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ORIGINAL RESEARCH

Efgartigimod in Patients with Generalized Myasthenia Gravis Refractory or Intolerant to IVIg

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ABSTRACT

***Introduction*:** Generalized myasthenia gravis (gMG) is a rare chronic autoimmune disorder of the neuromuscular junction caused by pathogenic autoantibodies directed against a postsynaptic target. The therapeutic landscape of gMG has recently expanded with the introduction of FcRn inhibitors. This study aimed to assess the real-world effectiveness and safety of efgartigimod (EFG) in AChR-positive gMG patients who failed or were intolerant to intravenous immunoglobulin (IVIg).

***Methods*:** EFG was administered as four consecutive weekly intravenous infusions at 10 mg/kg. Treatment efficacy was evaluated using the Myasthenia Gravis Activity of Daily Living (MG-ADL) and Myasthenia Gravis quantitative (QMG) scales at baseline and after 4 weeks. Incidence of adverse events and prednisone use were collected at each time point.

***Results*:** Thirteen patients (6 women and 7 men, mean age 52.9 years) received EFG following IVIg therapy. After one treatment cycle, both MG-ADL and QMG scores showed significant clinical improvement. MG-ADL responder rate (MG-ADL reduction > 2) was 84.6% while 69.2% on QMG (QMG reduction > 3). Minimal symptom expression was reached in one patient, accompanied by a mean reduction in daily prednisone dose of 6.9 mg.

***Conclusion*:** In this real-world cohort, efgartigimod demonstrated a rapid and meaningful clinical benefit with a favorable tolerability profile in patients with AChR-positive gMG after IVIg failure or intolerance. These findings support the potential role of EFG as an effective therapeutic option in this difficult-to-treat population.

Keywords: Efgartigimod; FcRn inhibition; Rescue therapy; Myasthenia gravis; AChR antibodies; Myasthenic crisis

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Key Points

Why carry out this study?

Patients with generalized myasthenia gravis who are refractory or intolerant to intravenous immunoglobulin represent a subgroup with limited therapeutic options for rescue treatment

FcRn inhibition with efgartigimod offers a targeted strategy to rapidly reduce pathogenic IgG autoantibodies involved in disease activity

The aim of this multicenter real-world study was to evaluate the efficacy and safety of efgartigimod in patients with AChR-positive generalized myasthenia gravis after IVIg failure or intolerance

What was learned from the study?

Efgartigimod treatment resulted in clinically meaningful and statistically significant improvements in MG-ADL and QMG scores after a single treatment cycle

A high responder rate and a favorable safety profile were observed, supporting the potential role of efgartigimod as an effective rescue therapy in severe generalized myasthenia gravis

INTRODUCTION

Generalized myasthenia gravis (gMG) is a rare autoimmune disease that targets the neuromuscular junction, causing fatigable weakness of voluntary muscles, worsening with repetitive activities, heat and stress while improving at rest [1]. Most patients with gMG (85%) are positive for acetylcholine receptor antibodies (AChR-Ab), which interfere with neuromuscular transmission through several mechanisms [2]. Consequently, patients experience weakness and fatigability affecting ocular, bulbar, respiratory, axial and limb muscles. In most severe cases, generalized muscle weakness may lead to myasthenic crisis due to respiratory failure [3]. Myasthenic crisis (MC) occurs in approximately 15%–20% of patients with gMG, usually within the first 3 years of the disease course [4]. Conventional therapies for gMG include acetylcholinesterase inhibitors and prednisone or non-steroidal immunosuppressant therapy (NSIST), which may be associated with significant side effects. In patients with significant clinical worsening despite IS and corticosteroids or in those with impending MC, rescue therapies, such as plasma exchange (PLEX) or intravenous immunoglobulin (IVIg), are commonly employed with favorable clinical outcomes [5]. Despite usually being effective, IVIg therapy is associated with several well-documented adverse effects that may lead to early discontinuation [6]. Minor IVIg-related adverse events include fever or chills, headaches, nausea, allergic reaction [7], flu-like symptoms, eczematous skin reactions, electrolyte disturbance and transient leukopenia. In addition, considering that immunoglobulins are derived from human plasma, it is possible that at times these are scarcely available on the pharmaceutical market. Therefore, it is useful to evaluate safe and effective therapeutic alternatives in patients with gMG.

