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ORIGINAL RESEARCH

Association Between Serum Anti-Acetylcholine Receptor (AChR-Ab) Titers and Clinical Outcomes in Patients with Ocular-Onset Myasthenia Gravis: An Academic Retrospective Cohort Study

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Background: Acetylcholine receptor antibodies (AChR-Ab) are established biomarkers in myasthenia gravis (MG). In ocular MG (OMG), their presence has been linked to disease severity, treatment response, and progression to generalized MG (GMG); however, the relevance of AChR-Ab titers or seropositivity to ocular manifestations remains uncertain.

Objective and Method: We retrospectively reviewed OMG patients treated at Rajavithi Hospital (2009–2024). AChR-Ab titers were measured using enzyme-linked immunosorbent assay (ELISA). Clinical and demographic factors associated with AChR-Ab seropositivity (AChR-Ab$^+$) and their titers were evaluated using logistic and linear regression analyses.

Results: Among the 111 patients with OMG, 51 patients (45.9%) were AChR-Ab$^+$ and 60 patients (54.1%) were AChR-Ab-seronegative (AChR-Ab$^-$). The AChR-Ab$^+$ group demonstrated a trend toward younger age compared to the AChR-Ab$^-$ group (43.82 ± 6.55 vs 46.53 ± 7.71 years; p = 0.051). AChR-Ab$^+$ patients were more likely to present with diplopia, a higher risk of progression to GMG, and more prevalence of thymoma, whereas ptosis was commonly observed in the AChR-Ab$^-$ group. Gender distribution, autoimmune comorbidities, the use and dosages of pyridostigmine or prednisolones were comparable between groups. Advanced age at onset was associated with lower likelihood of AChR-Ab$^+$ (OR 0.922; 95% CI: 0.864–0.983, p = 0.014). Diplopia (OR 5.394; 95% CI: 1.280–22.723, p = 0.022), progression to GMG (OR 4.640; 95% CI: 1.155–18.639, p = 0.031), and thymoma (OR 9.978; 95% CI: 2.094–47.547, p = 0.004) were positively associated with AChR-Ab$^+$ status. Regarding AChR-Ab titers, the presence of thymoma (β =1.556; 95% CI: 0.353–2.761, p = 0.012) was independent predictors, while titers declined with age, most pronounced after 50 years.

Conclusion: Serostatus of acetylcholine receptor antibodies may be useful for identifying MG patients at higher risk of generalization or thymoma, while antibody titers may provide complementary information, particularly in relation to thymoma, rather than serving as direct markers of disease severity or ocular manifestations.

Keywords: acetylcholine receptor antibodies, seropositivity, AChR-Ab titers, ocular myasthenia gravis, thymoma, ocular manifestations

Introduction

Myasthenia gravis (MG) is an autoimmune neuromuscular disorder characterized by fluctuating muscle weakness due to impaired neuromuscular transmission. The majority of MG cases are associated with autoantibodies targeting the acetylcholine receptor (AChR-Ab), which lead to postsynaptic dysfunction at the neuromuscular junction.[1] While Muscle-Specific Kinase (MuSK) and Low-Density Lipoprotein Receptor-Related Protein 4 (LRP4) antibodies define distinct MG subtypes, MuSK positivity is typically associated with severe bulbar and respiratory weakness,[2] whereas LRP4 positivity is more often linked to milder or predominantly ocular presentations.[3]

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Among the methods for detecting AChR-Ab, enzyme-linked immunosorbent assay (ELISA) is widely used due to its accessibility and cost-effectiveness. However, ELISA has relatively lower sensitivity, particularly in patients with low antibody levels or atypical clinical presentations. In contrast, cell-based assays (CBAs), particularly those using live cells expressing clustered acetylcholine receptors, offer markedly higher sensitivity and improved diagnostic accuracy.[4] Despite these advantages, CBAs are less commonly used in routine clinical practice due to their higher cost, limited accessibility, and the need for specialized laboratory facilities.

