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Case Report

Thymic Hyperplasia Radiologically Mimicking Thymoma in Acetylcholine Receptor Antibody–Positive Myasthenia Gravis Complicated by Post-Thymectomy Myasthenic Crisis Successfully Managed with Therapeutic Plasma Exchange: A Case Report from Bangladesh

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Abstract:

Myasthenia gravis (MG) is an autoimmune neuromuscular junction disorder commonly associated with thymic pathology. Distinguishing thymic hyperplasia from thymoma radiologically remains challenging, and post-thymectomy myasthenic crisis carries significant mortality risk.

A 34-year-old Bangladeshi man with AChR antibody–positive generalised MG underwent thymectomy for radiologically suspected thymoma; histopathology revealed thymic hyperplasia. Three weeks postoperatively he developed MGFA Class V crisis — ptosis, diplopia, dysarthria, dysphagia, neck and limb weakness (MRC 2/5), and type II respiratory failure requiring intubation and mechanical ventilation. Two sessions of centrifugal therapeutic plasma exchange (TPE) with citrate anticoagulation were performed via internal jugular access, exchanging ~2,500 mL per session with 5% albumin replacement. Following the second session, bulbar and ocular symptoms resolved, limb power improved, respiratory acidosis corrected, and ultimately patient was extubated and discharged ambulatory with pyridostigmine 360 mg/day and prednisolone 10 mg/day. This case highlights the imaging limitations in thymic pathology classification and demonstrates the life-saving utility of timely TPE in post-thymectomy myasthenic crisis, even in resource-limited settings.

Keywords: *Bangladesh, Mechanical ventilation; Myasthenia gravis; Myasthenic crisis; Therapeutic plasma exchange; Thymic hyperplasia; Thymectomy.*

Introduction

Myasthenia gravis (MG) is a chronic autoimmune disorder characterised by fluctuating, fatigable skeletal-muscle weakness resulting from antibody-mediated impairment of neuromuscular transmission. In most patients with generalised MG, autoantibodies are directed against the acetylcholine receptor (AChR), producing complement-mediated injury and a reduction in functional postsynaptic receptors[1,2]. International consensus guidance has refined the diagnostic, therapeutic, and crisis-management algorithms for MG over the past decade[3,4].

The thymus plays a central role in the immunopathogenesis of AChR-positive MG. Thymic abnormalities are common, with thymic hyperplasia and thymoma representing the two most clinically important entities[5,6]. Although contrast-enhanced computed tomography (CT) is routinely used to evaluate anterior mediastinal lesions, radiological differentiation between thymic hyperplasia and thymoma can be challenging because of overlapping imaging features, including soft-tissue density, focal enlargement, and occasionally calcification[6,7].

Thymectomy is an established therapeutic option in selected patients with generalised AChR-positive MG. The randomised MGTX trial demonstrated improved clinical outcomes and reduced corticosteroid requirements over a three-year follow-up[5]. Nevertheless, postoperative deterioration may occur, and myasthenic crisis remains a potentially fatal complication. Myasthenic crisis is defined as worsening respiratory or severe bulbar weakness producing respiratory failure that necessitates non-invasive or invasive ventilatory support[8,9].

Therapeutic plasma exchange (TPE) is widely used as a rescue therapy for severe MG exacerbations because it rapidly

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removes circulating pathogenic antibodies, complement components, and inflammatory mediators[10–12]. We report a case of radiologically suspected thymoma that proved on histopathology to be thymic hyperplasia and was subsequently complicated by post-thymectomy myasthenic crisis successfully treated with two sessions of TPE in a tertiary-care intensive care unit in Bangladesh.

Case Presentation

A 34-year-old Bangladeshi man presented with a one-year history of generalised AChR-positive MG. He had no history of hypertension, diabetes mellitus, ischaemic heart disease, thyroid dysfunction, or other autoimmune disorders, and reported no significant family or surgical history. Drug history was limited to pyridostigmine and prednisolone for MG, and no known drug allergies were reported. There was no recent infection, immunization, or exposure to ototoxic or neuromuscular-blocking medications in the perioperative period.