Among these, in recent years, biologic therapies primarily targeting neonatal Fc receptor (FcRn) blockade [9] stand out.

The neonatal Fc receptor (FcRn) is an MHC class I-like molecule that plays a critical role in maintaining both IgG and albumin levels by rescuing these molecules from lysosomal degradation within cells. FcRn blockade allows targeted reduction of all IgG subtypes without decreasing concentrations of other Ig isotypes [10]. Considering the direct pathogenicity of IgG in gMG, blocking FcRn allows targeted reduction of pathogenic IgG autoantibodies in gMG [11]. EFG is a human IgG1-derived Fc fragment that blocks FcRn function, resulting in a rapid lowering of endogenous IgG levels [12]. The ADAPT phase 3 trial demonstrated that EFG is well tolerated and effective in patients with gMG [13]. However, in the phase III trial, patients were enrolled after treatment with corticosteroids or IS, even in the absence of previous IVIg treatment. Immunoglobulins are widely used to manage patients with gMG with clinical worsening. However, cases of patients who show

refractoriness or intolerance to immunoglobulins are not explored in depth in the literature.

Therefore, since patients with gMG present a high burden of comorbidity that might limit the use of rescue therapy in case of impending MC [14], EFG could represent an interesting option due to its safety profile [15]. Our study aims to evaluate the validity of a therapeutic strategy in patients with difficult-to-manage gMG. There is some debate among clinicians regarding the use of EFG after IVIg because it may counteract its beneficial long-term effects, and there is concern that a rapid reduction in serum IgG might also cause paradoxical worsening due to the possible loss of IVIg's early effects [16–21]. Considering this, the efficacy of EFG in this setting should be investigated and evaluated to verify whether the improvements or detrimental effects are more significant. This multicenter observational study aims to evaluate the efficacy and safety of EFG in patients with generalized myasthenia gravis who experienced no clinical benefit from or were intolerant to IVIg because of adverse events.

METHODS

Study Design and Objectives

We conducted a multicenter, observational, retrospective study to evaluate the safety and efficacy of EFG in patients with IVIg refractory or intolerant to gMG in a real-world clinical setting. Three Italian referral centers for the diagnosis and treatment of myasthenia gravis participated in the study (UOSD Neurofisiopatologia, AUOP Giaccone, Palermo; UOC Neurophysiopathology, AORN Cardarelli, Naples; Neurology Unit, Department of Medicine, Surgery and Dentistry "Scuola Medica Salernitana," University Hospital San Giovanni di Dio e Ruggi D'Aragona, Salerno). The primary aim of this study was to evaluate the efficacy and safety of efgartigimod in patients with gMG after unsuccessful treatment with IVIg.

Participants

Patients were considered eligible for inclusion if they met the following criteria:

- Age ≥ 18 years and provided informed consent to participate in the study
- Diagnosis of gMG according to standard guidelines and recommendations
- Positive serum antibody test for anti-AChR
- Treatment with EFG as part of clinical practice
- Lack of clinical benefit following administration of one cycle of IVIg for 3 weeks, described as no improvement in the clinical scales administered (improvement on MG-ADL score < 2 points and/or on QMG < 3 points)
- Rapid deterioration of myasthenic symptoms or impending myasthenic crisis, independently of the timing
- Lack of tolerability, adverse events or intolerance resulting in the early discontinuation of IVIg or contraindication

Procedures

We enrolled patients affected by acetylcholine receptor antibody-positive (AChR–Ab+) gMG previously treated with one therapeutic cycle of IVIg at a dosage of 0.4 g/kg for 5 days. Efgartigimod was administered according to the schedule at a dosage of 10 mg/kg once weekly for 4 consecutive weeks at least 4 weeks after IVIg treatment.

