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Early use of rituximab in myasthenia gravis in a resource-limited setting: A retrospective cohort study from a tertiary center in Pakistan

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Abstract

Background: Myasthenia gravis (MG) is an autoimmune disorder of the neuromuscular junction, most commonly mediated by acetylcholine receptor antibodies. Standard treatments include corticosteroids, acetylcholinesterase inhibitors, and conventional immunosuppressants, while intravenous immunoglobulin (IVIg) or plasma exchange is reserved for refractory disease or myasthenic crises. Rituximab, a CD20-targeting monoclonal antibody, has demonstrated efficacy in treatment-resistant MG, particularly in MuSK-positive cases. However, its use early in the disease course, particularly soon after diagnosis or after failure of only one immunosuppressant remains underexplored, especially in resource-limited settings where access to IVIg and recurrent hospitalizations is often constrained. In our cohort, rituximab was initiated at a median disease duration of one year, although seven patients had experienced a prior myasthenic crisis. This study evaluates the efficacy and safety of early rituximab use in such settings. *Methods:* In this retrospective cohort study (Dec 2021–June 2024), 12 patients with generalized MG were treated with rituximab and followed for 18 months. Clinical outcomes, including MGFA-Post Intervention Status, corticosteroid dose reduction, and adverse effects, were assessed. *Results:* Seven patients had a history of myasthenic crisis before rituximab; only one had a recurrence after treatment. Overall, 91.7% of patients showed significant clinical improvement and/or reduced need for symptomatic and immunosuppressive therapy. At 18 months, Myasthenia Gravis Foundation of America (MGFA) post-intervention status indicated complete stable remission in 11%, pharmacologic remission in 11% while the rest had minimal manifestations only. Mean corticosteroid doses dropped by 22.5 mg after the first rituximab cycle, 26.4 mg after the second and 29.8 mg after the third. Seventy-five percent experienced no major treatment-related complications.
Conclusion: Early rituximab use in generalized MG appears effective and steroid-sparing, with potential to lessen disease burden and healthcare costs in resource-constrained settings.

Keywords: Myasthenia gravis, rituximab, myasthenic crisis, resource-limited settings, immunosuppression

INTRODUCTION

Myasthenia gravis (MG) is an immune-mediated disorder of neuromuscular transmission characterized by fatigable weakness affecting striated muscles. While most individuals have a generalized presentation at onset, the disease may be confined to extraocular muscles only in about 15 percent of cases.[1] In severe cases, bulbar involvement predominates, eventually culminating in a myasthenic crisis, which is treated with plasmapheresis or intravenous immunoglobulin. The goal of treatment in MG is to prevent crisis and achieve remission. This is achieved by symptomatic control with acetylcholinesterase inhibitors, in combination with long-term immunomodulatory therapy.[2]

Corticosteroids are the first line, but long-term use is limited by adverse effects. Hence, several steroid sparing agents including azathioprine, mycophenolate and cyclosporine have remained popular for long term clinical control. However, more recently, the use of biologic agents instead of conventional immunosuppressive agents have shifted the paradigm in the treatment of

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autoimmune diseases including MG.[3]

Rituximab is an anti-CD20 monoclonal antibody, which is being used as an off-label treatment in patients who have MG.[4] In 2022, the RINOMAX trial established the efficacy of rituximab in attainment of minimal myasthenic manifestations and reduction in requirement of rescue medications, in contrast to placebo.[5] While several studies in the past have shown promising effects on clinical outcomes, the definite role of rituximab as a therapeutic option is still uncertain. However, it is now being increasingly considered as a reasonable alternative in refractory cases.[6]

The primary objective of our study was to evaluate the role of early use of rituximab in patients with MG, with the aim of achieving earlier and sustained remission. By facilitating earlier disease control, this approach has the potential to lessen the long-term treatment burden associated with prolonged immunosuppressive therapy, recurrent hospitalizations, and the need for repeated rescue interventions such as plasma exchange or intravenous immunoglobulin

METHODS

Collection of patient data

A retrospective study was performed for MG patients referred to the Aga Khan University Hospital Neurology Clinics from December 2021 to June 2024. 12 patients were identified with refractory generalized disease. Both MuSK and AChR antibody-positive groups were included; seronegative cases were excluded. Patients who expired or were lost to follow-up were excluded from subsequent time-point analyses, and available-case analysis was performed.

