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Recurrent Myasthenic Crisis Triggered By Infection: Successful Management With Plasmapheresis

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ABSTRACT

Introduction: Myasthenia gravis (MG) is an autoimmune disorder of the neuromuscular junction characterized by fluctuating skeletal muscle weakness. The most severe complication is myasthenic crisis (MC), defined as acute respiratory failure requiring ventilatory support, which occurs in up to 20% of patients during the disease course. Infections are the most frequent precipitating factor, complicating diagnosis and worsening prognosis. Plasmapheresis (therapeutic plasma exchange, TPE) is an effective therapy that provides rapid clinical improvement in acute management.

Case series: We report a 46-year-old woman with recently diagnosed generalized MG who experienced recurrent MC three weeks after hospital discharge. In her first admission, she developed acute respiratory failure requiring intubation, intensive care, and three sessions of plasmapheresis. On the second admission, she presented with progressive shortness of breath, hoarseness, dysphagia, fever, productive cough, and headache. She had stopped pyridostigmine due to dysphagia. Physical examination showed ptosis, limb weakness, and hypoxemia, while chest radiography revealed pneumonia. Despite oxygen therapy, her respiratory function deteriorated rapidly, leading to intubation and mechanical ventilation. Management included broad-spectrum antibiotics, ventilatory support, and two additional sessions of plasmapheresis, which resulted in significant improvement in both respiratory and neurological function. She was discharged in stable condition after 17 days of hospitalization.

Conclusion: This case underscores the diagnostic and therapeutic challenges of recurrent MC triggered by infection. Prompt recognition, timely initiation of plasmapheresis, and multidisciplinary care are crucial in reducing morbidity and mortality. Preventive measures, adherence to immunosuppressive therapy, and infection control are essential to minimize recurrence and improve long-term outcomes

KEYWORDS: Myasthenia Gravis; Myasthenic Crisis; Infections; Recurrence; Plasmapheresis

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INTRODUCTION

Myasthenia gravis (MG) is an antibody-mediated autoimmune disorder of the neuromuscular junction that causes impaired synaptic transmission and fluctuating skeletal muscle weakness, typically worsening with exertion and improving with rest.[1] Ocular symptoms such as ptosis and diplopia are common initial manifestations, but many patients progress to generalized disease affecting bulbar, facial, limb, and respiratory muscles. The most severe complication is myasthenic crisis (MC), defined as acute respiratory insufficiency requiring intubation and mechanical ventilation, occurring in 15–20% of patients and remaining a major source of morbidity despite advances in intensive care.[1,2]

Infections, particularly of the respiratory tract, are the most frequent triggers of MC, responsible for one-third to half of cases. These infections not only worsen respiratory weakness but also mimic crisis symptoms, such as dyspnea and ineffective cough, making timely diagnosis difficult. Hypoxemia from infection may mask neuromuscular failure, while systemic inflammation promotes antibody production and accelerates receptor degradation.[3,4] Such overlap frequently delays recognition and worsens outcomes.[2,4]

Plasmapheresis (therapeutic plasma exchange, TPE) has proven to be a rapid and effective rescue therapy in MC by removing pathogenic autoantibodies and immune complexes. Compared with intravenous immunoglobulin, TPE offers faster symptom relief, particularly in critically ill patients with respiratory compromise. Early initiation, usually five exchanges within 1–2 days, is associated with better outcomes.[5,6]

This case report describes a 46-year-old woman with newly diagnosed MG who developed recurrent MC triggered by pneumonia shortly after initial discharge. The case underscores the challenges of infection-induced crises, the importance of early recognition and respiratory support, and the effectiveness of repeated plasmapheresis in acute management.[7,8]

CASE REPORT

A 46-year-old woman presented to the Emergency Department with three days of progressive shortness of breath, hoarseness,

dysphagia, and right lower limb weakness, showing evening exacerbation with partial morning improvement. On admission day, she developed fever, productive cough with difficulty expectorating, and headache. She had not taken pyridostigmine for four days due to dysphagia. She was diagnosed with generalized myasthenia gravis (MG) in May 2025 and had been hospitalized the previous month for myasthenic crisis with acute respiratory failure, requiring ICU care, intubation, and three plasmapheresis sessions. Type 2 diabetes mellitus was also diagnosed during that admission. Past history included Grade 2 hypertension for eight years and hypertensive heart disease for one year.