The relationship between AChR-Ab titers and MG severity has been investigated with inconsistent findings.[5,6] AChR-Ab titers are influenced by demographic and clinical factors, including age at onset, sex, thymoma, concomitant autoimmune diseases, and racial background, which may modulate disease phenotype.[7–9] Higher AChR-Ab titers have been associated with poorer treatment response and an increased likelihood of generalization to generalized MG (GMG).[8,9] Their relevance to ocular phenotypes is unclear, though AChR-Ab seropositivity (AChR-Ab$^+$) has been observed in patients with both ptosis and diplopia.[7,10,11] A Thai cohort of GMG patients reported no significant association between antibody titers and clinical characteristics.[12] Treatment interventions—such as pyridostigmine, corticosteroids, plasma exchange, and thymectomy—can further modify antibody levels over time.[11,13] These factors contribute to heterogeneity across studies, limiting the generalizability of antibody–phenotype correlations.

This study therefore aims to evaluate the prognostic value of AChR-Ab serostatus and titers in OMG, and their associations with clinical features, and long-term outcomes using real-world data from Rajavithi Hospital.

Materials and Methods

Patient Selection and Baseline Characteristic

A retrospective cohort study was conducted on patients diagnosed with OMG who underwent testing for AChR-Ab between January 1, 2009, and December 31, 2024, at Rajavithi Hospital, Bangkok, Thailand. Inclusion criteria encompassed patients aged 18 to 80 years with a confirmed diagnosis of OMG based on the criteria established by Osserman and Genkins,[1] which required the presence of persistent extraocular muscle weakness, such as diplopia, ptosis, or both. In addition, patients were required to fulfill at least one of the following laboratory diagnostic criteria: (1) a positive of AChR-Ab, (2) abnormal findings on single-fiber electromyography (SFEMG), (3) a positive clinical response to edrophonium chloride (Tensilon test), or (4) a positive result on repetitive nerve stimulation (RNS) testing. Patients were excluded if they had incomplete clinical records, uncertain dates of symptom onset or treatment initiation, a follow-up period of less than 24 months, or absence of documented baseline AChR-Ab test results.

Data Collection

Data were collected from electronic medical records, including demographic including age at diagnosis, gender, baseline severity of OMG, disease onset. Clinical characteristics encompassed the duration of ocular symptoms, type of symptoms involvement (ptosis, diplopia, both or subsequent generalization status documented during follow-up), the presence of thymic abnormality, preexisting autoimmune diseases, or systemic vascular diseases.

Diagnostic tests including SFEMG, RNS, and the Tensilon test. Laboratory findings included AChR-Ab status categorized as AChR-Ab$^+$ or Acetylcholine Receptor Antibody-seronegative (AChR-Ab$^-$) and its titers. The presence of AChR-Ab was evaluated and collected from electronic medical records at initial presentation or as early as feasible after symptom onset as part of the diagnostic and prognostic workup using ELISA. The cutoff value for AChR-Ab$^+$ was defined according to the laboratory reference standard. A positive result was defined according to the manufacturer's reference range, typically using a cutoff value of > 0.5 nmol/L.

Treatment-related information included medications administered (such as pyridostigmine, corticosteroids, and immunosuppressants). Clinical outcomes based on treatment response and disease progression and baseline characteristics were compared between AChR-Ab$^+$ and AChR-Ab$^-$ patients to determine associations with seropositivity and antibody titers.

The primary outcome was to examine clinical, demographic, and treatment-related predictors of AChR-Ab$^+$ status, the secondary outcome was to evaluate factors associated with AChR-Ab titers. Covariates included age at onset, sex, presenting

ocular symptoms (isolated ptosis, diplopia, or both), thymic pathology, preexisting autoimmune diseases, and treatment -related factors variables.

Ethical Approval

The study was approved by the Rajavithi Hospital Research Ethics Committee (certificate number 162/68) and conducted in accordance with the Declaration of Helsinki. Informed consent was obtained and all data were de-identified to maintain confidentiality.

