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ORIGINAL RESEARCH

Long-Term Clinical Outcomes of Rituximab Across Serological Subtypes of Myasthenia Gravis

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ABSTRACT

Rituximab, a B-cell-depleting monoclonal antibody, has gained increasing attention as a therapeutic option for myasthenia gravis (MG), particularly in refractory cases. However, its long-term efficacy across different serological subtypes remains to be investigated. This study aimed to evaluate the long-term clinical outcomes of rituximab treatment in patients with generalized myasthenia gravis (MG), including those with anti-acetylcholine receptor (AChR) antibodies, anti-muscle-specific kinase (MuSK) antibodies, and double-seronegative profiles. A retrospective cross-sectional study was conducted at the Neuromuscular Unit between 2012 and 2024. Thirty-six patients who received rituximab and had a minimum follow-up of 12 months were included. Treatment outcomes were assessed using MGFA and MGFA-PIS scores at baseline, at 6 months, and at the final evaluation. Subgroup analyses were performed based on antibody status. Sustained clinical improvement was observed in all serological subgroups. At six months, 70% of patients showed improvement or remission according to MGFA-PIS, increasing to 100% at final evaluation. AChR+ and MuSK+ patients demonstrated early and persistent responses, while a delayed but significant benefit was seen in double-seronegative patients. Rituximab appears to be a safe and effective long-term treatment for generalized MG, with benefits observed across all major antibody subtypes. The lack of a significant difference between the 6-month and final follow-up in AChR+ and MuSK+ patients suggests that the early improvement was sustained in the long term.

Keywords: Myasthenia Gravis. Rituximab. Anti-acetylcholine receptor antibody. Anti-muscle-specific kinase antibody. Seronegative.

Miyastenia Gravis'in Serolojik Alttiplerinde Rituksimabın Uzun Dönem Klinik Sonuçları

ÖZET

Rituksimab, B hücrelerini tüketen bir monoklonal antikor olarak, özellikle tedaviye dirençli olgularda miyastenia gravis (MG) tedavisinde giderek artan bir ilgi görmektedir. Ancak, farklı serolojik alt tiplerdeki uzun dönem etkinliği hâlen araştırılmaktadır. Bu çalışma, anti–asetilkolin reseptörü (AChR) antikorları, anti–kas-spesifik kinaz (MuSK) antikorları ve çift seronegatif profilleri içeren jeneralize miyastenia gravis (MG) hastalarında rituksimab tedavisinin uzun dönem klinik sonuçlarını değerlendirmeyi amaçlamıştır. 2012–2024 yılları arasında Nöromüsküler Ünitede retrospektif, kesitsel bir çalışma yürütülmüştür. Rituksimab tedavisi alan ve en az 12 aylık takip süresi bulunan 36 hasta çalışmaya dahil edilmiştir. Tedavi sonuçları, başlangıçta, 6. ayda ve son değerlendirmede MGFA ve MGFA-PIS skorları kullanılarak değerlendirilmiştir. Antikor durumuna göre alt grup analizleri yapılmıştır. Tüm serolojik alt gruplarda kalıcı klinik iyileşme gözlenmiştir. Altıncı ayda hastaların %70'i MGFA-PIS'e göre iyileşme veya remisyon göstermiş olup, bu oran son değerlendirmede %100'e yükselmiştir. AChR+ ve MuSK+ hastalar erken ve kalıcı yanıt gösterirken, çift seronegatif hastalarda gecikmiş ancak anlamlı bir fayda saptanmıştır. Rituksimab, tüm ana antikor alt tiplerinde yarar sağlayan, jeneralize MG için güvenli ve etkili bir uzun dönem tedavi seçeneği olarak görünmektedir. AChR+ ve MuSK+ hastalarda 6. ay ile son takip arasındaki farkın anlamlı olmaması, erken dönemde elde edilen iyileşmenin uzun dönemde de sürdüğünü göstermektedir.

Anahtar Kelimeler: Miyastenia Gravis. Rituksimab. Anti–asetilkolin reseptörü antikoru. Anti–kas-spesifik kinaz antikoru. Seronegatif.

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Autoimmune Myasthenia Gravis (MG) is characterized by impaired neuromuscular transmission leading to fluctuating muscle weakness and fatigability[1]. The predominant autoantibodies in MG are directed against acetylcholine receptors (AChR+) and are detected in approximately 85% of patients. Muscle-specific kinase (MuSK+) antibodies are present in a subset of patients, which disrupts AChR clustering and maintenance. Double seronegative MG is defined by the absence of these two known autoantibodies. In such cases, particularly when

clinical suspicion is high, single-fiber electromyography (SFEMG) plays a crucial role in supporting the diagnosis[1]. The autoantibody profile plays an essential role in MG pathogenesis, influencing diagnostic strategies, prognostic assessments, and therapeutic decision-making[2].