Symptoms began on 18 February 2025 with fluctuating ptosis and diplopia, progressing over several weeks to dysarthria, dysphagia, neck weakness, and generalised limb weakness. Generalised AChR-positive MG was diagnosed on 03 March 2025 on the basis of typical clinical features and a strongly positive serum AChR antibody titre (9.66 nmol/L; reference value < 0.5 nmol/L).

He was treated with pyridostigmine and oral corticosteroids; prednisolone was gradually tapered from 60 mg daily to a maintenance dose of 10 mg daily. He remained clinically stable for approximately one year. He was not receiving azathioprine or another long-term steroid-sparing immunosuppressive agent before surgery. Definitive thymectomy was deferred for approximately one year because of financial constraints—a relevant barrier to timely surgical care in resource-limited settings.

Contrast-enhanced chest CT on 05 May 2025 demonstrated a 4.4 × 2.6 cm mixed-density anterior mediastinal lesion with coarse calcification, radiologically suspicious for thymoma. On the basis of the radiological suspicion and established MG, an elective trans-sternal thymectomy was performed on 30 April 2026. His perioperative blood biochemistry reports were normal (table I).

Table I: Perioperative haematological and biochemical investigations

Parameter	30 Apr
Hb (g/dL)	13.2
WBC (×10⁹/L)	11.64
Platelets (×10⁹/L)	331
Creatinine (mg/dL)	0.63
Sodium (mmol/L)	141
Potassium (mmol/L)	4.03

Unexpectedly, histopathological examination demonstrated preserved thymic architecture with prominent cortical and medullary differentiation, reactive lymphoid follicles containing germinal centres, and Hassall corpuscles, without evidence of malignancy. The findings were diagnostic of thymic follicular hyperplasia rather than thymoma.

Approximately three weeks postoperatively, the patient developed progressive ocular, bulbar, respiratory, and limb weakness. He was admitted in ward on 21 May 2026 and transferred to the Medical Intensive Care Unit (MICU) next day because of impending respiratory failure.

On MICU admission, the patient was conscious but visibly fatigued and in respiratory distress. Neurological examination revealed bilateral ptosis, ophthalmoparesis with diplopia, dysarthria, severe dysphagia with pooled secretions, neck flexor weakness, and symmetrical generalised limb weakness with MRC grade 2/5 power in both upper and lower limbs. Deep tendon reflexes were preserved and sensory examination was normal. Single-breath count was 10 (normal ≥ 25). The constellation of findings was consistent with MGFA Class V myasthenic crisis, and endotracheal intubation with invasive mechanical ventilation was initiated for impending neuromuscular respiratory failure.

Serial serum AChR antibody measurements (Table II) confirmed persistent seropositivity, with titres declining during medical therapy. Preoperative urine culture and sensitivity testing on 04 March 2026 and 04 April 2026 showed no growth of pathogenic organisms. During the crisis, an infection workup—including chest radiography, blood cultures, urinalysis, and inflammatory markers—did not identify pneumonia, urinary tract infection, or systemic sepsis as a precipitating factor.

Table II: Serial serum AChR antibody titres (reference value < 0.5 nmol/L)

Date	Reporting laboratory	AChR antibody nmol/L)
12 Apr 2025	National Institute of Neurosciences (NINS), Dhaka	9.66
01 Apr 2026	Bangladesh Medical University, Dhaka	7.30
03 May 2026	Bangladesh Medical University, Dhaka	6.70

Arterial blood gas analysis prior to TPE demonstrated respiratory acidosis (pH 7.24; $PaCO_2$ 49 mmHg; HCO_3^- within normal range), consistent with acute ventilatory failure. Following TPE and ventilatory support, the pH normalised to approximately 7.37 with resolution of the respiratory acidosis.

Therapeutic Intervention:

Given the severity of the post-thymectomy myasthenic crisis—with bulbar involvement, ventilatory failure, and MRC grade 2/5 limb weakness—urgent TPE was initiated as rescue therapy. Two sessions of centrifugal apheresis with citrate anticoagulation were performed via an internal jugular

catheter. Each session exchanged approximately 2,500 mL of plasma using 5% human albumin as the replacement fluid (Table 3). Both procedures were well tolerated, with continuous haemodynamic monitoring and supplemental intravenous calcium to mitigate citrate-induced hypocalcaemia; no procedural or vascular-access complications were documented.