Patients were classified as having refractory MG if they met one or more of the following criteria: (a) inadequate control of clinical symptoms despite treatment with corticosteroids and one or more oral immunosuppressive agents; (b) inability to taper immunotherapy without precipitating clinical relapse; (c) dependence on frequent rescue therapies such as intravenous immunoglobulin (IVIg) or plasmapheresis; or (d) development of significant adverse effects from immunosuppressive medications. Those who had marked generalized muscle weakness (MGFA clinical classification III-V) that may include bulbar and/or respiratory involvement were included in the category of severe MG.

All clinical exams were completed or supervised by one senior consultant neurologist. Physical examinations were performed before and after rituximab treatment. The following parameters were recorded from the medical notes:

- Prednisone dosage, pre and post rituximab administration 6, 12 and 18 months.
- Requirement for intravenous immunoglobulin (IVIG) or plasmapheresis, pre and post rituximab administration.
- Number of immunosuppressive agents received prior to initiation of rituximab.
- Myasthenia Gravis Composite Scale (MGCS) scores, pre and post rituximab administration at 6, 12 and 18 months. The MGCS is a validated 10-item outcome measure incorporating both patient-reported symptoms and physician-assessed examination findings, with total scores ranging from 0 to 50; higher scores reflect greater disease severity.
- Myasthenia Gravis Foundation of America Post-Intervention Status at 12 and 18 months. The Myasthenia Gravis Foundation of America (MGFA) Post-Intervention Status (PIS) classification was used to assess treatment response at follow-up. This system categorizes patients into complete stable remission (CSR), pharmacologic remission (PR), and minimal manifestations (MM), based on the presence of symptoms and the need for ongoing myasthenia-related therapy.
- Rituximab-related side effects.

Administration of rituximab

Eligible patients included all individuals diagnosed with MG who received rituximab as part of their treatment. Rituximab was administered intravenously at a dose of 1gm, given as two infusions spaced two weeks apart, followed by subsequent 1gm infusion every six months. Patients were followed in the neurology clinic for at least 18 months following the administration of the first dose of rituximab.

Safety

To assess the safety profile for rituximab, we reviewed the physician notes as well as complete blood count (CBC) and liver function test (LFT)

profiles available in our electronic medical record.

Statistical analysis

Participants were enrolled using a non-randomized consecutive sampling technique. Statistical analysis was performed using STATA 17. Given the retrospective design, small sample size, and non-normal distribution of clinical variables, analyses were primarily descriptive in nature. Continuous variables were summarized as median (interquartile range) and/or mean ± standard deviation, given the skewed distribution and to facilitate comparison with prior literature. Categorical variables were expressed as frequencies and percentages.

Longitudinal changes in clinical outcomes, including MGCS, prednisone dose, and MGFA-PIS, were assessed descriptively at baseline and at 6-, 12-, and 18-month follow-up intervals. Steroid dose changes were additionally categorized as reduced, increased, unchanged, or discontinued.

Ethical considerations

The study was approved by the Ethical Review Committee of Aga Khan University Hospital.

RESULTS

Twelve patients with generalized myasthenia gravis were identified; eight patients were male. All were seropositive. Ten had positive acetylcholine receptor antibodies and two had positive MuSK antibodies. The median duration of MG was 3 years. Rituximab was started at a median duration of 1 year after the onset of illness. Before administration of rituximab, four patients had a failure of response to single steroid sparing agent, seven had a failure of response to two or more steroid sparing agents and one of them was started on Rituximab early in the course of illness due to severe symptoms and failure to manage exacerbation of MG symptoms despite plasma exchange and IVIg.