The treatment efficacy was evaluated using clinical scales (MG-ADL, QMG) at baseline and at the end of the first cycle of treatment. Patients were stratified at baseline using the MGFA scale, following the Myasthenia Gravis Foundation of America criteria. Serum IgG levels and general laboratory blood examinations were assessed during treatment.

Data Collection

Data were retrospectively collected from medical records at each participating center between October 2023 and July 2025. EFG was prescribed as part of routine clinical practice. Some patients received treatment within an Expanded Early Access Program for efgartigimod IV for patients with generalized myasthenia gravis (GENERATIVE protocol).

The following variables were included in the data collection: current age, gender, and weight; comorbidities; age at onset of MG and age at treatment initiation; disease duration at baseline; MGFA clinical classification at disease onset and at baseline; history of thymectomy and thymic status; previous treatments, including prednisone, NSIST, and chronic IVIg or PLEX; the number of cycles administered during the year before starting EFG. Previous and baseline dosages of prednisone and NSIST were recorded. During EFG therapy, we recorded changes in baseline medications, adverse events, and treatment discontinuations.

Efficacy and Safety Assessment

Clinical efficacy was evaluated using the Myasthenia Gravis-Activities of Daily Living Scale (MG-ADL) and Quantitative Myasthenia Gravis Scale (QMG) score, administered at baseline and at completion of the first EFG treatment cycle (week 4). A clinically relevant response was defined as a reduction of at least 2 points on the MG-ADL scale or at least 3 points on the QMG score. The occurrence of minimal symptom expression (MSE) was evaluated, considered 0 or 1 points on the MG-ADL scale. As part of the safety assessment, adverse events (AEs) related to efgartigimod were recorded during treatment.

Statistical Analysis

Baseline demographic and clinical characteristics were summarized using descriptive statistics, with categorical variables reported as frequencies and percentages and continuous variables as mean ± standard deviation (SD). Distributional assumptions for MG-ADL and QMG scores were assessed using boxplots and the Shapiro-Wilk normality test. In the absence of significant outliers and when normality criteria were met, within-group changes over time were analyzed using repeated-measures analysis of variance (ANOVA). Treatment efficacy was further explored by evaluating the change in clinical scale scores between baseline and week 4 (ΔMG-ADL = MG – ADLW4 – MG – ADLT0). To investigate the potential relationship between clinical improvement and patient-related variables, additional ANOVA analysis was performed, including sex, age at disease onset, thymectomy status, MGFA classification at disease onset and presence of comorbidities as covariates.

Ethics Approval

All human and animal studies were approved by the appropriate ethics committee and were conducted in accordance with the ethical standards set out in the 1964 Declaration of Helsinki and its later amendments. The study was approved by the appropriate Ethics Committee "Palermo I" (protocol code 11/2022), approved on 12 December 2022, and it was conducted in conformity with the principles of the Declaration of Helsinki. All participating centers adhered to the study protocol. Signed patient-informed consent forms were obtained for the study and publication.

RESULTS

Demographic Characteristics

We enrolled 13 patients with gMG from 41 patients (31%) treated with EFG in the three referral centers. The group's demographic characteristics are reported in Table 1.

The cohort consisted of six women (46%) and seven men (54%) with an average age of 53 ± 15 years; 53% of the test patients were affected by thymoma; 84% had comorbidities. The main concomitant therapies received by study patients were pyridostigmine (69%) and prednisone (84%). Seventy percent of patients had class III MGFA (of which 78% had IIIA and 12% IIIB), three patients (23%) had class IVB MGFA, and only one patient (8%) had class V MGFA. According to the Italian criteria for prescription, all patients presented a MG-ADL score > 5 at baseline, and EFG was administered after initiation of steroids, pyridostigmine and/or IS.