On initial examination, she was alert (GCS E4V5M6). Vital signs: BP 165/113 mmHg, HR 101 bpm, RR 24/min, temperature 37.5°C, and SpO_2 93% on room air, improving to 97% with 3 L/min oxygen. Neurological findings included bilateral ptosis and right lower limb weakness (MRC 4/5) without other focal deficits. Shortly after ward transfer, dyspnea worsened despite oxygen supplementation, requiring escalation to Jackson Rees at 12 L/min. A prior RNS test (May 2025) had shown >10% decremental response in the left orbicularis oculi. Chest radiography confirmed pneumonia. Working diagnosis was recurrent myasthenic crisis triggered by infection, complicated by T2DM, ARDS, and HHD. The patient required intubation, mechanical ventilation, and two further plasmapheresis sessions in the ICU. After 17 days of intensive management, her respiratory and neurological status improved, and she was discharged in stable condition..

DISCUSSION

Myasthenia gravis (MG) exacerbation—defined as an acute worsening of muscle weakness due to heightened autoimmune activity at the neuromuscular junction—is a common event, affecting approximately one-third of all MG patients.[9,10] Our case is particularly significant as it describes a recurrent Myasthenic Crisis (MC) within a remarkably short period of just three weeks post-discharge, suggesting that the initial treatment failed to achieve sustained immunological stability.[11]

The recurrence was driven by a dual etiology, a dangerous combination of the two most frequent triggers for MC: an active infection and medication non-adherence.[12] The patient presented with pneumonia, which is cited as the precipitating factor in up to two-thirds of crisis cases.[13] Infection-induced systemic inflammation and metabolic stress severely impair neuromuscular transmission.[10,14] This infectious component, combined with the patient's temporary cessation of pyridostigmine due to worsening dysphagia, led to an accelerated and severe autoimmune attack on the neuromuscular junction³. This highlights a critical, modifiable risk factor in recurrent MG: the intersection of acute physiological stress (infection) and inadequate maintenance therapy (non-adherence). Furthermore, chronic comorbidities, such as Type 2 Diabetes Mellitus and Hypertension, independently elevated the patient's risk profile for exacerbation (HR 1.09 and 1.15, respectively).[10]

This case underscores the persistent diagnostic pitfall inherent in infection-triggered MC. Respiratory symptoms associated with pneumonia (e.g., ineffective cough, dyspnea) clinically overlap with, and can thus mask, the subtle onset of neuromuscular weakness, potentially delaying the essential intervention of intubation and immunotherapy.[14] While the patient's MG diagnosis was previously confirmed by clinical features and a Repetitive Nerve Stimulation (RNS) test showing a >10% decremental response—a finding correlated with bulbar and respiratory involvement—the focus during her second presentation was quickly forced onto acute respiratory support.[15] The educational value here is the need for clinicians, particularly in the Emergency Department and ICU, to maintain a uniformly high index of suspicion for impending MC in any known MG patient presenting with infection, regardless of recent stabilization.

In the acute management of MC, rapid immunomodulation is mandatory. Therapeutic Plasma Exchange (TPE) was chosen as the primary intervention, both initially and for the recurrent crisis. TPE is often preferred over intravenous immunoglobulin (IVIG) in severe, intubated patients due to its mechanism of directly removing circulating autoantibodies and immune complexes, offering a faster and more predictable onset of clinical effect—a critical advantage in acute respiratory failure.[2,5] Our patient, requiring two additional sessions of TPE beyond the initial regimen, demonstrated significant improvement in both neurological and respiratory function, reinforcing TPE's role as an effective, second-line intervention for recurrent, highly inflammatory crises. Although the standard protocol is often five alternate-day sessions TPE proved essential in stabilizing the patient during the recurrent episode, providing the necessary window for antibiotics to manage the underlying infection.[5,6]

The successful management, culminating in patient discharge after 17 days (within the expected recovery window for severe MC), emphasizes that immediate, aggressive treatment can overcome even highly unstable disease.[12] However, the short-term recurrence carries adverse prognostic implications, including a potentially higher risk of future mortality and slower recovery.[12] The major clinical implication derived from this case is the urgent need for robust long-term relapse prevention strategies, which requires a fundamental shift in follow-up care. This comprehensive approach must first focus on Intensified Patient Education: counseling should move beyond general compliance to ensure patients can recognize prodromal signs of relapse, understand how to manage dysphagia without abruptly stopping medication, and appreciate the absolute necessity of maintaining strict adherence to immunosuppressive and anticholinesterase therapy.[7,8,11] Secondly, it mandates Aggressive Infection Control, incorporating proactive measures like routine vaccination and the strict avoidance of medications known to impair neuromuscular transmission, such as aminoglycosides and fluoroquinolones, during concurrent infection.[13] Crucially, Meticulous Comorbidity Control is also vital, as strict management of chronic diseases like T2DM and Hypertension independently raises the risk of MC.[10,13]

In conclusion, this case serves as a poignant reminder that while advancements in critical care and TPE have dramatically improved MC survival, the ultimate prevention of recurrence hinges on effectively addressing the combined pathological effects of infection, immunological instability, and patient non-adherence in the long-term clinical setting.

