EJCRIM
European Journal
of Case Reports
in Internal Medicine

ATYPICAL BULBAR MYASTHENIA GRAVIS IN AN ELDERLY MALE UNMASKED BY LEVOFLOXACIN: A DIAGNOSTIC CHALLENGE

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Received: 28/03/2025
Accepted: 08/04/2025
Published: 23/04/2025

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: Written informed consent obtained from the patient to secure permission for publishing their clinical history.
Acknowledgments: We would like to express our gratitude to the interdisciplinary team of physicians, nurses, and healthcare professionals who contributed to the diagnosis and management of this case.

How to cite this article: Thiriveedi M, Chimakurthy A, Patel S, Baddam S, ElBeblawy R, Reddy PJ. Atypical bulbar myasthenia gravis in an elderly male unmasked by levofloxacin: a diagnostic challenge. *EJCRIM* 2025;**12**:doi:10.12890/2025_005386

ABSTRACT

Background: Myasthenia gravis (MG) is an autoimmune neuromuscular disorder that typically presents with ocular symptoms. Isolated bulbar symptoms, such as dysphagia and dysarthria, are rare, and most commonly seen in men with late-onset MG. We report one such rare case of MG in an 82-year-old male presenting with progressive bulbar weakness, seemingly triggered by levofloxacin use.

Case Report: An 82-year-old male with multiple comorbidities presented with progressive weakness, dysphagia, and drooling following levofloxacin therapy. Examination revealed neck drop, weak lower facial muscles, and dysarthria. Initial neuroimaging and labs were inconclusive. Neurology initiated pyridostigmine with rapid improvement. Elevated acetylcholine receptor antibodies confirmed MG. He was started on efgartigimod alfa in the outpatient setting with improvement in MG composite scores.

Conclusions: Atypical bulbar MG can mimic other neurologic disorders and is prone to delayed diagnosis, especially in the elderly. Clinicians should maintain a high index of suspicion, particularly when symptoms worsen after medication exposures like fluoroquinolones. Early diagnosis and appropriate treatment can significantly improve outcomes and quality of life.

KEYWORDS

Myasthenia gravis, bulbar symptoms, acetylcholine receptor antibodies, levofloxacin, elderly, autoimmune neuromuscular disorder

LEARNING POINTS

- Levofloxacin can exacerbate or unmask myasthenia gravis.

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INTRODUCTION

Myasthenia gravis (MG) is an autoimmune neuromuscular junction disorder caused by antibodies targeting acetylcholine receptors or associated proteins[1]. It presents with fluctuating muscle weakness and fatigue. While ptosis and diplopia are common initial signs, atypical bulbar presentations—such as dysphagia and dysarthria—can pose diagnostic dilemmas. These are more often seen in elderly males. Diagnostic delay is common due to symptom overlap with stroke or motor neuron disease. We describe a case of late-onset bulbar MG in an elderly male, potentially unmasked by levofloxacin.

CASE DESCRIPTION

An 82-year-old male with a history of hypertension, atrial fibrillation, obstructive sleep apnoea, and obesity (body mass index 37.3 kg/m^2) presented to his primary care physician for right olecranon bursitis and urinary tract infection and was prescribed levofloxacin. Three days later, he presented to the emergency department with worsening shortness of breath and weakness. Diagnosed with pneumonia, he initially declined admission but returned 2 days later with worsening generalized weakness, ambulation difficulty, dysphagia, and drooling.

On examination, he was alert and oriented with stable vital signs. Cardiopulmonary and abdominal exams were unremarkable. The right elbow appeared swollen and erythematous. Neurologically, he exhibited lower facial and neck weakness; extremity strength, coordination, and sensation were intact. Reflexes were diffusely diminished. Laboratory workup was unremarkable *(Table 1)*, and a chest X-ray showed left lower lobe pneumonia *(Fig. 1)*. Levofloxacin was continued, and ceftriaxone was added for levofloxacin-resistant *Escherichia coli* and *Klebsiella* noted on recent urine cultures.

Initially attributed to infection and age-related decline, his symptoms progressed, including copious secretions, garbled speech, and worsening dysphagia. He developed atrial fibrillation with rapid ventricular response, managed with a diltiazem drip. Modified barium swallow revealed decreased tone and pooling of secretions in the vallecula, with evidence of presbyesophagus.

Further history revealed progressive dysphagia, weight loss (>45 kg), and difficulty managing secretions over several months. His wife reported symptom worsening after starting levofloxacin. The antibiotic was discontinued, and neurology was consulted on day 7 of hospitalization. A trial of pyridostigmine 60 mg every 8 hours was initiated. Magnetic resonance imaging (MRI) brain showed no acute infarct; cervical/thoracic spine MRI showed multilevel degenerative arthropathy and severe foraminal stenosis (C4-C7). Steroids were deferred due to concurrent infections.

By the next day, he showed significant improvement of strength but developed bradycardia (HR 30/min), likely due to pyridostigmine. The dose was reduced to 30 mg every 8 hours, leading to symptom recurrence. He was started on a 5-day course of intravenous immunoglobulins (IVIG, 400 mg/kg). Pyridostigmine was switched to 180 mg extended release, which improved symptoms with stable heart rate. Computed tomography (CT) scan of the chest showed no thymoma. Bedside electromyography was unsuccessful due to intensive care unit device artifacts. Acetylcholine receptor antibody (AChR) levels were elevated at 20.9 nmol/l *(Table 1)*. The patient improved, tolerated oral intake, and was discharged to a rehabilitation facility. He was later initiated on weekly efgartigimod alfa infusions, with MG composite score improving from 13 to 5 at 6 months follow up.