Statistical Analysis

Descriptive statistics will summarize patient demographics, clinical characteristics, and treatment details. Continuous variables, Independent t-tests or Mann–Whitney *U*-tests were used as appropriate. Categorical variables, Chi-square or Fisher's exact tests were employed as appropriate. Comparisons between AChR-Ab$^+$/AChR-Ab$^-$ were performed using the Student's *t*-test or Mann–Whitney *U*-test for continuous variables and the chi-square or Fisher's exact test for categorical variables. Logistic regression models were employed to identify factors associated with AChR-Ab$^+$. Clinical variables were selected based on clinical relevance and a univariable analysis threshold of $p < 0.20$. Variables meeting this criterion were included in multivariable logistic regression models to identify independent predictors of AChR-Ab$^+$ status, with adjusted odds ratios (OR) and corresponding 95% confidence intervals (CIs) reported. Multivariable linear regression models were applied to determine predictors of AChR-Ab titers. A p-value of < 0.05 was considered statistically significant. All analyses were performed using a standard statistical software package.

Results

Clinical Characteristics of OMG Patients

In total, 182 patients were diagnosed with OMG within this timeframe. Seventy-one were excluded. Thirty-two patients were excluded due to absence of baseline AChR-Ab testing, 25 patients were incomplete medical records, 14 were insufficient follow-up duration. In all, 111 OMG patients who fulfilled the inclusion criteria were enrolled and subsequently included in the final analysis of the study.

Table 1 presents the baseline characteristics of OMG patients stratified by AChR-Ab serostatus. Fifty-one patients (45.9%) were AChR-Ab$^+$, while 60 patients (54.1%) were AChR-Ab$^-$. AChR-Ab$^+$ patients were slightly younger at onset than AChR-Ab$^-$, without a statistical significance (43.82 ± 6.55 vs 46.53 ± 7.71 years; $p = 0.051$). Thymoma was notably observed in AChR-Ab$^+$ group ($p = 0.005$), while ptosis occurred more frequently in the AChR-Ab$^-$ group ($p = 0.057$). Gender, diplopia, coexisting ptosis and diplopia, smoking, presence of coexisting autoimmune or systemic vascular diseases, use of pyridostigmine or prednisolones, and their respective dosages were comparable between the two groups.

AChR-Ab$^+$ by Clinical Ocular Manifestations and Thymoma

AChR-Ab$^+$ status declined with age across all subgroups, with no difference observed in patients presenting with ptosis (Figure 1A). Diplopia, progression to GMG and thymoma demonstrated higher probabilities of AChR-Ab$^+$ (Figure 1B–D).

Factors Associated with AChR-Ab$^+$

Table 2 summarizes the factors influencing AChR-Ab$^+$. Advanced age at onset was significantly associated with lower likelihood of AChR-Ab$^+$ (OR 0.922; 95% CI: 0.864–0.983, $p = 0.014$). Diplopia (OR 5.394; 95% CI: 1.280–22.723, $p = 0.022$), progression to GMG (OR 4.640; 95% CI: 1.155–18.639, $p = 0.031$), and the presence of thymoma (OR 9.978; 95% CI: 2.094–47.547, $p = 0.004$) were positively associated with AChR-Ab$^+$ status. Ptosis, coexisting ptosis and diplopia lost statistical significance in the final model.

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Table 1 Baseline Characteristics of OMG Patients Stratified by AChR-Ab Serostatus

