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Olfactory Dysfunction in Patients With Acetylcholine-Receptor-Antibody-Positive Myasthenia Gravis

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Background and Purpose Myasthenia gravis (MG) is an antibody-mediated disease characterized by fluctuating muscle weakness and fatigue due to impaired neuromuscular junction transmission. Although primarily considered a motor dysfunction disorder, there is emerging evidence that MG can also present with nonmotor symptoms, including olfactory impairment. However, the prevalence and clinical relevance of olfactory dysfunction in MG remain poorly understood. This study compared olfactory function between MG patients and healthy controls with the aim of identifying clinical factors associated with olfactory impairment in MG.

Methods Acetylcholine receptor (AChR)-antibody-positive MG patients and healthy controls were recruited from a single-center outpatient clinic. Olfactory function was assessed using the KVSS II (Korean version of the Sniffin' Sticks II) test, comprising odor threshold, discrimination, and identification subtests. We compared olfactory function and clinical factors between MG patients and healthy controls after adjusting and matching for age and sex.

Results This study enrolled 51 MG patients and 43 healthy controls. Logistic regression analyses revealed that the MG patients had a significantly increased risk of olfactory dysfunction (odds ratio=3.6, 95% confidence interval=1.4−9.1, p=0.008), which remained after adjusting for age and sex. Among the MG patients, those with olfactory dysfunction were older (p=0.002) and had lower AChR antibody titers (p=0.029).

Conclusions Olfactory dysfunction was significantly more prevalent in the MG patients than in the healthy controls. These findings highlight the need for further research into the underlying mechanisms and potential clinical implications of olfactory impairment in MG.

Keywords myasthenia gravis; olfactory impairment; smell dysfunction; neurologic autoimmune disease.

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INTRODUCTION

Myasthenia gravis (MG) is an antibody-mediated disease characterized by fluctuating weakness and fatigue of voluntary muscles due to impaired neuromuscular junction transmission. MG is primarily caused by autoantibodies targeting components of the neuromuscular junction, most commonly the acetylcholine receptor (AChR).[1] While MG is traditionally regarded as a disease restricted to motor dysfunction, there is emerging evidence that it can also involve nonmotor symptoms such as cognitive deficits, autonomic dysfunction, and olfactory impairment.[2-4] Although less studied, these nonmotor symptoms might be useful for understanding the broader pathophysiological mechanisms of MG and their impact on the quality of life of affected patients.

Olfactory dysfunction is a recognized manifestation in several neurological and autoimmune conditions such as Parkinson's disease and systemic lupus erythematosus. The presence of olfactory dysfunction in MG has been reported previously,[3,5,6] but its prevalence

and clinical significance remain poorly understood.

This study aimed to characterize olfactory dysfunction in MG patients by comparing their olfactory function with that of healthy controls, and to identify the clinical characteristics associated with olfactory dysfunction in MG. By exploring this understudied component of MG, we hoped to contribute to a more-comprehensive understanding of the disease, with potential implications for its classification and management.

METHODS

Study participants and data collection

MG patients and healthy controls who visited the outpatient clinic of Severance Hospital between 2017 and 2024 were recruited for this study. MG was diagnosed based on the presence of at least one of the following signs or symptoms of muscle fatigue: 1) decrease of 10% or more in the compound muscle action potential in response to a train of five low-frequency supramaximal repetitive nerve stimuli, 2) positivity for serum autoantibodies associated with MG, or 3) positivity in pharmacological tests using an acetylcholinesterase inhibitor. Those in the MG group had been diagnosed with and were currently being treated for AChR-antibody-positive MG. The healthy control group comprised volunteer participants with no past medical history of MG or its symptoms. The following exclusion criteria were applied while enrolling subjects for both the MG and healthy control groups: 1) aged 18 years or younger, 2) cognitive impairment, or 3) a condition that could affect olfactory function, such as chronic rhinosinusitis and Parkinson's disease. We excluded both MG patients and healthy controls with cognitive impairment as determined by a score of 27 or lower in the Mini-Mental Status Examination (MMSE).

After enrollment, the following further information about the MG patients was collected by retrospectively investigating their electronic medical records: ages at symptom onset and diagnosis, Myasthenia Gravis Foundation of America (MGFA) clinical classification at diagnosis, repetitive nerve stimulation test (RNST) results, AChR antibody titer at diagnosis, MG Activities of Daily Living (MG-ADL) score, and ongoing treatment for MG.