All the patients included in the study had received at least one intravenous administration of immunoglobulin (IVIg) before efgartigimod as treatment for gMG. The median time between

Table 1 Main baseline demographic and clinical characteristics of patients treated with efgartigimod

Clinical variable	Total ($N=13$)
Age (years)	52.9 (15.8)
Age at onset (years)	42.5 (20.3)
Gender (male, %)	7 (53.8)
Body weight (kg)	73.3 (15.8)
Disease duration (years)	10.4 (12.5)
Thymoma (*n*, %)	7 (53.8)
Comorbidity (*n*, %)	11 (84.6)
Prednisone dose at baseline (mg/day)	22.0 (20.9)
MG-ADL at baseline	10.5 (3.2)
QMG at baseline	15.7 (4.7)
Concomitant treatments at baseline	
Pyridostigmine (*n*, %)	9 (69.2)
Prednisone (*n*, %)	11 (84.6)
Azathioprine (*n*, %)	2 (15.4)
Mycophenolate mofetil (*n*, %)	3 (23.1)
Methotrexate (*n*, %)	1 (7.7)
MG subtype (onset)	
Early onset (*n*, %)	8 (61.5)
Late onset (*n*, %)	5 (38.5)
MGFA baseline	
IIA (*n*, %)	0
IIB (*n*, %)	0
IIIA (*n*, %)	7 (53.8)
IIIB (*n*, %)	2 (15.4)
IVA (*n*, %)	0
IVB (*n*, %)	3 (23.1)
V (*n*, %)	1 (7.7)
Unsatisfactory IVIg response (*n*, %)	9 (69.2)
IVIg intolerance (*n*, %)	4 (30.8)

Continuous variables are expressed as mean with standard deviation (in brackets) and categorical variables as numeric count and percentages

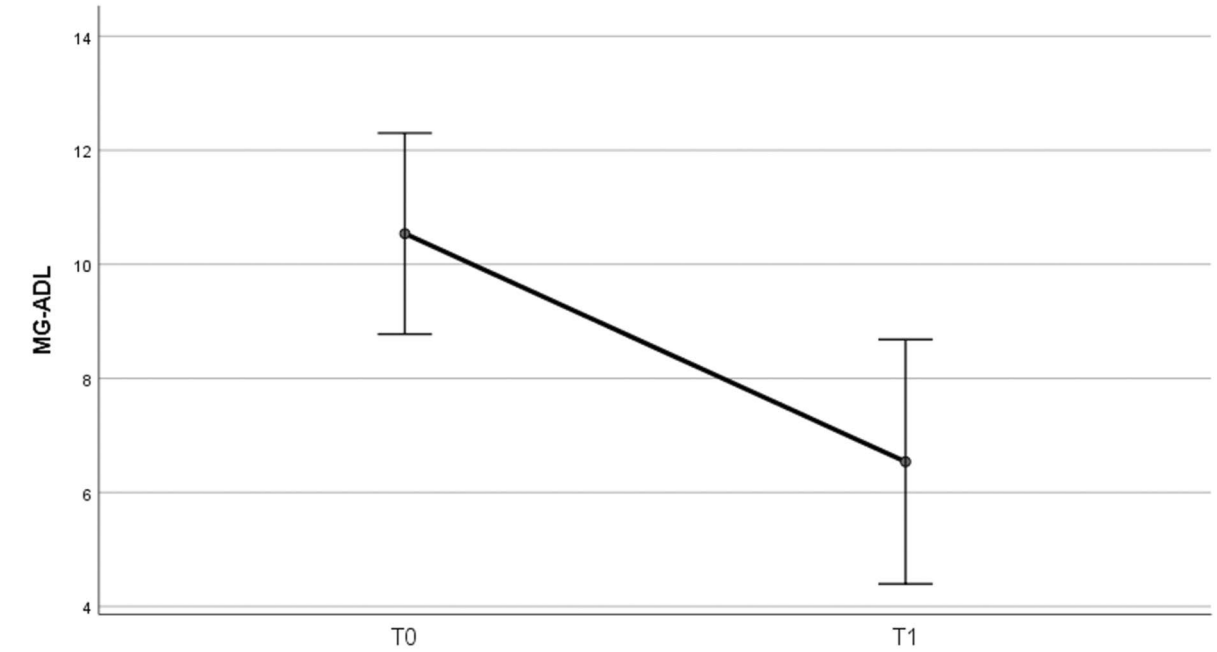
IVIg intravenous immunoglobulins, *MGFA* Myasthenia Gravis Foundation of America, *MG-ADL* Myasthenia Gravis Activity of Daily Living, *QMG* Myasthenia Gravis quantitative scale

