and the presentation was ocular-bulbar predominant rather than proximal leg weakness predominant
Chronic progressive external ophthalmoplegia	Ptosis and ophthalmoplegia may be chronic and progressive	Less consistent with fluctuating fatigability and positive ice-pack response
Miller Fisher syndrome	Ophthalmoplegia may follow infection	Reflexes were preserved, there was no ataxia, and the subacute fatigable course favored myasthenia gravis
Botulism	Ptosis, diplopia, bulbar weakness, and autonomic symptoms may occur	Pupils were reactive, there were no autonomic symptoms or exposure history, and the fluctuating chronic course favored myasthenia gravis

TABLE 2: Differential diagnosis for ptosis, diplopia, dysarthria, and fatigability.

The key clinical findings supporting the diagnosis of generalized myasthenia gravis, including fluctuating ocular symptoms, fatigable bulbar symptoms, and evening gait difficulty, are summarized in Table *3*, along with their diagnostic significance. The treatment protocol addressed both streptococcal pharyngitis and generalized myasthenia gravis. She was instructed to complete antibiotic therapy for group A streptococcal pharyngitis. Antibiotic selection was reviewed in the context of suspected myasthenia gravis, and she was advised to avoid or use caution with medications known to worsen neuromuscular transmission, including fluoroquinolones, macrolides, aminoglycosides, magnesium, beta-blockers, and neuromuscular blocking agents [11]. Her antihypertensive regimen included amlodipine 5 mg daily, which was continued because calcium-channel blockers are not among the highest-risk medication classes for worsening myasthenia gravis, though she was advised to discuss all medication changes with neurology.

Clinical feature	Finding in this case	Diagnostic significance
Concurrent infection	Rapid antigen test positive for group A Streptococcus	Provided a plausible explanation for upper respiratory symptoms and fatigue but did not explain focal fatigable ocular findings
Ptosis	Progressive right eyelid drooping over several weeks	Common ocular manifestation of MG
Diplopia	Intermittent double vision	Suggests fluctuating extraocular muscle involvement
Fatigability	Symptoms worsened later in the day	Characteristic temporal pattern of MG
Sustained upward gaze	Provoked strabismus and worsening ocular misalignment	Supports fatigable ocular weakness
Ice-pack test	Improved eyelid elevation	Bedside clue supporting ocular MG in the appropriate clinical context
Bulbar symptoms	Slurred speech worsened with prolonged conversations	Suggests possible bulbar fatigability beyond isolated ocular disease
Limb/functional symptoms	Trouble walking in the evening	Raises concern for generalized fatigability
Neurologic examination	Otherwise nonfocal	Helped reduce concern for focal central neurologic deficit
Initial diagnostic plan	MG antibody panel, neurology referral, EMG, and chest imaging	Appropriate next steps for confirmatory evaluation and thymic assessment

TABLE 3: Pertinent clinical findings and diagnostic significance.

MG: Myasthenia gravis

After neurology evaluation within one week, pyridostigmine was started at 30 mg orally three times daily and later increased to 60 mg three times daily as tolerated. At the two-week follow-up, she reported marked improvement in right ptosis, diplopia frequency, end-of-day dysarthria, and fatigue. Disease-related functional impairment was assessed using the Myasthenia Gravis Activities of Daily Living (MG-ADL) profile, a validated eight-item patient-reported instrument designed to evaluate ocular, bulbar, respiratory, and limb-related symptoms and monitor clinical response to treatment [12]. Myasthenia Gravis Activities of Daily Living score improved from 7 at presentation to 3 at two-week follow-up. She reported fewer episodes of diplopia, improved eyelid opening, less end-of-day dysarthria, and no dyspnea, dysphagia, or choking.

Because she had bulbar symptoms and concurrent infection, she was given strict emergency precautions for dyspnea, dysphagia, choking, worsening dysarthria, neck flexor weakness, inability to count aloud, rapidly progressive limb weakness, or respiratory distress. The follow-up plan included neurology management, chest imaging review for thymic disease, gynecologic evaluation for postmenopausal bleeding, and age-appropriate malignancy screening. The clinical timeline from symptom evolution to diagnostic referral is shown in Figure *2*.