Matching

Previous studies have revealed that age and sex affect olfactory function.[7] We therefore matched the stratified age groups and sex between the MG patients and the healthy controls at a one-to-one ratio. Age was stratified into five groups: 20–29, 30–39, 40–49, 50–59, and ≥60 years. Participants who could not be matched due to unequal group sizes were excluded. This matching method minimized the potential confounding effects of age and sex in the comparisons of olfactory function.

Assessment of olfactory function

Olfactory function was measured in each participant using the Korean version of the Sniffin' Sticks (KVSS) II test.[7] This test is a semiobjective assessment tool utilizing felt-tip pens filled with liquid odorants. The original Sniffin' Sticks test was first developed by Hummel in Germany, and it has been culturally adapted and validated for Korean populations.[8]

The KVSS II test evaluates olfactory function using three subtests: odor threshold test, odor discrimination test, and odor identification test. The odor threshold test uses a series of 16 pens containing n-butanol at increasing concentrations. The lowest concentration at which n-butanol is reliably detected by the subject is defined as their odor threshold. The odor discrimination test consists of 16 trials. In each trial, the subject is presented with three pens, two of which have the same odor and one has a different odor. This subtest evaluates the subject's ability to discriminate different odors. The odor identification test used 16 pens containing common odors, and the subject is asked to identify the odor from a list of options. Each subtest has a maximum score of 16, and the threshold-discrimination-identification (TDI) score calculated as the sum of the scores in the three subtests is used to quantify the global olfactory function of the subject. Subjects with TDI scores of <30 are defined as having olfactory dysfunction: scores of 23–30 are defined as hyposmia and scores of <23 are defined as anosmia.[7,8]

Statistical analyses

Statistical analyses were performed using the Statistical Package for the Social Sciences Statistics software (version 27, IBM Corp.). Student's *t*-test was used to compare continuous variables between groups, with the chi-square test used for categorical variables and the Mann–Whitney U test for ranked variables such as the MGFA clinical classification. Univariate regression analyses were performed to assess the relationships between variables, and multivariate regression analysis with purposeful variable selection was used to adjust for known confounders, including age and sex. The adequacy of the logistic regression model was assessed using the likelihood ratio test for overall significance, the Hosmer–Lemeshow goodness-of-fit test for calibration, the variance inflation factor for multicollinearity, and the area under the receiver operating characteristic curve for classification accuracy.

Ethical approval and informed consent

This study was approved by the Severance Hospital Institutional Review Board (Approval No. 4-2017-0292), and was

conducted in accordance with the Declaration of Helsinki. The requirement to obtain informed consent from the participants was waived due to the retrospective design of the study.

RESULTS

Study participants

This study enrolled 51 MG patients and 43 healthy controls (Fig. 1). Screening based on MMSE scores did not result in the exclusion of any participants in either the MG or healthy control group. Females constituted 34 (66.7%) of the 51 MG patients and 28 (65.1%) of the 43 healthy controls. In both groups the most common age group was 50–59 years, with an age of 46.1±15.5 years (mean±standard deviation) in the MG group and 44.7±14.0 years in the healthy group (Supplementary Table 1 in the online-only Data Supplement).

In the MG group, the onset age was 33.7±15.7 years and the disease duration at enrollment was 143.7±114.0 months. Twenty (39.2%) patients had thymoma. The most common MGFA clinical classification at diagnosis was class I (*n*=22, 43.1%), followed by class II (*n*=20, 39.2%). Most of the patients (*n*=38, 74.5%) were taking prednisolone at enrollment, while 30 (58.8%) were taking other oral immunosuppressive agents and 17 (33.3%) were taking pyridostigmine.