Laboratory data	Measured value	Reference range
White blood cell count (×10^3/µl)	11.22	4.8-10.8
Haemoglobin (g/dl)	15.9	13.5-17.5
Platelet count (×10^3/µl)	242	130-400
Sodium (mmol/l)	138	136-145
Potassium (mmol/l)	3.9	3.5-5.1
Chloride (mmol/l)	101	98-107
Bicarbonate (mEq/l)	28	22-29
Blood urea nitrogen (mg/dl)	7	8-22
Creatinine (mg/dl)	0.5	0.7-1.2
Glucose (mg/dl)	84	70-104
Calcium (mg/dl)	8.4	8.8-10.2
Thyroid stimulating hormone (µIU/ml)	1.99	0.27-4.20
Creatine kinase (U/l)	85	24-204
Erythrocyte sedimentation rate (mm/hr)	2	0-15
AChR binding antibody (nmol/l)	20.9	<0.02
MuSK antibody (nmol/l)	0.00	0-0.02

Abbreviations: AchR, acetylcholine receptor; MuSK, muscle-specific kinase.

Table 1. Laboratory values on admission and AChR antibody levels.

DISCUSSION

Myasthenia gravis is the most common neuromuscular junction disorder, with an annual incidence of 0.21-2 patients per 100,000[2]. Peak incidence occurs in younger women and older men[3]. Late-onset MG often presents with bulbar symptoms such as dysphagia, dysarthria, and dysphonia, accounting for 30% of all MG cases.

Diagnosis is confirmed via detection of AChR or muscle-specific tyrosine kinase (MuSK) or low-density lipoprotein receptor-related protein 4 (LRP4) antibodies.

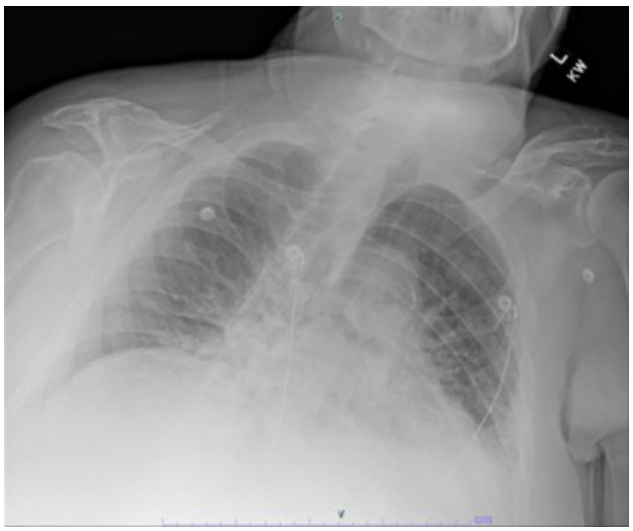

Figure 1. Chest X-ray on admission showing left lower lobe pneumonia.

Electrodiagnostic testing is used when antibody tests are negative, or symptoms are atypical. Thymoma screening and evaluation for other autoimmune diseases are essential.

Our case underscores the diagnostic challenge posed by atypical MG in the elderly, especially when presenting without classic ocular symptoms. Diagnostic delay was due to overlapping features of infection, age-related decline, and lack of ocular involvement.

Reports highlight similar delays: Basiri et al. described a 50-year-old male misdiagnosed with motor neuron disease[4]; Adamu et al. reported a 15-month delay in a 72-year-old male with atypical bulbar MG[5]. Schon et al. estimated an average delay of 4.5 months in the elderly[6]. Owing to the vast majority of possible causes of neuromuscular symptoms including stroke, Parkinson's, motor neuron disease, and neuropathies, myasthenia is thought to be under-diagnosed in elderly patients[7].

Levofloxacin and other fluoroquinolones have known neuromuscular blockade properties and may exacerbate MG[8]. Awareness of such triggers is critical. Our patient scored a 7 on the Naranjo scale which suggests this is a probable adverse drug reaction[9]. Diagnostic delay remains a concern; literature cites misdiagnoses lasting months. High clinical suspicion, detailed history, and antibody testing are pivotal.

The mainstay of treatment for MG is the use of anticholinesterases like pyridostigmine. Most patients require immunosuppressive therapy with glucocorticoids. Thymectomy is considered in younger patients. For myasthenic crisis, IVIG, plasma exchange, and newer biologics such as monoclonal antibodies or FcRn inhibitors are used[10].

Prompt diagnosis and therapy are critical in reducing morbidity, especially in elderly patients.

CONCLUSION

This case highlights the importance of recognizing atypical presentations of MG in elderly patients, particularly when presenting with isolated bulbar symptoms and without classical ocular involvement. Delayed diagnosis can significantly impact morbidity, especially when compounded by concurrent infections and polypharmacy. Clinicians should maintain a high index of suspicion for MG in elderly patients with unexplained dysphagia, drooling, and neck weakness. Early initiation of targeted therapy such as anticholinesterases and immunomodulators can lead to rapid clinical improvement and prevent complications. Our case underscores the need for thorough clinical evaluation and awareness of medication-induced exacerbations in diagnosing late-onset MG.

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