Concurrent supportive care included invasive mechanical ventilation with lung-protective settings, deep-vein thrombosis prophylaxis, stress-ulcer prophylaxis, careful fluid and electrolyte management, and continuation of oral pyridostigmine via the nasogastric tube. Intravenous immunoglobulin (IVIG) was considered but not administered, as TPE was preferred for its rapid onset of action and demonstrated efficacy in severe crises with prominent bulbar and respiratory involvement[10,11].

Table III: Technical details of the two therapeutic plasma exchange sessions

Parameter	Session 1	Session 2
Date	22 May 2026	24 May 2026
Vascular access	Internal jugular catheter	Internal jugular catheter
Modality	Centrifugal apheresis	Centrifugal apheresis
Exchange volume	~2,500 mL	~2,500 mL
Replacement fluid	5% albumin	5% albumin
Anticoagulation	Citrate	Citrate
Duration (approximate)	2.5 hours	2.5 hours
Procedural complications	None documented	None documented

Follow-up and Outcome:

Marked clinical improvement was observed after the second TPE session. Ptosis and diplopia improved, dysarthria and dysphagia resolved, limb power increased from MRC grade 2/5 to 4/5, single-breath count rose from 10 to 30, and respiratory acidosis resolved (Table 4). These improvements permitted successful weaning from mechanical ventilation and extubation on 25 May 2026.

The patient was discharged on the same day in an ambulatory and haemodynamically stable condition on pyridostigmine 360 mg/day (in divided doses) and prednisolone 10 mg/day, with planned outpatient review at two weeks and subsequent consideration of a steroid-sparing immunosuppressive agent. There was no residual bulbar weakness and no TPE-related complication at discharge.

Table IV: Neurological status before and after therapeutic plasma exchange

Parameter	Before TPE	After TPE
Ptosis	Present (bilateral)	Markedly improved
Diplopia	Present	Improved
Dysarthria	Present	Resolved
Dysphagia	Present	Resolved
Neck flexor weakness	Present	Resolved
Limb power (MRC)	2/5	4/5
Single-breath count	10	30
Deep tendon reflexes	Normal	Normal
Sensory examination	Normal	Normal
Mechanical ventilation	Required (intubated)	Successfully weaned and extubated

Table V: Timeline of patient's clinical course

Date	Clinical event
18 Feb 2025	Onset of fluctuating ptosis and diplopia.
03 Mar 2025	Generalised AChR-positive MG diagnosed on clinical and serological grounds; pyridostigmine and corticosteroid therapy initiated.
12 Apr 2025	Baseline serum AChR antibody titre: 9.66 nmol/L.
05 May 2025	Chest CT showed a 4.4 × 2.6 cm anterior mediastinal lesion suspicious for thymoma.
Mar 2025 – Apr 2026	Maintained on tapering corticosteroid (60 mg → 10 mg/day) and pyridostigmine; thymectomy deferred owing to financial constraints.
01 Apr 2026	Repeat AChR antibody titre: 7.30 nmol/L; preoperative workup unremarkable.
30 Apr 2026	Trans-sternal thymectomy performed; histopathology subsequently confirmed thymic hyperplasia without malignancy.
21 May 2026	Admission with worsening ocular, bulbar, limb, and respiratory weakness.
22 May 2026	Transferred to MICU; intubated for MGFA Class V crisis; first TPE session performed.
24 May 2026	Second TPE session performed; marked clinical improvement.
25 May 2026	Successfully extubated; discharged ambulatory without residual bulbar weakness.
Post-discharge	Outpatient follow-up at two weeks scheduled to assess sustained recovery.

Discussion:

This case illustrates two clinically important issues in the management of AChR-positive MG: the limitations of cross-sectional imaging in distinguishing thymic hyperplasia from thymoma, and the role of timely rescue therapy in post-thymectomy myasthenic crisis.

Radiological versus histopathological diagnosis of thymic lesions: In the present patient, CT findings strongly suggested thymoma because of an anterior mediastinal mixed-density lesion with coarse calcification. However, definitive histopathology revealed thymic follicular hyperplasia. This radiological–pathological discordance is well recognised: although calcification has been considered more typical of thymoma, both entities may overlap on conventional CT, particularly when hyperplastic thymic tissue is asymmetric, focal, or unusually dense[6,7]. Chemical-shift and dynamic contrast-enhanced magnetic resonance imaging, when available, can improve discrimination but do not replace tissue diagnosis. Histopathology remains the diagnostic gold standard, distinguishing the preserved corticomedullary architecture and germinal-centre–containing lymphoid follicles of hyperplasia from the neoplastic epithelial proliferation of thymoma[6,7].