Effect on dose of prednisone

The mean dose of prednisone prior to Rituximab administration was 30.8 mg. The prednisone dose decreased a mean of 73.1% after cycle 1, 85.7% after cycle 2 and 96.7% after cycle 3. At 12 months, nine patients had a decrease in their prednisone dose, one had an increase, and one had no change in the dose. The mean dose after 12 months of initiation of Rituximab was 2.0 mg. At 18 months, one patient had no change in their dose, while the rest had consistent decline in their dose. The mean dose after 18 months of initiation of Rituximab was 1.0 mg.

Clinical response to rituximab

Eleven patients demonstrated clinical improvement following rituximab therapy, while one patient experienced clinical worsening. The mean baseline MGCS was 9.9, which decreased to 2.1 after 6 months, 0.45 at 12 months, and 0.30 at 18 months of follow-up, as illustrated in Figure 2. At 12 months, 10 patients showed improvement in MGCS, while one patient had no change compared with the previous assessment; one patient had expired prior to evaluation. At 18 months, one additional patient was lost to follow-up, and among the remaining patients, MGCS remained stable or showed sustained improvement, with no evidence of clinical deterioration. Longitudinal trends in MGCS and corticosteroid dose reduction are shown in Figure 3.

At 12- and 18-month follow-up, one patient had no symptoms or signs of myasthenia gravis for at least 1 year and was not receiving acetylcholinesterase inhibitors, steroids or steroid sparing therapy apart from rituximab. One patient had no symptoms or signs of myasthenia gravis for at least 1 year but did receive some form of therapy for myasthenia gravis other than acetylcholinesterase inhibitors. The rest of all had no symptoms causing functional limitation due to myasthenia gravis but had evidence of weakness on careful neurological examination as exhibited in Figure 4.

The clinical response to rituximab in our patients over time is summarized in Table 3.

Effect on frequency of crisis

Four patients had two crises each before initiation of Rituximab (patient 1,2, 4 and 6). Patient 1 received five sessions of plasma exchange followed by 2g/kg IVIg over five days in the second crisis. Following this crisis a few months later, he had his first cycle of rituximab (induction dose). Patient 2 had two crises within nine months of diagnosis; he received five sessions of plasma exchange as rescue therapy both times. He received his first cycle of rituximab early in the course of disease following the plasma exchange.

Patient 4 had his first crisis two years after

Table 1: Overview of key disease features

Patient	Age/Sex	Medical comorbid conditions	Duration of MG in years	Antibody status (Anti-AchR / Anti-MuSK)	Thymectomy status	Prior treatments received (Steroid sparing / Rescue therapy	Reason for the initiation of Rituximab	Total cycles of Rituximab
1	38/M	None	13	Anti-AchR Ab	Yes (Thymoma present on imaging)	MMF, AZA, PLEX (once), PLEX followed by IVIg (once)	Refractory MG	2
2	39/M	None	01	Anti-AchR Ab	Yes (Thymoma present on imaging)	AZA, MMF, PLEX (twice)	Refractory & Severe MG	3
3	38/F	Obesity	04	Anti-AchR Ab	Yes (No thymic abnormality on imaging)	CsA, PLEX (once), Crisis 2 months post-Ritux - treated with IVIg	Severe MG	6
4	55/M	HTN, dyslipidemia	10	Anti-AchR Ab	Yes (Thymic hyperplasia on imaging)	MMF, AZA, CsA, IVIg (once), PLEX (once)	Refractory MG	3
5	76/M	HTN, DM, IHD	03	Anti-AchR Ab	No	AZA, CsA	Refractory MG	4
6	60/M	HTN	02	Anti-AchR Ab	No	CsA, IVIg (once), PLEX (once)	Severe MG	5
7	74/M	DM, HTN, IHD, OSA	01	Anti-AchR Ab	No	PLEX followed by IVIg (once)	Severe MG	1
8	35/M	Asthma	03	Anti-MuSK Ab	Yes (No thymic abnormality on imaging)	AZA, MMF, CsA	Refractory MG	1
9	87/M	HTN, IHD, atrial fibrillation	04	Anti-AchR Ab	No	MMF, AZA, CsA	Refractory MG	5
10	24/F	None	03	Anti-AchR Ab	No	MMF, AZA	Severe & Refractory MG	6
11	58/F	HTN, dyslipidemia	05	Anti-MuSK Ab	No	AZA	Severe MG	5
12	60/F	HTN	01	Anti-AchR Ab	No	AZA, PLEX (once)	Refractory MG	1