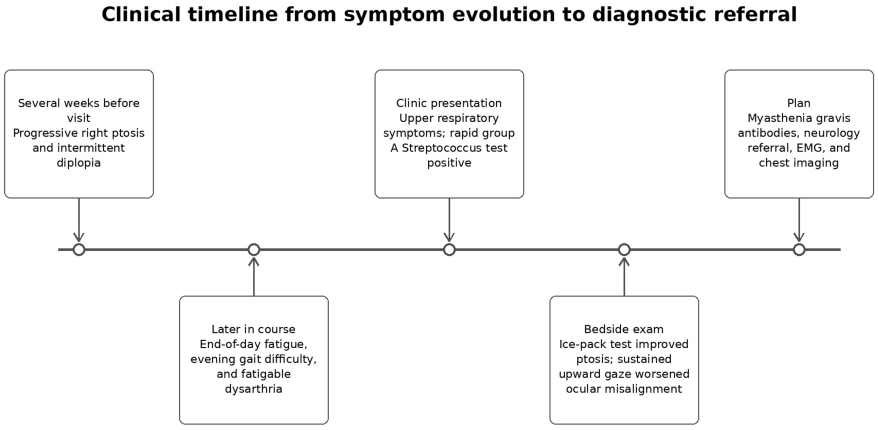


FIGURE 2: Clinical timeline from symptom evolution to diagnostic referral.

This original timeline summarizes the patient's several-week progression of right ptosis, intermittent diplopia, end-of-day fatigue, evening gait difficulty, and fatigable dysarthria, followed by clinic presentation for upper respiratory symptoms, positive group A Streptococcus testing, bedside findings concerning for myasthenia gravis, antibody testing, and neurology referral for confirmatory evaluation. This figure was created by the authors for this manuscript and does not include patient-identifying media, third-party copyrighted materials, or BioRender elements.

Discussion

This case illustrates generalized myasthenia gravis identified during an outpatient visit for streptococcal pharyngitis. The patient's positive rapid group A Streptococcus test explained her acute upper respiratory symptoms but did not explain several weeks of fluctuating right-sided ptosis, intermittent diplopia, fatigable dysarthria, and evening gait difficulty. Although true diagnostic delay did not occur in this case because the neuromuscular workup was initiated during the same visit, the scenario illustrates how a confirmed common diagnosis can prematurely close the diagnostic process if discordant symptoms are not pursued. Anchoring bias, in which clinicians focus on an initial piece of information without sufficiently adjusting to later data, has been demonstrated in physician decision-making and can contribute to delayed recognition of serious disease [13].

Several previously published case reports demonstrate how myasthenia gravis may be initially obscured by more common diagnoses, although the diagnostic circumstances and clinical outcomes differ. Menon and Mokkath described a 48-year-old man with hypertension who initially presented with dysphagia, dysarthria, and other bulbar symptoms suggestive of an acute ischemic stroke [14]. Persistent symptoms and subsequent development of fatigable weakness prompted additional evaluation, ultimately establishing acetylcholine receptor antibody-positive myasthenia gravis. Similarly, Mangrio et al. reported an 82-year-old man presenting with unilateral ptosis, dysphagia, and dysarthria who was initially evaluated for acute ischemic stroke before being diagnosed with myasthenia gravis [15]. Both reports emphasize the importance of recognizing fluctuating ocular and bulbar weakness rather than attributing symptoms exclusively to a more familiar neurologic diagnosis. In contrast, the current case involved a confirmed acute infectious condition, and the neuromuscular symptoms were recognized during the initial outpatient evaluation rather than after a documented period of diagnostic delay.

Aili and Maimaiti described a 33-year-old woman whose myasthenia gravis initially manifested predominantly as fluctuating dysphagia and a pharyngeal foreign-body sensation, resulting in an initial psychiatric misdiagnosis [16]. Her symptoms subsequently worsened following upper respiratory infections, and repeated infection-associated exacerbations contributed to the eventual recognition of generalized myasthenia gravis. This report is particularly relevant because it demonstrates the relationship between systemic infection and worsening neuromuscular weakness. Similar to that case, the present patient experienced symptoms suggestive of a neuromuscular junction disorder in the setting of an acute upper respiratory infection. However, the present patient exhibited prominent ocular manifestations, including fatigable ptosis and intermittent diplopia, which provided additional diagnostic clues. Importantly, although infection may precipitate worsening in patients with myasthenia gravis, the coexistence of streptococcal pharyngitis does not establish a causal relationship with the onset of the autoimmune disorder.