Characteristic, (%)	Total (111)	AChR-Ab$^+$ (51)	AChR-Ab$^-$ (60)	p-value
Age (mean±SD, years)	45.29 ± 7.29	43.82 ± 6.55	46.53 ± 7.71	0.051
Late onset	17 (15.3)	5 (9.8)	12 (20.0)	0.188†
Female gender	98 (88.3)	44 (86.3)	54 (90.0)	0.543
Remission	90 (81.1)	38 (74.5)	52 (86.7)	0.103
Clinical manifestations				
Ptosis	50 (45.1)	18 (35.3)	32 (55.3)	0.057
Diplopia	19 (17.1)	12 (23.5)	7 (11.7)	0.098
Both ptosis and diplopia	18 (16.2)	6 (11.8)	12 (20.0)	0.241
GMG	24 (21.6)	15 (29.4)	9 (15.0)	0.066
Smoking	4 (3.6)	1 (1.9)	3 (5.0)	0.623†
Thymoma	15 (13.5)	12 (23.5)	3 (5.0)	0.005†
Autoimmune diseases				
Thyroid disease	8 (7.2)	6 (11.8)	2 (3.3)	0.140†
RA	4 (3.6)	3 (5.9)	1 (1.7)	0.332†
Sjögren's syndrome	2 (1.8)	2 (3.9)	0 (0)	0.209†
SLE	4 (3.6)	2 (3.9)	2 (3.3)	>0.99†
Systemic vascular diseases				
Hypertension	1 (0.9)	0 (0)	1 (1.7)	>0.99†
Diabetes mellitus	3 (2.7)	1 (1.9)	2 (3.3)	>0.99†
Dyslipidemia	2 (1.8)	0 (0)	2 (3.3)	0.499†
Treatment				
Pyridostigmine				
Daily dose (mean±SD, mg/day)	131.13 ± 42.89	129.31 ± 46.71	132.67 ± 39.70	0.683
High dose pyridostigmine	19 (17.1)	8 (15.7)	11 (18.3)	0.712
Concurrent PSL				
Daily dose (mean±SD, mg/day)	17.77 ± 4.89	18.35 ± 4.61	17.27 ± 5.11	0.246
High dose PSL	80 (72.1)	37 (72.6)	43 (71.7)	0.918
Concurrent Azathioprine	12 (10.8)	9 (17.7)	3 (5.0)	0.062†
Follow-up time (mean±SD, months)	42.56 ± 10.98	41.47 ± 11.21	43.48 ± 10.79	0.338

Notes: †The p-value were obtained from the Fisher's exact test.
Abbreviations: OMG, ocular myasthenia gravis; AChR-Ab, anti-acetylcholine receptor antibodies; AChR-Ab$^+$, Acetylcholine Receptor Antibody-seropositive; AChR-Ab$^-$, Acetylcholine Receptor Antibody-seronegative; Late onset, age > 50 years; GMG, generalized myasthenia gravis; RA, rheumatoid arthritis; SLE, systemic lupus erythematous; PSL, prednisolones; High dose, daily dose of prednisolones > 15 mg/day.

Factors Associated with AChR-Ab Titers

Table 3, Thymoma was independently associated with AChR-Ab titers (β =1.556; 95% CI: 0.353–2.761, p = 0.012). In the adjusted model, neither ptosis, diplopia, the combination of ptosis and diplopia, nor progression to GMG were significantly associated.

Discussion

In this study, AChR-Ab seropositivity was associated with younger age, thymoma, and progression to GMG. Elevated antibody titers mainly associated with thymoma may serve as complementary markers for thymoma detection rather than as indicators of overall disease severity or ocular involvements.

The influence of age on AChR-Ab$^+$ in MG remains variable among previous studies inconclusive.[5,7] Peeler et al reported higher seropositivity in older patients,[7] whereas Wang et al found no age-related association.[14] In our study, younger patients showed higher rate of AChR-Ab$^+$; however, their titers did not consistently correlate with age. Age at onset may not insufficient to determine antibody status. Factors such as race,[8] disease phenotypes,[15] and immunosuppressive therapy may have an influence on seropositivity.[5]