Comparative analysis of olfactory function of MG patients and healthy controls

The TDI score was 29.8±5.4 in the MG patients and 33.3±4.8 in the healthy controls. Olfactory dysfunction was observed in 26 (51.0%) of the 51 MG patients and 10 (23.3%) of the 43 healthy controls. Due to the results obtained in previous studies of olfactory function, we first compared the risk of olfactory dysfunction by using logistic regression analyses adjusted for age and sex. As indicated in Table 1, the risk of olfactory dysfunction was 3.6-fold higher in the MG patients than in the healthy controls (95% confidence interval=1.4–9.1, *p*=0.008). After matching the sex and the stratified age groups, 39 patients remained in each group. The demographic and clinical characteristics of the matched groups are presented in Supplementary Tables 1 and 2 (in the on-

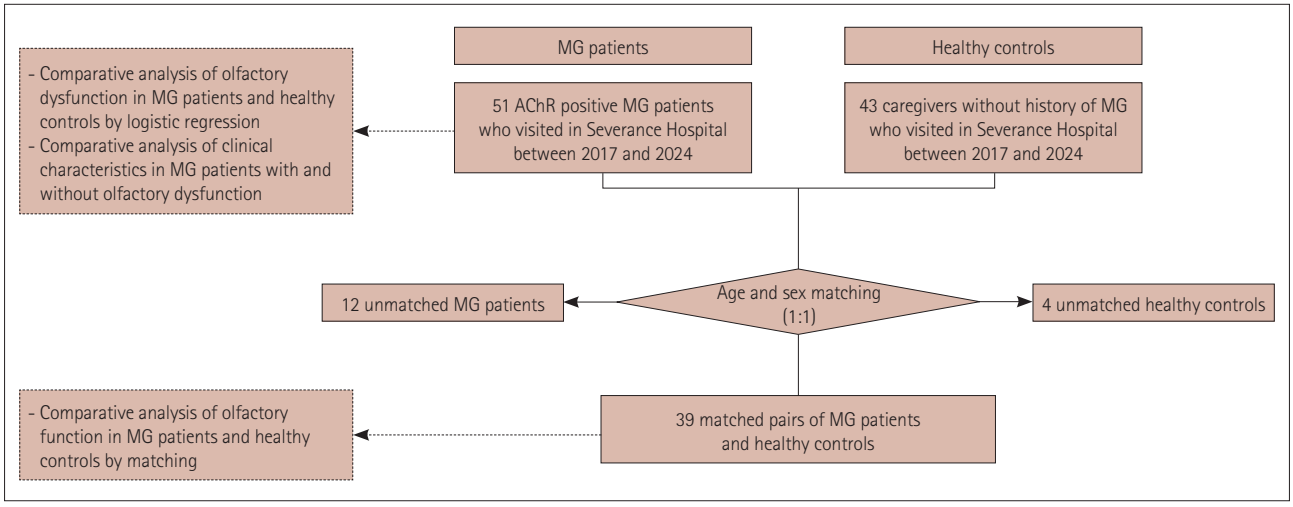

Fig. 1. Flowchart of participant enrollment. AChR, acetylcholine receptor; MG, myasthenia gravis.

Table 1. Risk of olfactory dysfunction in logistic regression analyses adjusted for age and sex

Variable	Univariate analysis*			Multivariate analysis†		
	OR	95% CI	*p*	OR	95% CI	*p*
Sex (male as a reference)	0.862	0.360–2.065	0.739	0.789	0.303–2.054	0.627
Age	1.041	1.010–1.074	0.009	1.043	1.010–1.077	0.010
MG	3.432	1.402–8.404	0.007	3.560	1.400–9.053	0.008

*In the univariate logistic regression analyses, the likelihood ratio test revealed that both MG (χ^2=7.79, *p*=0.005) and age (χ^2=7.27, *p*=0.007) were significantly associated with thymoma, whereas sex was not (χ^2=0.11, *p*=0.739). The Hosmer–Lemeshow goodness-of-fit test indicated an adequate fit for all univariate models (sex: χ^2=0.00, df=8, *p*>0.999; age: χ^2=8.19, df=8, *p*=0.416; MG: χ^2=0.00, df=8, *p*>0.999); †In the multivariate analysis, the overall model was statistically significant (likelihood ratio: χ^2=15.11, df=3, *p*=0.002), indicating that at least one predictor contributed significantly to the model. The Hosmer–Lemeshow test confirmed an adequate model fit (χ^2=8.10, df=8, *p*=0.424). No evidence of multicollinearity was observed among the independent variables, with variance inflation factors of 2.58 for sex, 3.30 for age, and 2.05 for MG. The model showed an accuracy of 73.4% and an area under the receiver operating characteristic curve of 0.726.
CI, confidence interval; MG, myasthenia gravis; OR, odds ratio.