CONCLUSION

This case emphasizes the life-threatening challenge of recurrent myasthenic crisis (MC), triggered by pneumonia and complicated by anti-cholinesterase non-adherence. The patient's rapid recovery after 17 days, requiring intubation and therapeutic plasma exchange (TPE), validates the critical need for early recognition and prompt respiratory and immunomodulatory intervention. The short interval between crises highlights the necessity of a rigorous, multidisciplinary approach—integrating intensive care, infection control, and immunotherapy—to stabilize the disease. Crucially, long-term success depends on continuous patient education regarding adherence to maintenance therapy and infection prevention to mitigate future relapse risk.

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REFERENCES

1. Dresser L, Wlodarski R, Rezania K, Soliven B. Myasthenia Gravis: Epidemiology, Pathophysiology and Clinical Manifestations. Vol. 10, Journal of Clinical Medicine. 2021.
2. 2Rifani Masharto A, Lubis B, Lubis AP, Nadeak R. Plasmapheresis in Myasthenia Gravis Crisis. Maj Anest Crit Care. 2023;41(3):185–8.
3. Bril V, Gilhus NE. Aging and infectious diseases in myasthenia gravis. J Neurol Sci [Internet]. 2025;468(July 2024):123314. Available from: https://doi.org/10.1016/j.jns.2024.123314
4. Nelke C, Stascheit F, Eckert C, Pawlitzki M, Schroeter CB, Huntemann N, et al. Independent risk factors for myasthenic crisis and disease exacerbation in a retrospective cohort of myasthenia gravis patients. J Neuroinflammation [Internet]. 2022;19(1):1–12. Available from: https://doi.org/10.1186/s12974-022-02448-4
5. Al-Ahmer I, Elshony H. Determinants of quality of life changes with plasmapheresis in patients with myasthenia gravis. Egypt J Neurol Psychiatry Neurosurg. 2021;57(1).
6. Pavlekovics M, Engh MA, Lugosi K, Szabo L, Hegyi P, Terebessy T, et al. Plasma Exchange versus Intravenous Immunoglobulin in Worsening Myasthenia Gravis: A Systematic Review and Meta-Analysis with Special Attention to Faster Relapse Control. Biomedicines. 2023;11(12).
7. Yee S, Rankin WR, Moore T, Brown A, Rappaport D, Martini WA. Myasthenic Gravis Crisis in an 85-Year-Old Male Requiring Emergent Intubation and Plasmapheresis. Cureus. 2025;17(3).
8. Roper J, Fleming ME, Long B, Koyfman A. Myasthenia Gravis and Crisis: Evaluation and Management in the Emergency Department. J Emerg Med [Internet]. 2017;53(6):843–53. Available from: https://doi.org/10.1016/j.jemermed.2017.06.009
9. Petrucelli N, Barra ME, Koehl JL. Evaluation of Medication Exposure on Exacerbation of Disease in Patients With Myasthenia Gravis. Neurohospitalist. 2024;14(1):52–7.
10. Qi CZ, Lin Y, Li Y, Vu T, De Ruyck F, Gelinas D, et al. Characteristics and healthcare utilization of patients with myasthenia gravis exacerbation. J Neurol Sci [Internet]. 2025;468(November 2024):123322. Available from: https://doi.org/10.1016/j.jns.2024.123322
11. Chien CY, Chang CW, Liao MF, Chu CC, Ro LS, Wu YR, et al. Myasthenia gravis and independent risk factors for recurrent infection: a retrospective cohort study. BMC Neurol [Internet]. 2023;23(1):1–11. Available from: https://doi.org/10.1186/s12883-023-03306-3
12. Sharma S, Lal V, Prabhakar S, Agarwal R. Clinical profile and outcome of myasthenic crisis in a tertiary care hospital: A prospective study. Ann Indian Acad Neurol. 2013;16(2):203–7.
13. Godoy DA, de Mello LJV, Masotti L, Di Napoli M. The myasthenic patient in crisis: An update of the management in Neurointensive care unit. Arq Neuropsiquiatr. 2013;71(9 A):627–39.
14. Schroeter M, Thayssen G, Kaiser J. Myasthenia gravis - Exacerbation and Crisis. Aktuelle Neurol. 2018;45(4):271–7.
15. Pham TKA, De Tran V, Nguyen KT, Van Pham P, Tran TTT, Dewey RS, et al. Characterization of myasthenia gravis using clinical classification and repetitive nerve stimulation. Arch Balk Med Union. 2021;56(2):165–71.