The management of generalized MG typically involves symptomatic treatment with acetylcholinesterase inhibitors such as pyridostigmine and immunomodulatory therapies including intravenous immunoglobulin (IVIG) and plasma exchange (PLEX) for acute exacerbations[2]. Long-term immunosuppressive regimens commonly include corticosteroids, azathioprine, mycophenolate mofetil, cyclosporine, methotrexate, and cyclophosphamide[2]. Despite these therapeutic approaches, approximately 10% of patients exhibit treatment-refractory disease, characterized by persistent disease activity despite prolonged and adequate immunosuppressive therapy[3].

MG is widely recognized as a prototypical B–cell–driven autoimmune disease, emphasizing the crucial involvement of B lymphocytes in disease development and progression[4]. Given this pathophysiological basis, B-cell-depleting therapies have emerged as a promising treatment strategy. Rituximab, a monoclonal antibody targeting the CD20 antigen on B lymphocytes, has demonstrated clinical efficacy in various autoimmune diseases and has been increasingly utilized in MG[4]. While rituximab is well-established as an effective treatment for MuSK+ MG patients, its therapeutic role in AChR+ and seronegative MG patients remains a subject of ongoing investigation, with conflicting evidence regarding its long-term efficacy[5-9].

This study aims to evaluate the long-term efficacy of rituximab across different MG serotypes and to assess its impact on clinical outcomes. By analyzing the differential therapeutic response among seropositive and seronegative MG subgroups, this study seeks to contribute to the optimization of targeted immunotherapy in MG.

Material and Method

Study Design

This retrospective cross-sectional study was conducted at Bursa Uludag University Hospital, between 2012 and 2024. The study was approved by the Ethics Committee.

Participants

The diagnosis of MG was established based on a comprehensive evaluation, including detailed clinical history, neurological examination, serological testing for disease-specific autoantibodies, and confirmatory electrophysiological studies such as repetitive nerve stimulation or single-fiber electromyography.

Inclusion criteria were as follows:

- Diagnosis of generalized MG based on the international consensus criteria[10];
- Rituximab treatment administered from January 2012 to January 2024;
- A minimum follow-up duration of 12 months after rituximab treatment.

Participants were categorized into three groups based on their antibody status: AChR+, MuSK+, or double seronegative. Alternative clinical diagnoses were excluded through appropriate investigations. All patients who received rituximab as part of their treatment protocol were followed at the Neuromuscular Unit of Bursa Uludag University Hospital from 2012 to 2024, with a minimum follow-up period of 12 months post-treatment. A flow chart summarizing patient selection and inclusion is presented in Figure 1.

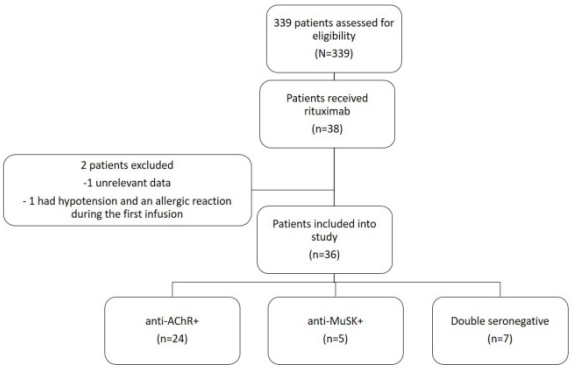

Figure 1:
Flow diagram summarizing the selection and inclusion of patients in the study.

Data Collection and Outcome Measures

Treatment outcomes were evaluated using the Myasthenia Gravis Foundation of America Post-Intervention Status (MGFA-PIS), which classifies patient status as complete stable remission (CSR), pharmacological remission (PR), or minimal manifestations (MM). Changes in clinical status following treatment were categorized as improved (I), unchanged (U), worsened (W), exacerbated (E), or death (D). MGFA-PIS scores were recorded at two time points: the 6-month follow-up and the final evaluation.

In addition, Myasthenia Gravis Foundation of America (MGFA) clinical classification scores were assessed at three time points: baseline (before rituximab treatment), the 6-month follow-up, and the final evaluation. The requirement for rescue treatments during the follow-up period was also documented.

Subgroup analyses were performed based on antibody status (AChR+, MuSK+, or double-seronegative) to evaluate potential differences in treatment response across serological profiles.