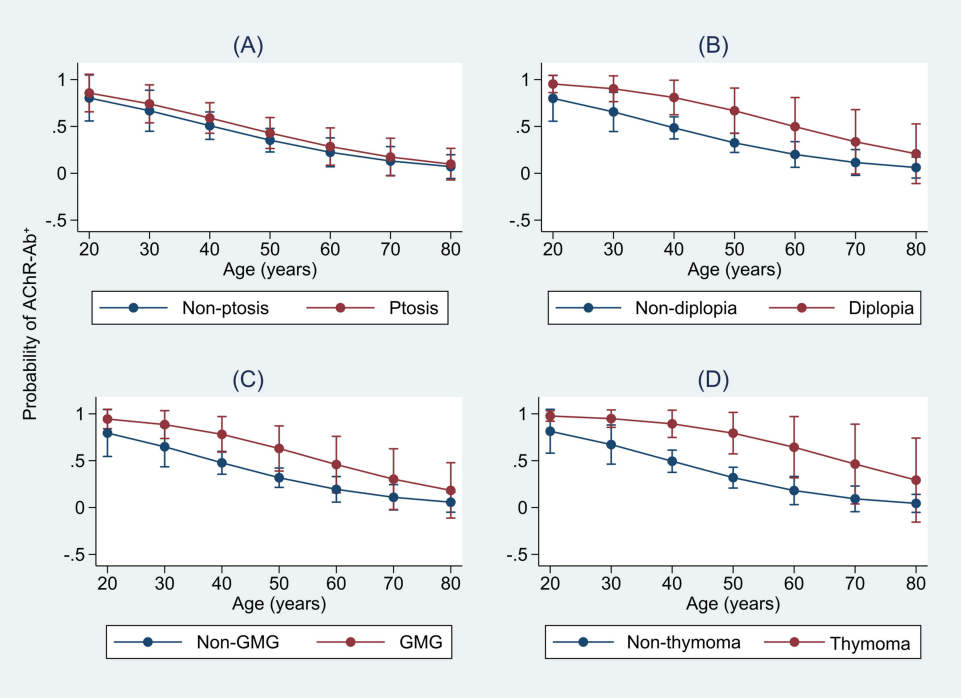

Figure 1 Age-Stratified Probability of AChR-Ab$^+$ Across Clinical Subgroups. AChR-Ab$^+$, Acetylcholine Receptor Antibody-seropositive; GMG, subsequent generalized myasthenia gravis. (**A**) Probability of AChR-Ab$^+$ by ptosis; (**B**) diplopia; (**C**) subsequent GMG; (**D**) thymoma. Statistical differences analyzed by multivariable logistic regression model.

Kim et al found higher titers in AChR-Ab$^+$ patients with both symptoms of ptosis and diplopia (5.27 ± 4.89 nmol/L) compared to those with one symptom (1.62 ± 3.60 nmol/L), despite similar seropositivity rates.[16] Ptosis alone may reflect more localized dysfunction with lower or undetectable antibody levels.[17] In contrast, either ptosis or diplopia was

Table 2 Logistic Regression Analysis of Factors Associated with AChR-Ab$^+$

	Univariable Analysis			Multivariable Analysis		
	OR	**(95% CI)**	**p-value**	**OR**	**(95% CI)**	**p-value**
Age (yr)	0.946	0.893–1.001	0.056	0.922	0.864–0.983	0.014
Ptosis	0.477	0.222–1.029	0.058	1.509	0.449–5.069	0.505
Diplopia	2.330	0.840–6.459	0.104	5.394	1.280–22.723	0.022
Both ptosis and diplopia	0.533	0.184–1.541	0.246		Not-included	
GMG	2.361	0.931–5.984	0.070	4.640	1.155–18.639	0.031
Thymoma	5.846	1.548–22.085	0.009	9.978	2.094–47.547	0.004

Notes: Statistically significant difference when p < 0.05.
Abbreviations: AChR-Ab$^+$, Acetylcholine Receptor Antibody-seropositive; OR, odds ratio; CI, confidence interval; GMG, generalized myasthenia gravis.

Table 3 Linear Regression Analysis of Predictors for AChR-Ab Titers

	Univariable Analysis			Multivariable Analysis		
	β	**(95% CI)**	**p-value**	**β**	**(95% CI)**	**p-value**
Ptosis	−0.596	−1.457–0.265	0.130	0.230	−1.172–0.713	0.630
Diplopia	−0.609	−1.939–0.720	0.453		Not-included	
Both ptosis and diplopia	0.108	−1.890–0.474	0.238		Not-included	
GMG	1.111	0.100–2.123	0.032	0.906	−0.219–2.031	0.113
Thymoma	1.625	0.408–2.842	0.009	1.556	0.353–2.761	0.012

Notes: Statistically significant difference when p < 0.05.
Abbreviations: AChR-Ab, Acetylcholine Receptor Antibody; CI, confidence interval; GMG, generalized myasthenia gravis.