HTN: hypertension, DM: Diabetes Mellitus, IHD: Ischemic Heart Disease, OSA: Obstructive Sleep Apnea, MMF: mycophenolate mofetil, CsA: cyclosporine, AZA: azathioprine, IVIg: intravenous immunoglobulin, PLEX: plasmapheresis; Anti-AchR Ab: Anti-acetylcholine receptor antibody, Anti-MuSK ab: Anti-Muscle-Specific Kinase antibody

Table 2: Patient demographics at baseline

Parameters	Values
Male: Female	2:1
Median age, years (IQR)	56 (38–74)
Median duration of MG, years (IQR)	3 (1.5-4.5)
Median disease duration at rituximab initiation, years (IQR)	1 (0.5 - 2.5)
Antibody status, n (%)	
AChR antibody positive	10 (83.3%)
MuSK antibody positive	2 (16.7%)
Number of patients with crisis prior to initiation of Rituximab, n (%)	7 (58.3%)
Mean dose of prednisone before initiation of Rituximab (in mg/day) **(n = 12)**	30.8 ± 17.7
Steroid sparing therapy prior to rituximab	
Azathioprine	7
Mycophenolate Mofetil	5
Cyclosporine	3
None	1
N.B. Patients may have received more than one steroid-sparing agent.	
MGCS score prior to rituximab initiation, mean ± SD (median [IQR]) **(n = 12)**	9.9 ± 6.0 (9 [6–15])
Comorbid medical illness	
Diabetes	3
Hypertension	8
Ischemic Heart disease	3
Atrial fibrillation	1
Obesity	1
Dyslipidemia	2
OSA	1
Asthma	1
Thymectomy	5 (41.7)
Presence of Thymoma	2 (16.7)

N.B. Data are presented as mean ± standard deviation or median (interquartile range), as appropriate. Percentages are calculated based on the total cohort unless otherwise specified.
OSA: Obstructive Sleep Apnea, Anti-AchR Ab: Anti-acetylcholine receptor antibody, Anti-MuSK ab: Anti-Muscle-Specific Kinase antibody.

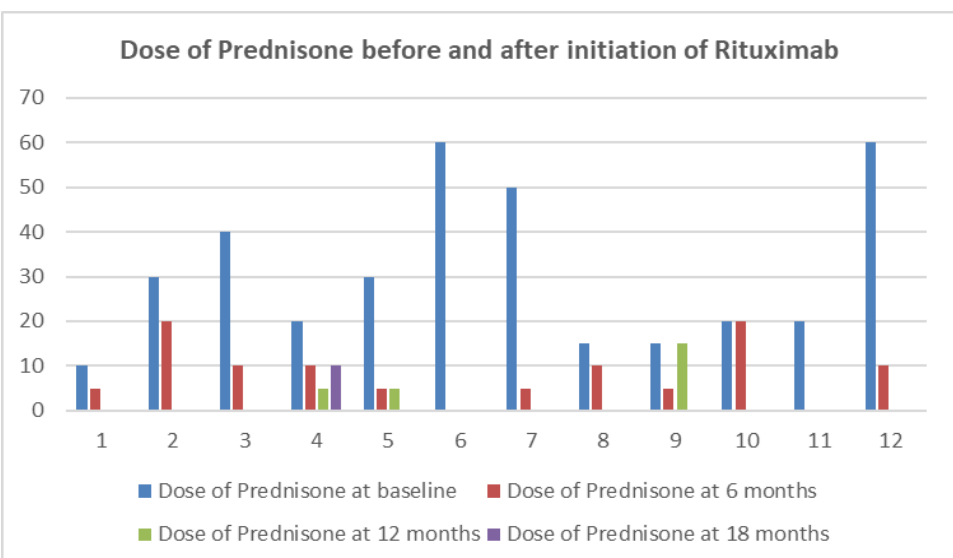

Figure 1. Dose of prednisone before and after initiation of rituximab at 6, 12 and 18 months.