The similarities and differences among these cases can be understood through the variable distribution of

weakness associated with impaired neuromuscular transmission. Ocular and bulbar muscles are particularly susceptible to activity-dependent fatigability, resulting in clinical presentations ranging from isolated ptosis or diplopia to dysarthria, dysphagia, and generalized weakness. The specific symptoms and their severity may influence which alternative diagnoses are initially considered. The distinguishing feature of the present case is the recognition of a separate neuromuscular symptom pattern despite a confirmed common infection that could otherwise explain nonspecific fatigue. Collectively, these reports support careful assessment of symptom duration, fluctuation, anatomical distribution, and response to sustained activity. They also highlight the importance of considering myasthenia gravis when ocular or bulbar symptoms are disproportionate to the apparent primary diagnosis, thereby reducing the risk of premature diagnostic closure and delayed neurologic evaluation.

The presentation was generalized rather than purely ocular. Isolated ocular myasthenia typically involves ptosis and diplopia without bulbar, limb, or respiratory involvement. In this patient, fatigable dysarthria and evening gait difficulty indicated disease beyond isolated ocular involvement and supported Myasthenia Gravis Foundation of America class IIb. This distinction matters because bulbar symptoms can signal increased risk for aspiration, respiratory compromise, and myasthenic crisis.

Concurrent infection is clinically important in myasthenia gravis. Respiratory or systemic infections are among the most common precipitants of myasthenic exacerbation and crisis [6,7]. Fever, systemic inflammation, and infection-related physiologic stress may reduce the safety margin of neuromuscular transmission. Management therefore requires treatment of the infection, careful antibiotic selection, avoidance of medications that may worsen myasthenia gravis, and clear emergency precautions.

The differential diagnosis was broad because ptosis, diplopia, dysarthria, fatigue, and gait difficulty can reflect ocular, neuromuscular, vascular, infectious, endocrine, or paraneoplastic disease. Third-nerve palsy, Horner syndrome from cervical artery dissection, brainstem ischemia, and giant cell arteritis are particularly important not to miss because they may require urgent imaging or treatment. Constitutional symptoms and postmenopausal bleeding also required attention because thymoma, paraneoplastic neuromuscular syndromes, and non-neuromuscular malignancies may enter the differential. Chest imaging is standard in confirmed or strongly suspected myasthenia gravis because thymoma occurs in a clinically meaningful minority of patients and may change management [1,2].

Medication counseling is another practical teaching point. Fluoroquinolones, macrolides, aminoglycosides, magnesium, beta-blockers, neuromuscular blocking agents, and selected other medications have been associated with worsening myasthenia gravis or impaired neuromuscular transmission [11]. In patients presenting with infection, the need for antimicrobial therapy should be balanced against the risk of neuromuscular worsening, and alternatives should be considered when clinically appropriate.

This case has several limitations. As a single case, it cannot establish frequency or causality. The follow-up lasted two weeks; at the two-week follow-up, she reported fewer episodes of diplopia, improved eyelid opening, less end-of-day dysarthria, and no dyspnea, dysphagia, or choking. However, longer follow-up would be needed to determine persistence of response, medication tolerance, thymic management, and risk of relapse. Nevertheless, the case provides a practical first-contact lesson: a common infectious diagnosis should not end evaluation when focal fatigable ocular or bulbar weakness is present.

Conclusions

In patients presenting with a common acute infection, a several-week history of fluctuating ptosis, diplopia, fatigable dysarthria, and end-of-day weakness should prompt bedside fatigability testing, myasthenia gravis serology, careful medication review, and urgent neurology referral. Streptococcal pharyngitis explained the acute upper respiratory symptoms in this case, but the concurrent pattern of fatigable ocular, bulbar, and limb symptoms supported generalized myasthenia gravis rather than nonspecific infection-related fatigue. Early recognition is clinically important because infection may precipitate worsening weakness or myasthenic crisis, and timely diagnosis permits appropriate treatment, chest imaging for thymic disease, medication counseling, and safety planning.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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