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associated with higher relapse risk in a Chinese cohort.[18] In the first year of disease, AChR-Ab titers may remain undetectable, even in patients presenting with ptosis or ophthalmoparesis. Some AChR-Ab$^-$ cases may reflect early disease before seroconversion or progression to GMG.[19] Lower AChR-Ab$^+$ in isolated ptosis may reflect earlier onset, milder OMG severity, or a less aggressive course. In our cohort, diplopia was associated with AChR-Ab$^+$, whereas other ocular phenotypes showed no significant associations, contrasting with the previous findings.[17] Because pathogenicity also reflects factors beyond titer—such as receptor affinity and complement activation[7] — the link between ocular features and AChR-Ab status remains inconclusive, and interpretation should not rely on ocular manifestations, integrating with thymic factor when assessing progression risk and planning follow-up.

Although generalized MG has been consistently linked to AChR-Ab$^+$, how well antibody titers reflect disease severity in established generalization is still unclear. Mazzoli et al have proposed that higher titers (≥25.8 pmol/mL) do not correspond to greater clinical severity, raising the possibility that the relationship may flatten at the upper range.[6] Differences across studies— including variations in patient characteristics, thymic characteristics, disease duration, testing timing and variability in assay sensitivity and treatments — likely contribute to the inconsistent associations seen in the literature.[6,7,18,20] In our cohort, AChR-Ab$^+$ was associated with an increased risk of progression to GMG. However, a definitive titer threshold predictive of generalization could not be determined, as antibody levels were obtained at initial ocular onset rather than at the time of progression, limiting interpretation of longitudinal changes. In clinical practice, AChR-Ab$^+$ or higher titers may justify closer monitoring or earlier intervention. However, their interpretive value should be considered with caution, as factors such as presence of thymoma, thymectomy, and immunosuppressive therapy can also influence disease course.[7,16,21]

In the present study, thymoma emerged as the strongest factor associated with both AChR-Ab seropositivity and higher antibody titers, reflecting disrupted self-tolerance and enhanced B-cell–mediated antibody production,[22] further supported by the observation that thymectomy lowers AChR-Ab titers.[21] Awareness of thymoma may therefore inform closer clinical monitoring, as elevated titers in patients with ocular MG could indicate a higher risk of disease progression.

Although higher baseline AChR-Ab titers are linked to greater disease severity, their association with treatment response is inconsistent.[23] Bi et al reported that higher AChR-Ab titers are associated with unsuccessful prednisolone discontinuation.[13] Changes in their titers over time may reflect clinical improvement or relapse.[11,13] Serial monitoring of AChR-Ab titers may provide valuable insights into their relationship with disease activity and treatment response. Previous study has shown a time-dependent decline in AChR-Ab titers, with an approximate 50% reduction by 12 months—most notably within the first year,[24] potentially reflecting delayed seroconversion or initially low titers.[19] In our study, AChR-Ab titers did not appear to influence remission rates. Unfortunately, due to financial constraints, longitudinal AChR-Ab measurements were unavailable, limiting assessment of their potential role as a dynamic biomarker. A further study incorporating regular monitoring of AChR-Ab titers is warranted to better understand their utility in tracking disease progression and guiding therapeutic decisions.

While Palace et al found that treatment with immunosuppressive agents resulted in a sustained reduction in serum AChR-Ab levels,[25] Lindstrom et al reported that there is no correlation between the presence or titer of AChR-Ab and corticosteroids therapy.[5] In our cohort, azathioprine was more frequently prescribed to seropositive patients, likely reflecting its preferential use in individuals with more severe disease rather than a direct effect on antibody status, representing a potential selection bias; consequently, we decided to not include this variable in our regression model. Gender and other autoimmune diseases were not associated with AChR-Ab positivity in the present study, possibly due to sample size or phenotypic differences. While smoking can exacerbate MG through pro-inflammatory mechanisms, it does not appear to directly affect antibody titers,[26] consistent with our findings.