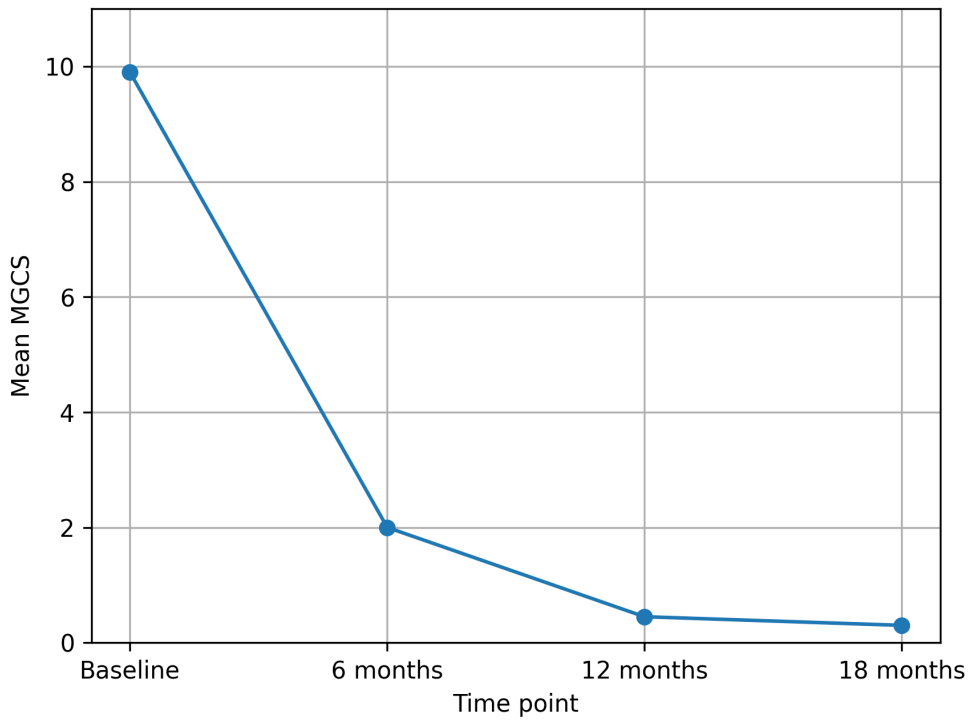

Figure 2. Change in mean MGCS following rituximab therapy.

Mean MGCS values at baseline and at 6, 12, and 18 months after initiation of rituximab therapy. One patient expired before the 12-month assessment and one was lost to follow-up before 18 months; these patients were excluded from subsequent analyses. MGCS; Myasthenia Gravis Composite Scale