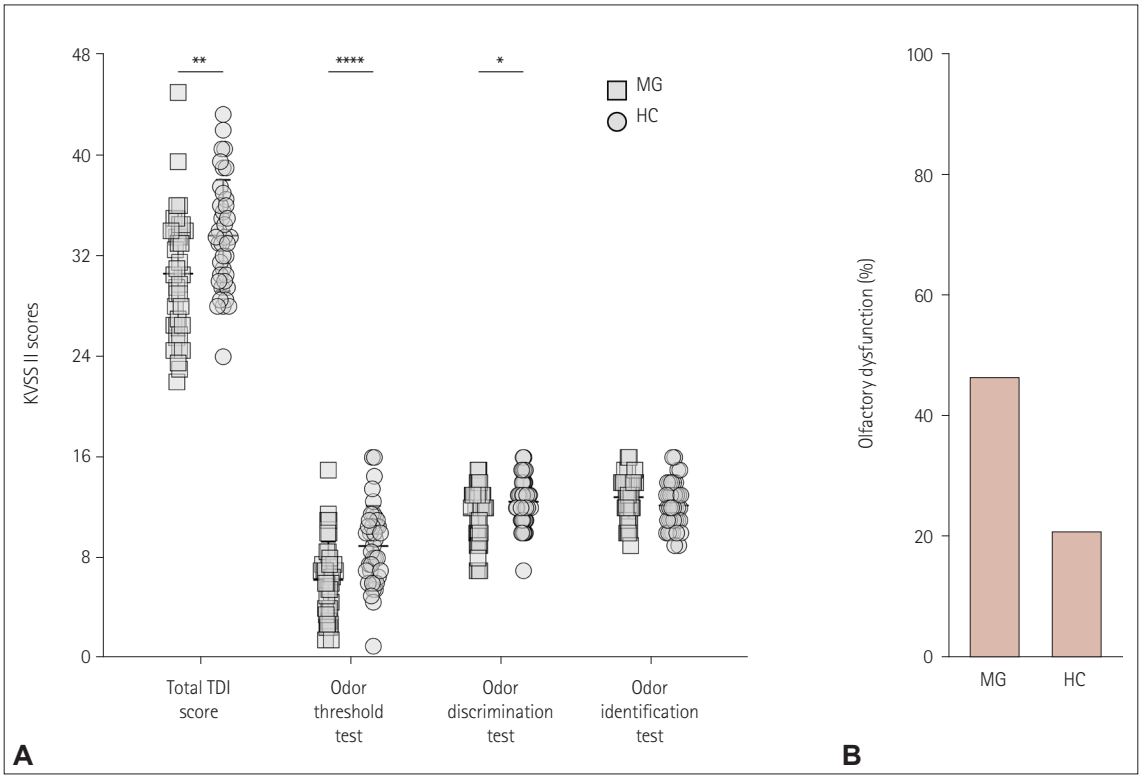

Fig. 2. Comparison of olfactory function between MG patients and HC after age and sex matching. A: Total and subtest scores in the Korean version of the Sniffin' Sticks (KVSS) II test. B: Proportion of subjects with olfactory dysfunction in each group. *p<0.05; **p<0.01; ***p<0.001; ****p<0.0001. HC, healthy control; MG, myasthenia gravis; TDI, threshold-discrimination-identification.

line-only Data Supplement). After matching, the following scores remained significantly lower for the MG patients than the healthy controls: total TDI score (30.6±4.9 vs. 33.5±4.4, p=0.002), odor threshold test (6.3±3.0 vs. 9.0±3.2, p<0.001), and the odor discrimination test (11.5±2.2 vs. 12.5±1.9, p=0.034) (Fig. 2A), with corresponding effect sizes (Cohen's d) of -0.648, -0.867, and -0.482, respectively, indicating moderate-to-large group differences for the total TDI score and odor threshold test, and a small-to-moderate difference for the odor discrimination test. Among the matched participants, 18 (46.2%) MG patients and 8 (20.5%) healthy controls were diagnosed with olfactory dysfunction (p=0.030) (Fig. 2B).