Some limitations should be addressed. First, the retrospective design resulted in incomplete data for certain variables, serial longitudinal measurements of serology. Second, MuSK and LRP4 antibodies were not assessed. Although MuSK antibodies are typically associated with myasthenia gravis involving severe bulbar manifestations,[2] such features are unlikely in our predominantly ophthalmic cohort. In addition, LRP4 testing was not routinely performed because commercially produced assays are unavailable in Thailand. Third, reliance on ELISA without confirmatory CBA—due to cost constraints—may have underestimated seropositivity and assay changes over time could have affected consistency.

The present study highlights that thymoma, subsequent progression to generalized myasthenia gravis, and younger age at onset are associated with AChR-Ab seropositivity, whereas ocular manifestations have minimal impact. Further investigation of additional biomarkers and long-term monitoring is needed to better inform prognosis and guide individualized management in ocular MG.

Conclusion

Serostatus of acetylcholine receptor antibodies may be useful for identifying MG patients at higher risk of generalization or thymoma, while antibody titers may provide complementary information, particularly in relation to thymoma, rather than serving as direct markers of disease severity or ocular manifestations.

Consent Statement

All participants gave written informed consent before the data collection process began.

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Disclosure

None of the authors have any conflict of interest to disclose for this work.

References

1. Osserman KE, Genkins G. Studies in myasthenia gravis: review of a twenty-year experience in over 1200 patients. *Mt Sinai J Med*. 1971;38(6):497–537.
2. Zhou Y, Chen J, Li Z, et al. Clinical features of myasthenia gravis with antibodies to MuSK based on age at onset: a multicenter retrospective study in China. *Front Neurol*. 2022;13:879261. doi:10.3389/fneur.2022.879261
3. Li M, Han J, Zhang Y, et al. Clinical analysis of Chinese anti-low-density-lipoprotein-receptor-associated protein 4 antibodies in patients with myasthenia gravis. *Eur J Neurol*. 2019;26(10):1296–e84. doi:10.1111/ene.13979
4. Yan C, Li W, Song J, et al. Cell-based versus enzyme-linked immunosorbent assay for the detection of acetylcholine receptor antibodies in Chinese juvenile myasthenia gravis. *Pediatr Neurol*. 2019;98:74–79. doi:10.1016/j.pediatrneurol.2019.01.016
5. Lindstrom JM, Seybold ME, Lennon VA, Whittingham S, Duane DD. Antibody to acetylcholine receptor in myasthenia gravis. Prevalence, clinical correlates, and diagnostic value. *Neurology*. 1976;26(11):1054–1059. doi:10.1212/wnl.26.11.1054
6. Mazzoli M, Ariatti A, Valzania F, et al. Factors affecting outcome in ocular myasthenia gravis. *Int J Neurosci*. 2018;128(1):15–24. doi:10.1080/00207454.2017.1344237
7. Peeler CE, De Lott LB, Nagia L, Lemos J, Eggenberger ER, Cornblath WT. Clinical utility of acetylcholine receptor antibody testing in ocular myasthenia gravis. *JAMA Neurol*. 2015;72(10):1170–1174. doi:10.1001/jamaneurol.2015.1444
8. Park KH, Waters P, Woodhall M, et al. Myasthenia gravis seronegative for acetylcholine receptor antibodies in South Korea: autoantibody profiles and clinical features. *PLoS One*. 2018;13(3):e0193723. doi:10.1371/journal.pone.0193723
9. Heldal AT, Eide GE, Romi F, Owe JF, Gilhus NE. Repeated acetylcholine receptor antibody-concentrations and association to clinical myasthenia gravis development. *PLoS One*. 2014;9(12):e114060. doi:10.1371/journal.pone.0114060
10. Kim DH, Roh HC, Oh SY. Ophthalmologic clinical features of ocular myasthenia gravis. *Medicine*. 2023;102(2):e31972. doi:10.1097/md.0000000000031972
11. Marcuse F, Brandts L, Moens D, et al. The association between anti-acetylcholine receptor antibody level and clinical improvement in myasthenia gravis. *Eur J Neurol*. 2022;29(4):1187–1197. doi:10.1111/ene.15238
12. Jitpimolmard S, Taimkao S, Chotmongkol V, Sawanyawisuth K, Vincent A, Newsom-Davis J. Acetylcholine receptor antibody in Thai generalized myasthenia gravis patients. *J Med Assoc Thai*. 2006;89(1):68–71.
13. Bi Z, Cao Y, Liu C, et al. Remission and relapses of myasthenia gravis on long-term tacrolimus: a retrospective cross-sectional study of a Chinese cohort. *Ther Adv Chronic Dis*. 2022;13:20406223221122538. doi:10.1177/20406223221122538
14. Wang L, Wang S, Yang H, et al. No correlation between acetylcholine receptor antibody concentration and individual clinical symptoms of myasthenia gravis: a systematic retrospective study involving 67 patients. *Brain Behav*. 2021;11(7):e02203. doi:10.1002/brb3.2203
15. Ullah U, Iftikhar S, Javed MA. Relationship between low and high anti-acetylcholine receptor antibody titers and clinical severity in myasthenia gravis. *J Coll Physicians Surg Pak*. 2021;31(8):965–968. doi:10.29271/jcpsp.2021.08.965
16. Kim H, Oh SY. Diagnostic yields and clinical features of ocular myasthenia gravis. *Medicine*. 2021;100(25):e26457. doi:10.1097/md.0000000000026457
17. Axelsen KH, Kjær Andersen R, Andersen LK, Vissing J, Witting N. Ocular versus generalized myasthenia gravis: a continuum associated with acetylcholine receptor antibody titers. *Neuromuscul Disord*. 2024;43:39–43. doi:10.1016/j.nmd.2024.07.002
18. Wang L, Zhang Y, He M. Clinical predictors for the prognosis of myasthenia gravis. *BMC Neurol*. 2017;17(1):77. doi:10.1186/s12883-017-0857-7
19. Oda K, Ito Y. Myasthenia gravis: antibodies to acetylcholine receptor in ocular myasthenia gravis. *J Neurol*. 1981;225(4):251–258. doi:10.1007/bf00313297
20. Chou CC, Su IC, Chou IJ, et al. Correlation of anti-acetylcholine receptor antibody levels and long-term outcomes of juvenile myasthenia gravis in Taiwan: a case control study. *BMC Neurol*. 2019;19(1):170. doi:10.1186/s12883-019-1397-0