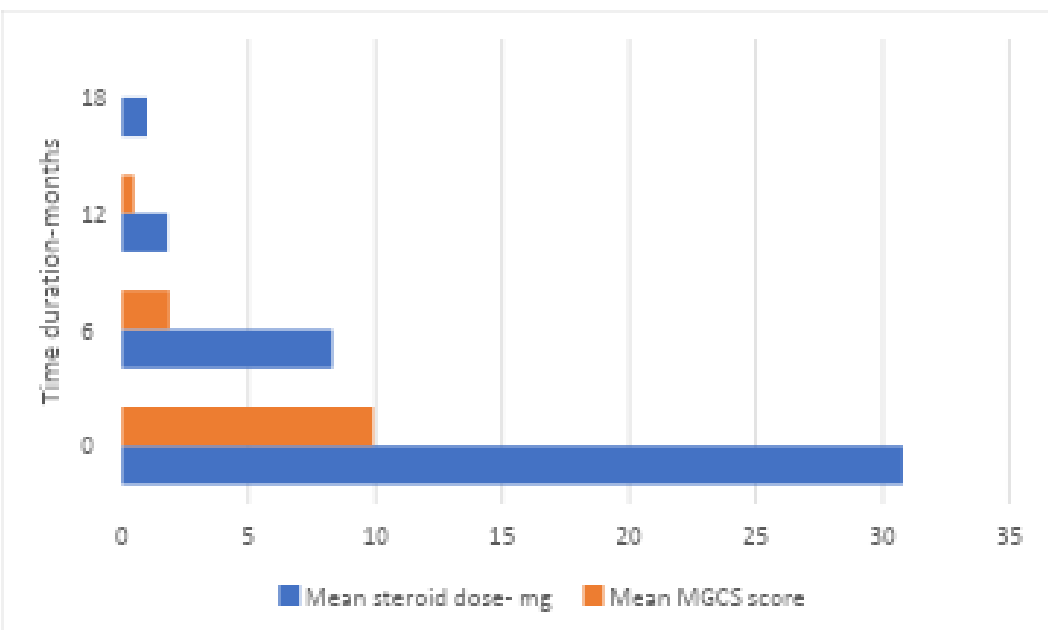

Figure 3. Trend of decrease in mean steroid dose and improvement in MGCS score at baseline, 6, 12 and 18 months. (MGCS; Myasthenia Gravis Composite Scale)

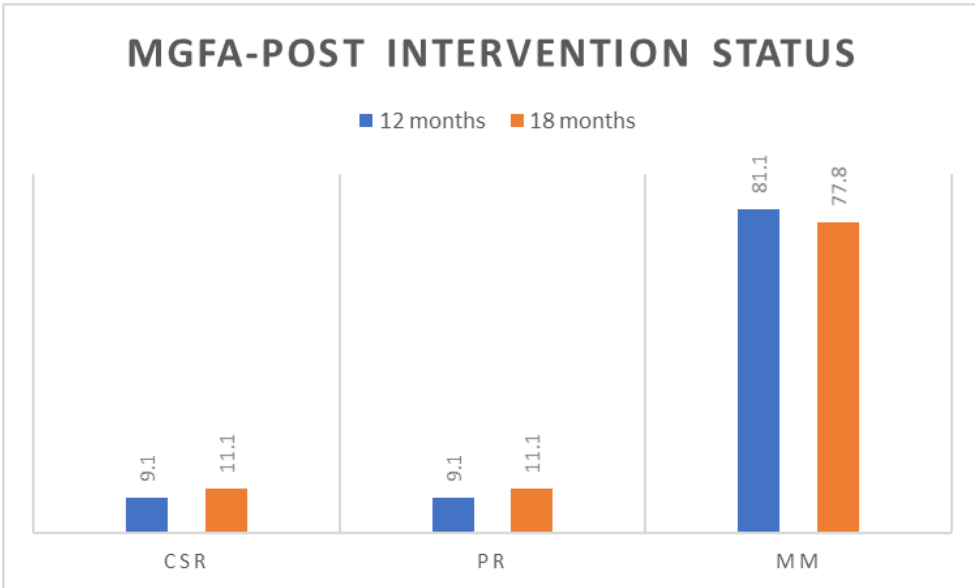

Figure 4. Percentage of patients with complete stable remission (CSR), pharmacological remission (PR) and minimal manifestations (MM).

diagnosis and received 2g/kg IVIg followed by another crisis seven years later for which he received five sessions of plasma exchange. During the second crisis, he was also treated with three doses of 1 g methylprednisolone pulse therapy. His bulbar symptoms did not improve despite rescue therapy and therefore he was given his induction dose of rituximab in the same admission. He had thymectomy two months later to further augment disease control. Patient 6 had two crises in the first year of diagnosis and received IVIg in the first crisis and plasma exchange in the second while being on immunosuppressive therapy with cyclosporine. plasma exchange after the second crisis was followed by 3 doses of 1 g methylprednsiolone as the patient's symptoms remained unresolved. Within the same admission, the first cycle of rituximab was administered. After initiation of rituximab, the above patients did not have any other crisis.