the last IVIg cycle and EFG initiation was 61 (IQR 35–109; range 30–491) days. In particular, nine (70%) patients did not achieve a significant clinical response following IVIg, while AEs after IVIg were experienced by four (30%) patients (intractable vomiting and headache, hypokalemia, hemolytic jaundice, severe hemolytic anemia).

Efficacy and Safety Assessment

All patients experienced clinical improvement after EFG treatment with a significant reduction in MG-ADL scores following administration ($F=16.64$, $p=0.002$; Fig. 1, Table 2). Analysis of MG-ADL and QMG scores calculated at the beginning and end of the first EFG cycle revealed an average reduction in MG-ADL score of 4 (SD 3.5) and QMG score of 3.5 (SD 1.9).

In particular, the administration of EFG to the patient with myasthenic crisis resulted in clinical improvement, demonstrated by the reduction in MG-ADL and QMG values, especially on the respiratory item. The MG-ADL reduction at the end of the EFG cycle was not associated with sex ($p=0.76$), age at disease onset ($p=0.35$), MGFA classification ($p=0.50$), thymoma ($p=0.16$) and comorbidity ($p=0.13$). Also, the QMG score improved after treatment ($F=41.4$, $p<0.0001$; Fig. 1, Table 2). The QMG reduction was not dependent on sex ($p=0.079$), age at disease onset ($p=0.93$), MGFA classification ($p=0.53$), thymoma ($p=0.28$) or comorbidity ($p=0.44$). Responder rate to MG-ADL was 84.6%, while on the QMG it was 69.2% (Table 2). Also, MSE was achieved in one patient after the first EFG cycle (8%). A mild reduction in concomitant prednisone was obtained after at least one complete course of EFG treatment in four patients (30.7%), with an overall mean reduction of about 7 mg (Table 2). In terms of safety, 92.3% of patients treated with EFG reported no AEs. Urticaria with angioedema (7.7%) was reported after EFG in one patient. During follow-up, patients were observed for a median of 9.4 (IQR 4.3–19.1; range 0.7–29.9) months and received a median of three EFG treatment cycles (IQR 2–7; range 1–11). At the last available evaluation, clinical response was maintained in 72.7%



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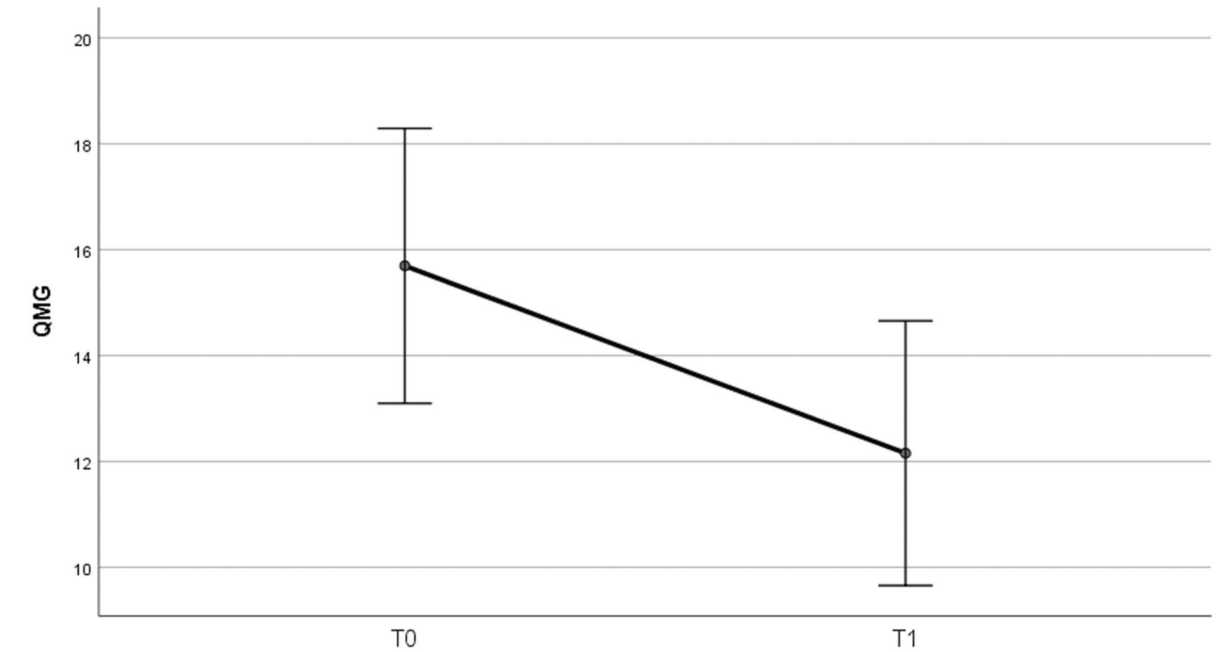