21. Hyunjin K, Young-Min L, Eun-Jae L, Yeo Jin O, Kwang-Kuk K. Factors predicting remission in thymectomized patients with acetylcholine receptor antibody-positive myasthenia gravis. *Muscle Nerve*. 2018;58(6):796–800. doi:10.1002/mus.26300
22. Isshiki Y, Mimura O, Gomi F. Clinical features and treatment status of antiacetylcholine receptor antibody-positive ocular myasthenia gravis. *Jpn J Ophthalmol*. 2020;64(6):628–634. doi:10.1007/s10384-020-00770-z
23. Sanders DB, Burns TM, Cutter GR, Massey JM, Juel VC, Hobson-Webb L. Does change in acetylcholine receptor antibody level correlate with clinical change in myasthenia gravis? *Muscle Nerve*. 2014;49(4):483–486. doi:10.1002/mus.23944
24. Seybold ME, Lindstrom JM. Patterns of acetylcholine receptor antibody fluctuation in myasthenia gravis. *Ann N Y Acad Sci*. 1981;377:292–306. doi:10.1111/j.1749-6632.1981.tb33739.x
25. Palace J, Newsom-Davis J, Lecky B. A randomized double-blind trial of prednisolone alone or with azathioprine in myasthenia gravis. Myasthenia Gravis Study Group. *Neurology*. 1998;50(6):1778–1783. doi:10.1212/wnl.50.6.1778
26. Miyazaki Y, Niino M, Sakushima K, et al. Association of smoking and generalized manifestations of myasthenia gravis. *Intern Med*. 2022;61(11):1693–1698. doi:10.2169/internalmedicine.8460-21