ORIGINAL RESEARCH

# PLASMA EXCHANGE THERAPY (TPE) IN MYASTHENIC CRISIS PATIENTS

**Reza Fazri Prasetyo[1], Bastian Lubis[1]**

[1]*Rumah Sakit Umum Pusat Haji Adam Malik, Jl. Bunga Lau No.17, Kemenangan Tani, Kec. Medan Tuntungan, Kota Medan, 20136, Sumatera Utara, Indonesia*

**Article Info**

Article History:
Received: 06 August 2025
Accepted: 07 August 2025
Published: 07 August 2025

**Keywords:** myasthenic crisisp; therapeutic plasma exchange (TPE); Myasthenic Crisis; Neuromuscular Disorders; Immunomodulatory Treatment

**Corresponding Author**:
Reza Fazri Prasetyo
Email: Reza.anest@gmail.com

**Abstract**

Myasthenic crisis is an acute exacerbation of myasthenia gravis that results in weakness of the respiratory muscles, potentially leading to acute respiratory failure and the necessity for mechanical ventilation support. Approximately 15-20% of individuals with myasthenia gravis will experience a myasthenic crisis at some point in their lives, making it one of the most dangerous and life-threatening complications if not addressed promptly. Fortunately, mortality rates associated with myasthenic crises are currently decreasing, partly due to the effectiveness of therapeutic plasma exchange (TPE). TPE therapy works by removing pathological antibodies, immune complexes, and cytokines from the plasma and replacing them with replacement fluid. In this report, we discuss the case of a 61-year-old man with myasthenia gravis who was admitted to the emergency department (ED) and then to the intensive care unit (ICU) for one day due to a myasthenic crisis. He was intubated and received mechanical ventilation support, followed by plasma exchange therapy, which resulted in clinical improvement.

**Jurnal Mutiara Ners**
**E.ISSN: 2620-4061**
**Vol. 8 No. 2 Bulan 2025 (P101-105)**

Homepage: **http://e-journal.sari-mutiara.ac.id/index.php/NERS/**

**DOI:** https://doi.org/10.51544/jmn.v8i2.6287

**How To Cite**: Prasetyo, Reza Fazri, and Bastian Lubis. 2025. "Plasma Exchange Therapy (TPE) In Myasthenic Crisis Patients." *Jurnal Mutiara Ners* 8 (2): 101–5. https://doi.org/https://doi.org/10.51544/jmn.v8i2.6287

1. **Introduction**

Myasthenia gravis (MG) is a chronic autoimmune neuromuscular disorder characterized by the presence of circulating autoantibodies targeting components of the neuromuscular junction, most commonly the acetylcholine receptor (AChR). These autoantibodies impair neuromuscular transmission by reducing the number and function of available AChR at the postsynaptic membrane, leading to fatigable muscle weakness. Clinically, MG presents with fluctuating weakness that predominantly affects the extraocular muscles, bulbar musculature, limb girdle, and respiratory muscles (1)(2). One of the most serious and potentially fatal complications of MG is myasthenic crisis, defined as an acute exacerbation of muscle weakness resulting in respiratory failure requiring mechanical ventilatory support. Myasthenic crisis occurs in approximately 15% to 20% of patients with MG and represents a major clinical emergency requiring intensive care. Alarmingly, it develops within the first two years of disease onset in approximately 74% of affected individuals, and in some cases, it may constitute the initial clinical presentation (3)(4). Due to the high morbidity, mortality risk, and resource demands associated with respiratory compromise in myasthenic crisis, prompt recognition and timely initiation of immunomodulatory therapies are essential. Among available options, Therapeutic Plasma Exchange (TPE) has emerged as an effective intervention to rapidly reduce circulating autoantibody levels, restore neuromuscular transmission, and improve respiratory function. As such, understanding the role and outcomes of TPE in managing myasthenic crisis is critical for optimizing patient care and improving prognostic outcomes in this high-risk population (5)(7).

The estimated incidence of myasthenia gravis is approximately 1 per 100,000 population. This condition is more commonly observed in individuals over the age of 50. Women are affected more frequently than men, although the disease can occur at any age. In females, symptom onset typically occurs at a younger age—around 28 years—whereas in males, it more frequently presents after the age of 60. According to the 2010 Indonesian Basic Health Research (RISKESDAS) report, the national incidence of myasthenia gravis was estimated to be 1 case per 100,000 population. Data collected from Dr. Cipto Mangunkusumo General Hospital, Jakarta, recorded a total of 94 diagnosed cases of myasthenia gravis during the period between 2010 and 2011 (4)(5).

The management of myasthenic crisis involves both general and specific therapeutic approaches. General management focuses on securing the airway and providing adequate ventilatory support, identifying and addressing precipitating factors, ensuring appropriate nutritional support, and preventing complications. In affected patients, airway protection is often achieved through endotracheal intubation and mechanical ventilation. Common triggers, such as infections, should be promptly treated with empirical antibiotic therapy. Nutritional support and proactive complication prevention are also essential components of comprehensive patient care (5)(6). Specific management strategies include the use of acetylcholinesterase inhibitors, therapeutic plasma exchange (TPE), intravenous immunoglobulin (IVIg), and immunosuppressive agents such as corticosteroids, azathioprine, cyclophosphamide, cyclosporine, mycophenolate mofetil, and tacrolimus. A retrospective study comparing the efficacy of TPE and IVIg in patients experiencing myasthenic crisis demonstrated that TPE was associated with better respiratory and functional outcomes (6).