B

Fig. 1 **A**, **B** MG-ADL and QMG reduction at baseline (T0) and after 4 weeks of follow-up (T1). *MG-ADL* Myasthenia Gravis Activity of Daily Living, *QMG* Quantitative Myasthenia Gravis

Table 2 MG-ADL and QMG baseline mean values and differences in the means of patients who completed follow-up

	T0	T1	Delta	Responder rate (*n*, %)
MG-ADL	10.5 (3.2)	6.5 (3.8)	4.0 (3.5)	11 (84.6)
QMG	15.7 (4.7)	12.2 (4.5)	3.5 (1.9)	9 (69.2)
Prednisone (mg)	22.0 (20.9)	15.1 (11.6)	6.9 (15.6)	4 (30.7)

Values are expressed as mean ± SD

MG-ADL Myasthenia Gravis Activity of Daily Living, *QMG* Quantitative Myasthenia Gravis

of patients based on the MG-ADL and QMG responder rate. The improvement remained clinically meaningful, with a mean reduction from baseline of 3.45 (SD 2.65) points in MG-ADL and 3.63 (SD 2.50) points in QMG.

DISCUSSION

IVIg has long represented a cornerstone in the management of severe gMG, especially in patients experiencing clinical worsening or MC [6]. Their immunomodulatory effects on plasma cells are mediated through multiple mechanisms, including $F\ (ab')_2$- and Fc-dependent pathways such as Fcγ receptor blockade, modulation of dendritic cells, expansion of regulatory T cells, and inhibition of the neonatal Fc receptor (FcRn) [22]. However, despite their widespread use, the exact mechanism of IVIg action remains incompletely understood, but FcRn blockade appears to play a central role, overlapping with efgartigimod's mechanism of action [16]. Limitations of IVIg include delayed therapeutic onset, inconsistent efficacy and relatively frequent adverse events (AEs), particularly in younger women or patients with significant comorbidities [6]. These considerations highlight the unmet need for alternative or adjunctive therapeutic strategies in patients with refractory or IVIg-intolerant gMG.

On the other hand, FcRn inhibitors interact with the FcRn in a pH-independent manner, maintaining high binding affinity under physiological conditions [23]. Their mechanism of action primarily results in a selective reduction of circulating IgG levels and interference with IgG transfer across barriers, while largely preserving other immunoglobulin isotypes.

Our multicenter, real-world experience provides new insights into the potential role of EFG in this specific and clinically challenging MG population. The most striking finding of this study is that all patients experienced clinical benefit from EFG, as evidenced by significant improvements in both the MG-ADL and QMG scores after only a single treatment cycle. The responder rates were high (84.6% for MG-ADL and 69.2% for QMG), with one patient achieving MSE. Importantly, EFG administration also allowed a reduction in concomitant prednisone use in nearly one-third of patients (30%), reinforcing its potential steroid-sparing effect.

