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Thymopentin-Induced Myasthenia Gravis: A Case Report Highlighting Clinical Vigilance

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Myasthenia gravis (MG) is an autoimmune disorder of neuromuscular transmission in which the thymus plays a pivotal role in the breakdown of immune tolerance.[1] We report the case of a 75-year-old Chinese woman with a history of triple-negative breast cancer (TNBC) who developed MG following the administration of thymopentin. The patient was initially diagnosed with early-stage TNBC in 2017 and underwent a modified radical mastectomy followed by adjuvant chemotherapy with the AC-T regimen.

Because of persistent lymphopenia, intermittent thymopentin therapy was initiated in April 2019 (at 10 mg every other day, 14 doses per course), with courses administered every six months. During the first few days of each treatment cycle, the patient experienced transient dizziness and a sensation of heaviness, both of which resolved spontaneously without intervention. Notably, a clear temporal association was observed between thymopentin administration and the onset or exacerbation of neurological symptoms.

After a scheduled course of thymopentin in October 2023, the patient first developed intermittent right-sided ptosis. Subsequently, two episodes of marked clinical deterioration were documented. In April 2024, she experienced worsening ptosis accompanied by chewing weakness, and in October 2024, she developed progressive neck weakness and dysphagia. Importantly, each episode occurred shortly after the completion of a thymopentin treatment course.

Physical examination revealed fatigable weakness of the cervical, masticatory, swallowing, and limb muscles, accompanied by right-sided ptosis. The neostigmine test was positive. Repetitive nerve stimulation demonstrated a decremental response (Table 1). Serological testing was positive for anti–acetylcholine receptor (AChR) antibodies (titer 1:100, cell-based assay; reference for positivity ≥ 1:10) and anti-titin antibodies (titer 1:320). Additional findings included positive antinuclear antibodies (ANA) and anti–Ro-52 antibodies, weakly positive anti-SSA antibodies, elevated immunoglobulin G levels following intravenous immunoglobulin administration, and decreased complement C3 and thyroid-stimulating hormone levels. Chest computed tomography revealed no thymic abnormalities or tumors. Based on these findings, a diagnosis of MG was established.

The patient was treated with intravenous immunoglobulin, methylprednisolone, and pyridostigmine, resulting in marked clinical improvement. The quantitative MG (QMG) score decreased from 20 at admission to 10 at discharge. At follow-up in May 2025, she remained stable on low-dose prednisone and pyridostigmine, with a QMG score of 3 (Figure 1).

The epidemiological and pathophysiological relationship between MG and TNBC remains incompletely understood. Notably, approximately 10.8% of patients with MG develop extrathymic malignancies either before or after the onset of MG, with breast cancer—particularly TNBC—and lung cancer being the most frequently reported.[2] Patients with TNBC exhibit immune dysregulation that parallels key mechanisms involved in MG pathogenesis, including impaired immune homeostasis, a pro-inflammatory tumor microenvironment, and shared genetic susceptibility.[3] The development of MG following TNBC may occur through several mechanisms, such as immune checkpoint inhibitor therapy, treatment-related thymic or immune cell injury, or antigenic cross-reactivity between tumor antigens and components of the neuromuscular junction.

The thymus plays a central role in the pathogenesis of AChR–positive MG, where a breakdown of central T-cell tolerance leads to the production of pathogenic autoantibodies.[1,4] In early-onset MG, this process is largely driven by intrathymic germinal centers that generate high-affinity anti-AChR antibodies.[1] Thymic epithelial cells secrete immunomodulatory peptides, including thymosins such as thymosin α1, thymopoietin, and serum thymic factor, which are essential for the regulation of T-lymphocyte maturation, differentiation, and function.[5]

Notably, this patient developed MG five years after completing chemotherapy, with symptom onset closely following thymopentin administration and improvement observed after its discontinuation

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TABLE 1. Positive Results of Repetitive Nerve Stimulation.