Rituximab Treatment Protocol

Rituximab was administered as an induction regimen consisting of 1000 mg infusions on day 1 and day 15. Maintenance therapy included 1000 mg infusions at intervals of six months or longer.

Statistical Analysis

Descriptive analyses were performed to summarize the demographic and clinical features of the study cohort. Continuous variables, such as age, disease duration, and clinical scale scores, were reported as means with standard deviations. Categorical variables, including gender, MGFA classification, antibody status, history of thymectomy, and treatment modalities, were described using frequencies and percentages.

Longitudinal changes in MGFA score during the follow-up period were analyzed using the non-parametric Friedman test for repeated measures. This study is reported in accordance with the guidelines[11].

Results

The mean age at MG diagnosis was 44.22 ± 19.25 years. The average follow-up duration after the initiation of rituximab treatment was 26.11±15.15 months. Among the 36 patients who received rituximab therapy, 9 were male and 27 were female. Regarding antibody status, 24 patients (66.7%) were AChR+, 5 patients (13.9%) were MuSK+, and 7 patients (19.4%) were seronegative. The demographics of patients and previous treatments are shown in Table I.

Before rituximab treatment, the majority of patients were classified as MGFA II (52.8%) or MGFA III (44.4%), with a small proportion categorized as MGFA V (2.8%). At the 6-month follow-up, 19.4% of patients had reached MGFA 0, 13.9% were classified as MGFA I, 55.6% as MGFA II, and 11.1% as MGFA III. At the final evaluation, 16.7% of patients remained in MGFA 0, 22.2% in MGFA I, 58.3% in MGFA II, and only 2.8% in MGFA III. These results indicate a sustained clinical improvement over time following rituximab treatment. Changes in MGFA classification at 6 months and at the final assessment are illustrated in Figure 2A.

MGFA-PIS outcomes demonstrated that nearly 70% of patients showed improvement or achieved remission at the 6-month follow-up, while approximately 30% remained unchanged. By the final evaluation, all patients had either improved or were in remission according to MGFA-PIS criteria. Despite these favorable MGFA-PIS results, it is noteworthy that 15 patients required additional rescue therapies during the follow-up period. Furthermore, myasthenic deterioration necessitating intensive care unit (ICU) admission was observed in two patients, highlighting that clinically significant exacerbations may still occur in a subset of individuals, even in the context of overall improvement.

Table I. Patient demographics and previous treatments.

		RXT treatment (N=36)
Gender (Female/Male)(n/n)		27/9
Age at onset (y)		44.22±19.25
Total duration of RTX treatment (m)		26.11±15.16
Antibody profile	Ach n(%)	24 (66.7%)
	MuSK n(%)	5 (13.9%)
	Seronegative n(%)	7 (19.4%)
MGFA score (before RTX)	MGFA I	-
	MGFA II	19 (52.8%)
	MGFA III	16 (44.4%)
	MGFA IV	-
	MGFA V	1 (2.8%)
MGFA score (after 6. Months of RTX treatment)	MGFA 0	7 (19.4%)
	MGFA I	5 (13.9%)
	MGFA II	20 (55.6%)
	MGFA III	4 (11.1%)
	MGFA IV	-
	MGFA V	-
MGFA score at final examination	MGFA 0	6 (16.7%)
	MGFA I	8 (22.2%)
	MGFA II	21 (58.3%)
	MGFA III	1 (2.8%)
	MGFA IV	-
	MGFA V	-
MGFA-PIS after 6 months	Death	-
	Worse	-
	Unchanged	11 (30.6%)
	Improved	18 (50.0%)
	Minimal manifestations	-
	Complete stable remission or pharmacological remission	7 (19.4%)
MGFA-PIS at final examination	Death	-
	Worse	-
	Unchanged	7(19.4)
	Improved	23 (63.9%)
	Minimal manifestations	-
	Complete stable remission or pharmacological remission	6 (16.7%)
Previous treatments before RTX		
Previous Pyridostigmine use		35 (96.97%)
Previous Corticosteroid use		34 (93.94%)
Previous 1 NSIST use		10 (27.8%)
Previous 2 NSIST use		18 (50.0%)
Previous ≥3 NSIST use		4 (11.1%)
Thymectomy		18 (50.0%)
Thymoma*		9 (50.0%)
Rescue treatment before RTX		36 (100%)
Rescue treatment after 3 months of RTX		15 (41.7%)
RTX= rituximab, NSIST= Non-steroid immunosupressive treatment *in thymectomized patients		

Regarding previous treatments, nearly all of the patients had used pyridostigmine and corticosteroids. A sizable proportion had received at least one (27.8%), two (50.0%), or three or more (11.1%) non-steroid immunosuppressive therapies (NSISTs). Additionally, 50% of patients had undergone thymectomy, and among these, 9 (50%) had thymoma.

