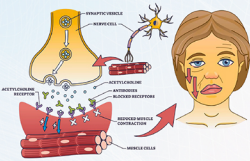


# EFGARTIGIMOD AS RESCUE THERAPY IN REFRACTORY MYASTHENIC CRISIS: A CASE REPORT

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## MYASTHENIA GRAVIS



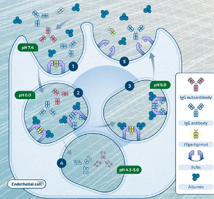
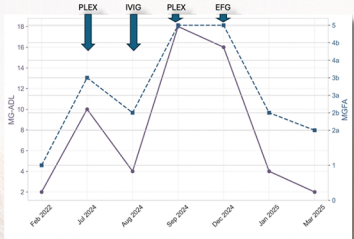
## BACKGROUND

Myasthenia gravis (MG) is an autoimmune disorder characterized by fluctuating skeletal muscle weakness caused by antibody-mediated impairment of neuromuscular transmission. The introduction of targeted therapies such as efgartigimod, a selective neonatal Fc receptor (FcRn) antagonist, has broadened treatment options for generalized MG, thanks to its ability to reduce pathogenic IgG antibody levels. However, the role of efgartigimod in the management of myasthenic crisis (MC), particularly in patients requiring prolonged ventilatory support, remains to be clarified.

## CASE PRESENTATION

We describe a 67-year-old man with ocular-onset MG, positive for anti-acetylcholine receptor (AChR) antibodies, diagnosed in 2022. He was initially treated with pyridostigmine and low-dose prednisone (5 mg/day), achieving partial symptom control. In July 2024, he developed a significant clinical worsening, with severe dysphagia and hypophonia. This episode led to hospitalization and seven plasma exchange (PLEX) sessions, resulting in a transient improvement (MG-ADL score from 10 to 4). Azathioprine was started as a steroid-sparing agent but discontinued shortly after due to hepatotoxicity.

In August 2024, the patient experienced a further acute exacerbation, manifesting as respiratory failure despite high-dose prednisone (50 mg/day) and intravenous immunoglobulin (IVIg). He required intubation, tracheostomy, and admission to the intensive care unit. Seven additional PLEX sessions were administered, but no sustained benefit was observed, and multiple attempts of ventilator weaning were unsuccessful. His clinical course was further complicated by the development of nosocomial pneumonia due to *Acinetobacter baumannii*, requiring prolonged multidrug antibiotic therapy and resulting in persistent airway colonization.



## THE ROLE OF EFGARTIGIMOD

Given the refractory nature of his condition and persistent ventilator dependence, efgartigimod was initiated in December 2024 at a dose of 10 mg/kg weekly for four weeks. The patient demonstrated rapid and marked clinical improvement: the MG-ADL score decreased from 16 to 4, he was successfully weaned from mechanical ventilation, and oral feeding was resumed. Mycophenolate mofetil (2 g/day) was then introduced for maintenance immunosuppression, and corticosteroids were gradually tapered. At three months follow-up, the patient remained clinically stable, with no new exacerbations and an improved quality of life.

## CONCLUSION

This case highlights the potential role of efgartigimod as a safe and effective rescue therapy in severe, refractory MG complicated by myasthenic crisis and prolonged ventilator dependence, despite of persistent bacterial colonization. Our experience supports the efficacy and safety of this drug in critically ill patients. Prospective studies are needed to further assess the role of efgartigimod in the acute management of myasthenic crisis.

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