Plasma exchange (PE) is a cornerstone therapeutic modality in the management of myasthenic crisis. It functions by removing circulating pathogenic substances, including autoantibodies and pro-inflammatory cytokines, thereby contributing to the improvement of the patient's clinical condition. In the context of a myasthenic crisis, early initiation of PE—ideally within 48 hours of symptom onset—is recommended to minimize the risk of complications and mortality. The standard protocol typically involves five to six sessions administered on alternate days, with each session exchanging approximately 1.0 to 1.5 plasma volumes. Clinical improvement is often observed after the third session, and

therapeutic benefits may persist for several weeks following the completion of treatment (7)(8).

## 2. Case Study

A 61-year-old male, weighing 60 kg, presented with acute onset dyspnoea that began one day prior to admission. The patient reported a five-month history of left-sided ptosis, characterized by diurnal fluctuation—worsening in the late afternoon and with physical exertion, and improving with rest. He had previously consulted an ophthalmologist; however, the ethology remained undetermined at that time. Two months prior to admission, the patient developed progressive dysphonia, difficulty in mastication, and dysphagia, for which he did not receive formal treatment. One month before presentation, he began experiencing diplopia, and four days prior to admission, his dysphagia had significantly worsened (8)(9).

The patient was evaluated at the neurology outpatient clinic of RSHAM Hospital, where electromyography (EMG) was performed on March 20, 2024, revealing a positive Harvey-Masland test—supportive of a diagnosis of myasthenia gravis. Despite the findings, the patient had not initiated treatment with pyridostigmine (Mestinon) and was admitted for observation. During hospitalization, the patient developed acute respiratory failure and was referred to the intensive care unit (ICU), where he underwent endotracheal intubation, mechanical ventilatory support, and central venous catheter placement. On the following day, therapeutic plasma exchange (TPE) was initiated, with a target plasma exchange volume of 1.0–1.5 times the patient's plasma volume, calculated based on body weight and haematocrit. A total of 3000 mL was exchanged using 5% albumin as the replacement fluid. Remarkably, six hours after the first TPE session, the patient demonstrated spontaneous breathing and was subsequently transferred to the general ward for continued care (10)(11).


Figure 1: Therapeutic Plasma Exchange (TPE) Procedure in a Patient with Myasthenic Crisis

## 3. Results

Plasma exchange (PE) is a therapeutic procedure in which plasma is separated from the patient’s blood and replaced with fresh frozen plasma (FFP), blood products, or plasma substitutes such as albumin, colloids, or other appropriate replacement fluids. The underlying rationale is that circulating pathogenic substances—such as toxins, immune complexes, or autoantibodies—accumulate within the plasma compartment; thus, their removal may provide significant clinical benefit. During the procedure, an estimated 1.0 to 1.5 times the patient's plasma volume is exchanged. Blood is circulated continuously through a filtration system at a flow rate of approximately 50 to 200 mL per minute, using filters with a pore diameter ranging from 0.2 to 0.6 micrometres. Each session typically lasts about 3 hours.

Based on current clinical guidelines, PE is usually administered in 5 to 7 sessions per standard therapeutic cycle. The procedure can be performed daily or on alternate days, depending on the clinical condition and therapeutic response (12)(13).

In this patient, therapeutic plasma exchange (TPE) was conducted as a single session with a target exchange volume of 1.5 times the estimated plasma volume. The procedure utilized 3 litters of 5% albumin as the replacement fluid, with 150 mL of processed blood volume, a total replacement volume of 1000 mL, no plasma blood product (PBP 0), and anticoagulation with heparin at a rate of 250 IU/hour. The patient demonstrated a prompt clinical response following the procedure (14)(15). Clinical improvement was evidenced by the return of spontaneous and comfortable respiration, allowing for successful weaning from mechanical ventilatory support. On physical examination, vital signs were stable, and neurological assessment revealed resolution of ptosis and normalization of motor strength. However, intermittent diplopia persisted. One week after TPE, the patient underwent percutaneous dilatational tracheostomy (PDT) and was subsequently transferred to the general medical ward for continued care (16)(17).

## 4. Conclusion

Myasthenic crisis is a life-threatening neurological emergency that requires immediate recognition and prompt intervention to prevent respiratory compromise. Therapeutic plasma exchange (TPE) remains a cornerstone in the acute management of myasthenic crisis due to its ability to rapidly remove pathogenic autoantibodies. In clinical practice, early initiation of TPE has been consistently associated with improved patient outcomes, including reduced duration of mechanical ventilation, shortened ICU stays, and faster recovery of neuromuscular function. The presented case highlights the critical importance of timely TPE administration, reinforcing its role as a first-line therapeutic modality in severe exacerbations of myasthenia gravis. From a scientific and medical standpoint, this case contributes valuable evidence to the growing body of literature supporting the efficacy of TPE in myasthenic crisis. It underscores the need for heightened clinical vigilance and early intervention protocols, which are essential for optimizing prognosis and guiding best practices in the management of neuromuscular emergencies.

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