Clinical characteristics of MG patients with and without olfactory dysfunction

We further compared the clinical characteristics between MG patients with and without olfactory dysfunction. The MG patients with olfactory dysfunction were significantly older (52.6±14.8 years vs. 39.3±13.3 years, p=0.002) and had a higher onset age (38.6±18.6 years vs. 28.5±10.0 years, p<0.001). Late-onset MG patients (onset age ≥50 years) were more likely to have olfactory dysfunction. Additionally, MG patients with olfactory dysfunction had a significantly lower AChR antibody titer at diagnosis (6.9±4.7 nmol/L vs. 10.2±5.8 nmol/L, p=0.029) (Table 2). The prevalence of thymoma did not differ significantly between the two groups (38.5% vs. 40.0%, p=0.910). There were also no significant differences between the two groups in sex distribution, disease duration, history of a myasthenic crisis, MGFA classification at diagnosis, RNST results at diagnosis, MG-ADL score at enrollment, or the type of treatment received.

DISCUSSION

This study addressed olfactory dysfunction in patients with MG by comparing olfactory function between MG patients and healthy controls. Olfactory dysfunction diagnosed using the KVSS II test was more prevalent in patients with MG than in the healthy controls. Furthermore, among the MG patients, those with olfactory dysfunction were more likely to have a higher age, higher onset age, and lower AChR antibody titer at diagnosis.

MG is an antibody-mediated disease, and the most common target of pathogenic autoantibodies in MG is the α1 subunit of the nicotinic AChR, which is known to be highly specific to the neuromuscular junction. However, nonmotor symptoms of MG patients such as autonomic dysfunction,

Table 2. Clinical characteristics of MG patients with and without olfactory dysfunction

	MG with olfactory dysfunction (*n*=26)	MG without olfactory dysfunction (*n*=25)	Total (*n*=51)	*p*
Sex				0.428
Male	10 (38.5)	7 (28.0)	17 (33.3)	
Female	16 (61.5)	18 (72.0)	34 (66.7)	
Age (yr)	52.6±14.8	39.3±13.3	46.1±15.5	0.002
Onset age (yr)	38.6±18.6	28.5±10.0	33.6±15.7	<0.001
Onset age group				0.032
Early onset (onset age <50 years)	18 (69.2)	24 (96.0)	42 (82.4)	
Late onset (onset age ≥50 years)	8 (30.8)	1 (4.0)	9 (17.6)	
Disease duration (month)	163.4±135.5	123.2±84.1	143.7±114.0	0.211
Presence of thymoma	10 (38.5)	10 (40.0)	20 (39.2)	0.910
History of myasthenic crisis	4 (15.4)	9 (36.0)	13 (25.5)	0.091
Decremental response ≥10% during repetitive stimulation at diagnosis				
Adductor digiti minimi	9 (36.0)	12 (48.0)	21 (41.2)	0.390
Flexor carpi ulnaris	16 (64.0)	18 (72.0)	34 (66.7)	0.544
Orbicularis oculi	17 (68.0)	16 (64.0)	33 (64.7)	0.765
Nasalis	19 (6.0)	18 (72.0)	37 (72.5)	0.747
Trapezius	16 (64.0)	16 (64.0)	32 (62.7)	>0.999
AChR antibody titer at diagnosis (nmol/L)	6.9±4.7	10.2±5.8	8.5±5.5	0.029
MGFA classification at diagnosis				0.753
Class I	11 (42.3)	11 (44.0)	22 (43.1)	
Class II	12 (46.2)	8 (32.0)	20 (39.2)	
Class III	1 (3.8)	4 (16.0)	5 (9.8)	
Class IV	0	0	0	
Class V	2 (7.7)	2 (8.0)	4 (7.8)	
MG-ADL score at enrollment	2.0±2.2	1.9±1.8	1.9±2.0	0.830
Treatment at enrollment				
Pyridostigmine	9 (34.6)	8 (32.0)	17 (33.3)	0.843
Prednisolone	20 (76.9)	18 (72.0)	38 (74.5)	0.687
Azathioprine	2 (7.7)	4 (16.0)	6 (11.8)	0.357
Cyclosporine	1 (3.8)	0	1 (2.0)	0.322
Tacrolimus	10 (38.5)	12 (48.0)	22 (43.1)	0.492
Mycophenolate mofetil	1 (3.8)	0	1 (2.0)	0.322