Nerve	Muscle	Frequency (Hz)	CMAP (mV) (the 4th or 5th wave)	CMAP (mV) (the 1st wave)	Decay rate (%)	Result
Accessory (right)	Trapezius	1	3.14	3.60	13	Positive
		3	2.91	3.46	16	Positive
		5	3.04	3.39	10	Positive
		10	2.60	3.39	26	Positive
Facial (left)	Orbicularis oculi muscle	1	0.23	0.28	18	Positive
		3	0.21	0.26	19	Positive
		5	0.24	0.27	11	Positive
		10	0.26	0.27	7	Negative

CMAP, compound muscle action potential. The positive threshold: A decrement of > 10% in CMAP amplitude of the 4th or 5th compared to the 1st response.

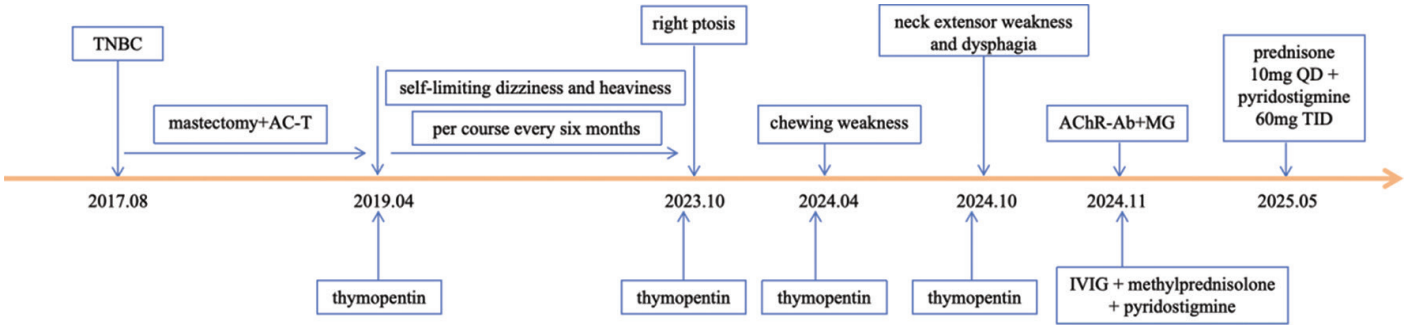

FIG. 1. This timeline outlines the progression of the patient's clinical condition following her initial diagnosis of TNBC.
TNBC, triple-negative breast cancer; MG, myasthenia gravis; IVIG, intravenous immunoglobulin.

in conjunction with MG-specific therapy. Clinical exacerbations consistently coincided with thymopentin treatment cycles, a pattern consistent with reports of thymic disease-related MG. Retrospectively, the transient dizziness and sensation of heaviness reported at the start of each thymopentin cycle since 2019 may represent immune-mediated prodromal neurological symptoms.

The presence of anti-titin antibodies—a specific marker of thymic or paraneoplastic autoimmunity—suggests underlying immune dysregulation potentially exacerbated by exogenous thymic hormones, even in the absence of thymoma. Exogenous thymic hormones may promote the survival or escape of autoreactive T-cell clones and modify the thymic microenvironment to favor autoantibody production, thereby explaining the observed temporal association. MG can arise from dysregulated thymic signaling even without structural thymic pathology.[6] Mechanisms involving HIF-1A and Th17/Treg imbalance provide a plausible theoretical framework for this case, and thymopentin may similarly interfere with these critical pathways that regulate T-cell fate and immune tolerance.

Additionally, the presence of ANA, anti–Ro-52, and anti-SSA antibodies indicates a baseline autoimmune predisposition, which may have lowered the threshold for developing drug-triggered MG. A history of TNBC further represents an independent risk factor for MG, potentially via shared immune dysregulation or paraneoplastic mechanisms.

This case underscores that thymopentin may trigger or exacerbate MG in cancer patients with pre-existing immune dysregulation. The consistent temporal association highlights a potential causal link and emphasizes the need for heightened clinical vigilance. Given their immunomodulatory effects, thymosin derivatives should be used with caution, and a careful benefit-risk assessment is warranted in oncology patients with underlying immune susceptibility to prevent autoimmune complications.

Informed Consent: Informed consent was obtained from the patient for publication of this case report.

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