Post-thymectomy myasthenic crisis : The patient's postoperative deterioration fulfilled criteria for myasthenic crisis[8,9], with severe bulbar weakness, single-breath count of 10, acute respiratory acidosis, and the requirement for endotracheal intubation. Reported precipitants of myasthenic crisis include intercurrent infection, surgery and anaesthesia, pregnancy, electrolyte disturbance, and offending drugs. Although infection is the most frequently identified trigger, no infectious source was identified in this patient. Surgical stress and the immunological perturbation that may follow thymectomy itself were the most plausible precipitants.

Choice of therapeutic plasma exchange: TPE and IVIG are the principal short-term immunomodulatory therapies for myasthenic crisis. Randomised data have shown comparable overall efficacy, but TPE may produce a faster onset of clinical benefit, particularly in patients with prominent bulbar or respiratory involvement[10]. The American Society for Apheresis assigns TPE in myasthenic crisis a Category I, Grade 1B recommendation, reflecting its position as a first-line therapy in severe disease[11,12]. In the present case, two sessions of centrifugal TPE with 5% albumin replacement produced a rapid and clinically meaningful improvement, supporting prompt initiation of TPE in patients with severe post-thymectomy crisis when appropriate apheresis infrastructure is available.

Equity-of-access and health-system considerations: An additional important aspect of this case is the approximately one-year delay between diagnosis and thymectomy, attributable to financial constraints. This reflects a practical barrier to timely surgical and multidisciplinary care that is common in many low- and middle-income settings. Although a single case cannot determine whether earlier thymectomy would have altered the postoperative course, this issue is highly relevant for health-system planning, financial protection, and equitable access to MG care.

Strengths and limitations

The strengths of this report include detailed clinical, serological, radiological, histopathological, and biochemical documentation across the patient's entire treatment trajectory; transparent reporting according to the CARE guideline; and contribution of regional data from Bangladesh, where published case-level descriptions of post-thymectomy crisis managed with TPE remain limited.

Limitations include the inherent constraints of a single case, the absence of formal validated outcome measures such as the Quantitative Myasthenia Gravis (QMG) score and the MG Activities of Daily Living (MG-ADL) scale, the lack of measurement of anti–muscle-specific tyrosine kinase (MuSK) and anti–lipoprotein receptor-related protein 4 (LRP4) antibodies, and a short period of post-discharge follow-up at the time of submission.

Conclusion

Thymic hyperplasia can radiologically mimic thymoma in AChR-positive MG, and definitive diagnosis requires histopathological confirmation. Post-thymectomy myasthenic crisis is a life-threatening complication that can occur even when the final pathology is benign hyperplasia rather than thymoma. Early recognition, intensive care monitoring, ventilatory support, and prompt initiation of TPE were central to the favourable outcome in this patient. This case contributes regional experience from Bangladesh and highlights the value of accessible apheresis services in the management of severe MG.

Learning Points

- Thymic hyperplasia can radiologically mimic thymoma in AChR-positive myasthenia gravis; histopathology remains the diagnostic gold standard.
- Post-thymectomy myasthenic crisis is a life-threatening complication and may occur even when final histopathology shows benign hyperplasia.
- Therapeutic plasma exchange can produce rapid neurological improvement and facilitate liberation from mechanical ventilation in severe MG exacerbations.
- In patients with severe bulbar or respiratory involvement, TPE may be preferred over IVIG for its faster onset of action.
- Financial and infrastructural barriers may delay definitive surgical and multidisciplinary care in resource-limited settings and should be considered in case planning and outcome interpretation.

Patient Perspective

"After the operation I suddenly became very weak and could not breathe properly. It was the most frightening time for me and my family. The doctors quickly admitted me to the intensive care unit and started plasma exchange. Within a few

days I felt much stronger, my speech and swallowing returned, and I could breathe on my own again. I am deeply grateful to the doctors, nurses, and the entire ICU team for their timely care."

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