One patient experienced hypotension and an allergic reaction at the initial administration, leading the patient to decline further rituximab infusion. During follow-up, one patient developed herpes zoster ophthalmicus and keratitis, while another experienced disseminated herpes zoster involving the trunk. No other serious complications were observed. CD19 levels were monitored in a subset of patients and are presented in Figure 2B.

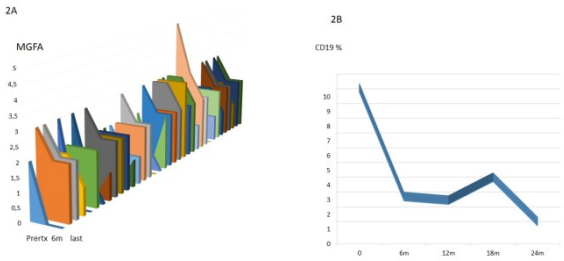

Figure 2:
Changes in MGFA classification and CD19 levels following rituximab treatment.
(A) *MGFA classification at 6 months and at the final assessment.*
(B) *CD19 levels of patients.*

Analysis of MGFA scores based on antibody status revealed that patients with AChR+ antibodies showed significant clinical improvement following rituximab treatment ($p<0.001$). Notably, patients with MuSK+ antibodies, as well as those who were double-seronegative, also exhibited meaningful clinical improvement ($p=0.02$), although the sample size in these subgroups was smaller. These improvements were observed at both the 6-month follow-up and the final evaluation, indicating a sustained therapeutic effect. In AChR+ patients, clinical improvement was evident as early as the 6-month follow-up and remained stable through the final evaluation. Similarly, the MuSK+ group demonstrated a sustained and marked therapeutic response at both time points, supporting the consistent and long-lasting efficacy of rituximab in this subgroup. In contrast, among double-seronegative patients, the therapeutic effect became more pronounced at the final assessment, suggesting a potentially delayed but clinically meaningful response (Table II).

Table II. Changes in MGFA scores and clinical improvement following rituximab treatment according to antibody status.

All patients (N=36)	Mean±SD	Median (min-max)	Test statistics	*p*
MGFA before RTX treatment	2.53±0.65	2 (2-5)[a]	45.15	<0.001
MGFA after 6-month follow	1.58±0.94	2 (0-3)[b]		
MGFA at final assessment	1.47±0.81	2 (0-3)[b]		
anti-AChR antibody (+) (n=24)				
MGFA before RTX treatment	2.50±0.72	2 (2-5)[a]	31.63	<0.001
MGFA after 6-month follow	1.63±0.88	2 (0-3)[b]		
MGFA at final assessment	1.46±0.78	2 (0-2)[b]		
Anti-MuSK antibody (+) (n=5)				
MGFA before RTX treatment	2.80±0.45	2 (2-3)[a]	8.40	0.02
MGFA after 6-month follow	1.20±1.10	2 (0-2)[b]		
MGFA at final assesment	1.60±0.89	2 (0-2)[b]		
Double seronegative (n=7)				
MGFA before RTX treatment	2.43±0.54	2 (2-3)[a]	6.71	0.04
MGFA after 6-month follow	1.71±1.11	2 (0-3)[a]		
MGFA at final assessment	1.43±0.98	1 (0-3)[b]		

Friedman Test [a-b]: Different letters indicate statistically significant differences

Discussion and Conclusion

Rituximab has emerged as a promising therapeutic option for MG, demonstrating effectiveness beyond specific subgroups and potentially benefiting a broader population. Our findings suggest that rituximab has a positive influence on the clinical course of MG, offering a viable treatment alternative for patients who do not respond to first-line therapies. These findings are consistent with earlier research highlighting the effectiveness of rituximab in the treatment of refractory MG[5-10,12-14]. In recent years, increasing evidence has supported its role in modulating the immune system by depleting B cells, which play a critical role in MG pathogenesis[5-10,12]. Notably, its beneficial effects were observed across all serological subtypes, including AChR-positive, MuSK positive, and double seronegative patients, highlighting its potential as a broadly effective therapeutic option in the treatment of myasthenia gravis.