Patients 3, 7 and 12 had one crisis each before initiation of rituximab. Patient 3 received plasma exchange as rescue therapy for her first crisis, while she was on immunosuppressive therapy with cyclosporine. Although imaging did not reveal a thymoma, she underwent thymectomy for symptom control. One year after surgery, her prednisolone and pyridostigmine doses could not be reduced, so rituximab was initiated. Two months later, she developed a second crisis that was treated with IVIg (2 g/kg over 5 days). After receiving two cycles of rituximab, she achieved good symptom control, was able to discontinue steroids, and has not had any further myasthenic crises.

Patient 7 was admitted with a crisis at the time of diagnosis of myasthenia gravis. IVIg (2g/kg in five divided doses) was administered, and it was followed by five sessions of plasma exchange as symptoms were refractory to initial rescue therapy. Rituximab was given during the same admission. Six months after rituximab, his bulbar symptoms had shown significant improvement. However, he died due to a cause unrelated to myasthenia gravis, before receiving his second cycle of Rituximab.

Patient 12 had her first myasthenic crisis within three months of diagnosis, while receiving high dose steroids (60 mg/day) and azathioprine. Five sessions of plasma exchange were administered as rescue therapy and azathioprine was continued. Soon after, she developed thrombocytopenia with azathioprine, and it had to be stopped. Patient 12 was then started on the first cycle of rituximab. Five days after receiving the induction therapy of rituximab, she developed herpes zoster ophthalmicus which was treated with acyclovir. Six months after follow-up, she had minimal symptoms of MG, prednisone dose was decreased to 10 mg/day, and there were no new episodes of myasthenic crisis. This patient

Table 3: Clinical outcomes following rituximab therapy at baseline, 6, 12, and 18 months

	Patients with crisis	Mean steroid dose- mg	Decrease in steroid dose - %	MGCS score, mean ± SD (median [IQR])	Patients achieving MGFA-PIS-CSR n (%)	Patients achieving MGFA-PIS-PR n (%)	Patients achieving MGFA-PIS-MM n (%)			
							MM0	MM1	MM2	MM3
Baseline (n=12)	7 (58.3)	30.8 ± 17.7	-	9.9 ± 6.0 (9 [6–15])	-	-	-	-	-	-
At 6 months (n=12)	-	**8.3 ± 6.5**	73.1	2.1 ± 3.3 (1 [0–3])	-	-	-	-	-	-
At 12 months (n=11) *		2.0 ± 0.4	85.7	0.45 ± 0.93 (0 [0–0])	9.1	9.1	0.0	18.2	63.6	0.0
At 18 months (n=10) *	1 (8.3)	1.0 ± 3.2	96.7	0.30 ± 0.95 (0 [0–0])	11.1	11.1	0.0	22.2	55.6	0.0

did not follow up for further administration of rituximab due to financial constraints.

Patients 5, 8, 9, 10 and 11 did not have any episode of myasthenic crisis during their course of illness; before and after rituximab therapy.

Safety and adverse events

Three patients reported adverse events with rituximab. Of those, one had arthralgias. Two patients experienced opportunistic infections.

Patient 12 developed herpes zoster ophthalmicus 5 days after administration of the induction dose of rituximab (cycle 1) as mentioned above. She received oral acyclovir and her infection improved. Patient 1 developed a flare of latent HCV infection for which his treatment was temporarily withheld. The rest of the patients did not have any significant adverse effects.