Data are mean±standard deviation or *n* (%) values.
AChR, acetylcholine receptor; MG, myasthenia gravis; MG-ADL, MG Activities of Daily Living; MGFA, Myasthenia Gravis Foundation of America.

cognitive deficits, and olfactory dysfunction are consistently reported.[3] In 2012, Leon-Sarmiento et al.[6] identified profound olfactory dysfunction in MG patients using the University of Pennsylvania Smell Identification Test and the Picture Identification Test. A subsequent study conducted in the Republic of Türkiye found similar results using the Sniffin' Sticks test.[5] However, to the best of our knowledge, the present study is the first to investigate olfactory dysfunction in East Asian MG patients, and to compare clinical characteristics between MG patients with and without olfactory dysfunction.

The mechanisms underlying nonmotor symptoms in MG, including olfactory dysfunction, have not been clearly elucidated. Several hypotheses have been proposed, one of which is that an increase in systemic cytokines observed in MG exerts widespread effects that influence not only the neuromuscular junction but also other systems throughout the body. For example, a recent study found that serum interleukin-6 (IL-6) was elevated in MG patients.[9,10] Previous studies of olfactory function have found the proinflammatory cytokine IL-6 to be associated with olfactory dysfunction not only in chronic rhinosinusitis and postinfectious symptoms, but also in normal aging.[11-15] Similarly, increased proinflammatory cytokines may contribute to age-related hyposmia in MG patients. On the other hand, since MG is an antibody-

mediated autoimmune disease, autoantibodies and their inflammatory responses might directly affect the olfactory function of affected patients. Although controversial, previous studies have found that some patients with MG showed elevation of AChR antibodies as well as cellular abnormalities in their cerebrospinal fluid.[16] The heterogeneity of AChR antibodies may be related to this nonmotor involvement of MG.[17] Additionally, cholinergic alterations in MG might also affect olfactory function. Similar to neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease, which have well-known associations with olfactory dysfunction, there is some evidence that MG is also associated with altered cholinergic pathways in the central nervous system.[2,18]

The findings of this study also suggest that there are differences between MG patients with and without olfactory dysfunction. Specifically, olfactory dysfunction was found to be more common in MG patients who are older, have late-onset disease, and present with lower AChR antibody titers at diagnosis. As reported previously, olfactory function naturally declines with age, and hence associations with age and onset age are expected.[15] However, the observed association between a lower AChR antibody titer at diagnosis and olfactory dysfunction warrants further investigation. This phenomenon might be attributable to an age difference between the study groups. Some previous studies found that the AChR antibody titer tended to be lower in late-onset MG patients than early-onset MG patients.[19,20] However, the association between age and AChR antibody titer has been challenged by several reports of different results. Overall, these findings highlight the need for future studies exploring nonmotor symptoms in MG, which may ultimately contribute to a refined subclassification of the disease based on such features.

This study had several limitations. First, no objective rhinological assessment was performed before enrolling participants. Since olfactory dysfunction due to various conditions such as chronic rhinitis, recent upper respiratory infections, and smoking is common, future studies should consider applying strict and objective exclusion criteria for participants with rhinological disorders. Second, the MG patients in this study were already undergoing MG treatment, including prednisolone, pyridostigmine, or other immunosuppressive agents. These medications are not known to exert any direct effects on olfactory function, but they might indirectly influence it such as by promoting secretion from the olfactory mucosa. Further studies on treatment-naïve patients are also necessary. Third, this study had a small sample. It was not possible to enroll as many participants as anticipated due to the low prevalence of MG. Although efforts were made to account for known confounders using multiple statistical methods such as regression analysis and matching, a larger cohort would provide results that are more robust and generalizable. Future studies with larger samples are essential to validate these findings and to confirm the relationships between olfactory dysfunction and the clinical characteristics of MG.

In summary, this study found that some patients with MG exhibit impaired olfactory function relative to healthy controls. Furthermore, the MG patients with olfactory dysfunction were found to be older and have a lower AChR antibody titer than those without olfactory dysfunction. These findings support the perspective that MG can exert effects beyond the neuromuscular junction. Further research into nonmotor symptoms might provide valuable insights into the pathophysiology of MG and contribute to advancements in its novel classification and treatment.

Supplementary Materials

The online-only Data Supplement is available with this article at https://doi.org/10.3988/jcn.2025.0242.

Availability of Data and Material

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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