Although double seronegative myasthenia gravis lacks detectable AChR or MuSK antibodies, emerging evidence suggests that these patients may still benefit from B-cell-targeted therapies, such as rituximab. Our study showed a meaningful clinical response in seronegative patients, particularly evident at the final evaluation, indicating a potentially delayed but sustained therapeutic effect. This finding is consistent with previous reports that have documented improvement in double seronegative myasthenia

gravis following rituximab treatment, despite the absence of known circulating autoantibodies[15,16]. The clinical response in these patients may reflect the involvement of B-cell-mediated mechanisms that contribute to disease pathogenesis. These results support the notion that rituximab may be a valuable treatment option even in seronegative MG, where therapeutic decisions are often more challenging due to the lack of clear immunological markers.

One of the key findings of our study is that the initial clinical improvement, reflected by the decrease in MGFA scores at the 6-month follow-up, was sustained through to the final evaluation, with no significant difference observed between the two time points. This suggests that the therapeutic benefit of rituximab persists over time, reinforcing its potential as a durable treatment option for MG. This is particularly relevant for patients with refractory MG, where effective and long-lasting treatment strategies are limited.

In our study, clinical improvement was evident in all serological subgroups. AChR+ and MuSK+ patients showed marked responses as early as the 6-month follow-up, which remained stable at the final assessment. Interestingly, in double-seronegative patients, the therapeutic effect became more prominent at the final evaluation, suggesting a delayed yet meaningful response. Although some studies have reported greater efficacy of rituximab in MuSK-positive patients, our findings, in line with several other reports, support its effectiveness across different antibody profiles, including AChR+, MuSK+, and double-seronegative MG.

Although rituximab is considered an effective treatment for MG, several challenges persist, particularly regarding the optimal dosing regimen, timing of administration, duration of therapy, and inconsistencies in clinical protocols across treatment centers[4-6,12,15,17-20]. While some studies suggest that a single course of rituximab may be sufficient for disease control, others demonstrate the need for repeated infusions to maintain long-term remission. This variability underscores the need for large-scale, controlled clinical trials to establish standardized treatment algorithms and to define the most effective administration strategies. Although rituximab was initially indicated for treatment-refractory MG, accumulating evidence from recent studies supports its potential role in managing patients with new-onset generalized MG[9,17,21].

Another crucial aspect to consider is the safety profile of rituximab. While it is generally well tolerated, the risk of adverse effects, such as allergic reactions, infections, and progressive multifocal leukoencephalopathy, should not be overlooked. Given that rituximab leads to prolonged B-cell depletion, patients may become more susceptible to infections, particularly from opportunistic pathogens. Additionally, PML- a rare but potentially fatal demyelinating disease caused by the JC virus- remains a concern in immunocompromised patients receiving rituximab. Therefore, close monitoring and individualized risk assessment are essential to ensure patient safety during treatment. Physicians should consider pre-treatment screening for infection risks and implement regular follow-ups to detect and manage potential complications early. In our cohort, two patients developed herpes zoster infections—one with disseminated involvement of the trunk and another with herpes zoster ophthalmicus and keratitis. No other serious adverse events were observed during the follow-up period**.** It is also worth noting that premedication protocols may have contributed to the low rate of early allergic reactions, potentially preventing infusion-related hypersensitivity events.

In conclusion, rituximab represents a valuable therapeutic alternative for MG patients, particularly in those who are refractory to conventional therapies. Its ability to induce and maintain clinical improvement makes it an attractive option in the treatment landscape of MG. However, uncertainties regarding optimal dosing, treatment duration, and long-term safety warrant additional studies. Future research should focus on establishing standardized treatment protocols and identifying patient subgroups that would benefit the most from this therapy. Furthermore, long-term observational studies are necessary to assess the impact of rituximab on disease progression, quality of life, and healthcare resource utilization in patients with MG.

Limitations

The retrospective nature of this study represents a limitation, as it prevented the systematic evaluation of other clinical scales. Additionally, the fact that some patients continued to require rescue therapies raises the possibility that improvements observed in MGFA and MGFA-PIS scores may have been influenced by concurrent treatments, potentially leading to an overestimation of clinical benefit. Additional limitations include the heterogeneity of baseline immunosuppressive treatments, the variability in the number of rituximab cycles administered, and the relatively small sample size within each serological subgroup, which may limit the generalizability of the findings.

Researcher Contribution Statement:
Idea and design: E.O.A., N.M.K., N.K.; Data collection and processing: E.O.A., N.M.K., N.K., Y.D, S.E.L.; Analysis and interpretation of data: E.O.A.; N.M.K.; Writing of significant parts of the article: E.O.A., N.M.K.; N.K.
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