DISCUSSION

In this retrospective case series of 12 patients with MG, rituximab demonstrated substantial clinical efficacy and a pronounced steroid-sparing effect, particularly among individuals with treatment-refractory disease and severe presentations at onset. The cohort had a median age of 56 years and median disease duration of 3 years and was predominantly AChR-antibody positive (83.3%). Most patients had either failed or were intolerant to one or more steroid-sparing immunosuppressants prior to rituximab initiation, rendering them ideal candidates for advanced immunomodulatory therapy. While four patients received rituximab after failing a single steroid-sparing agent, others underwent multiple immunosuppressant trials before its initiation, reflecting individualized clinical decision-making based on disease burden, side-effect profiles, and physician discretion.

Importantly, early use of rituximab after the failure of one steroid sparing agent was associated with clear clinical benefit. Though formal cost analysis was not undertaken, early intervention was linked to reduced need for adjunctive oral steroid sparing medications, fewer hospital visits for rescue therapy, and improved adherence. These observations suggest potential cost-effectiveness, which is particularly relevant in low- and middle-income countries (LMICs). Prior reports have similarly shown that post-rituximab treatment leads to significant reductions in steroid and immunosuppressant use and decreased reliance on IVIG or plasma

exchange; highlighting both clinical and logistical advantages of timely biologic intervention.[7]

A key finding in our study was the marked reduction in corticosteroid burden. The mean daily prednisone dose decreased from 30.8 mg at baseline to 8.3 mg at 6 months and further to 2.0 mg at 12 months—an 85.7% overall reduction. At 18 months, the percentage dose reduction of prednisone was 96.7%. These results are congruent with prior studies: Zhong *et al*. reported a ≥75% dose reduction in over 80% of patients within a year, while Choi *et al.* observed a decrease from 28 mg to 7.5 mg at 6 months.[8,9] Given the long-term adverse effects associated with corticosteroids, such reduction has meaningful implications for patient outcomes and quality of life.

Clinical improvement was further corroborated by objective measures in our study. The mean MGCS improved from 9.9 at baseline to 0.45 at 12 months. At 12- and 18-month follow-up, one (10%) patient achieved pharmacologic remission and one (10%) attained complete stable remission. The rest of the patients experienced minimal manifestations only. These outcomes echo findings from Nowak *et al*. and Baggi *et al*., who reported sustained clinical and functional improvement in rituximab-treated patients, with antibody profile influencing treatment response.[10] Notably, our study also documented a reduction in myasthenic crisis frequency—from 58.3% prior to treatment to 8.3% post-rituximab—underscoring its role in preventing life-threatening exacerbations and improving long-term disease control, especially in resource limited settings.

While rituximab is typically considered most effective in MuSK-antibody positive MG due to its rapid and sustained efficacy[11], our findings demonstrate that significant benefit can also be achieved in AChR-positive individuals. This aligns with emerging evidence that rituximab may be effective across antibody subtypes, particularly in those with refractory disease or intolerance to standard therapies.

Although rituximab was generally well tolerated, three patients (25%) experienced adverse effects. In two of them, adverse effects were significant enough to warrant temporary discontinuation of treatment. However, none required permanent cessation, and all were able to resume therapy following appropriate management. This aligns with broader safety data indicating that most rituximab-related adverse events are manageable with supportive care and do not necessitate long-term treatment withdrawal.[12]

This study is subject to several limitations, including a small sample size, its retrospective design, loss to follow-up in two patients at the 12-month assessment, and the lack of long-term follow-up data. We restricted the study to seropositive patients, which prevented assessment of rituximab outcomes in seronegative MG. Nonetheless, conducted within an LMIC setting, this study adds to the growing body of real-world evidence supporting the early use of rituximab in MG.

Despite systemic and economic constraints, rituximab demonstrated notable clinical benefit, steroid-sparing potential, and good tolerability. These findings support the incorporation of rituximab earlier in the treatment algorithm for selected patients and highlight the need for prospective, controlled studies to better define its optimal role and long-term utility in diverse healthcare environments.

DISCLOSURE

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Conflict of interests: None

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