Equally noteworthy is the excellent safety profile observed. Except for a single case of urticaria with angioedema, no other relevant AEs were reported, and no infectious complications occurred despite the cohort's high comorbidity burden [21, 27]. This result is particularly relevant given that our population included patients with long-standing, severe disease and prior failure or intolerance to rescue therapy—individuals typically considered at higher risk for drug-related complications. The observation of efficacy and safety, even in one patient with MC (MGFA class V), also confirms the potential role for EFG in MC [19].

Therefore, from this perspective, EFG may represent a promising therapeutic option for the treatment of gMG in patients who are refractory or intolerant to IVIg.

Our results are in line with emerging evidence from recent trials and real-world reports, as demonstrated in a recent Chinese study investigating the comparative efficacy and safety of

efgartigimod versus IVIg in the treatment of elderly AChR-antibody-positive patients with gMG [27]. The authors found that EFG showed greater efficacy in reducing MG-ADL scores than IVIg at weeks 4 and 8, with a good safety profile. In patients evaluated over the long term, the clinical response to EFG was maintained, as evidenced by reductions in MG-ADL and QMG scores. These findings suggest that, in patients who are refractory or intolerant to IVIg, EFG may provide not only a rapid short-term improvement but also a durable clinical benefit when treatment cycles are administered according to the patient's needs.

This study has several limitations that should be acknowledged. First, the small sample size limits the generalizability of these results, even though the multicenter design is a strength of this study. Second, laboratory assessments (including antibody titers and hematochemical parameters) were not centralized, introducing possible variability in measurements. Third, the retrospective, observational design may have led to underreporting of minor AEs and does not allow definitive conclusions on causality.

CONCLUSION

In this multicenter real-world study, EFG demonstrated a rapid and clinically meaningful improvement in patients with gMG who were refractory or intolerant to IVIg. Significant reductions in MG-ADL and QMG scores were observed after a single treatment cycle, with high responder rates and a favorable safety profile. In some patients observed during the follow-up period, prolonged treatment with EFG allowed maintaining the clinical benefit over time. Importantly, EFG was well tolerated even in patients with severe disease and a high burden of comorbidities, and it allowed reduced concomitant corticosteroid use in a subset of patients. Although limited by the small sample size and retrospective design, these findings suggest that EFG may represent a valuable rescue or alternative therapeutic option in challenging clinical scenarios, including patients unresponsive to IVIg. Nevertheless, prospective studies involving larger cohorts and longer follow-up are warranted to confirm these observations, further elucidate the long-term safety profile, and better define the role of EFG within the therapeutic algorithm of gMG, particularly as an alternative to IVIg in acute or rescue settings.

In conclusion, it is important to emphasize that the lack of clinical response to immunoglobulin treatment is not a predictor of the effectiveness of EFG. In this regard, further studies may be useful for understanding the mechanisms that potentially cause immunoglobulin refractoriness.

Author Contributions. Flora D'Amico, Nicasio Rini and Vincenzo Di Stefano planned the study. Nicasio Rini contributed to the study conception and design. The literature search and data analysis were performed by Vincenzo Di Stefano, Nicasio Rini and Flora D'Amico. Analysis and interpretation of data were performed by Vincenzo Di Stefano, Carmen Erra, Claudia Vinciguerra, Francesco Habetswallner and Filippo Brighina. The first draft of the manuscript was written by Nicasio Rini, Flora D'Amico and Sofia Campo. Vincenzo Di Stefano, Claudia Vinciguerra, Carmen Erra, Liliana Bevilacqua, Paolo Barone, Francesco Tuccillo, Francesco Habetswallner and Filippo Brighina revised the work. All authors read and approved the final manuscript. Vincenzo Di Stefano submitted the study.

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Declarations

Conflict of interest. On behalf of all authors, the corresponding author states that Flora D'Amico, Sofia Campo, Nicasio Rini, Claudia Vinciguerra, Liliana Bevilacqua, Carmen Erra, Paolo Barone, Francesco Tuccillo, Francesco

Habetswallner, Filippo Brighina and Vincenzo Di Stefano have no conflicts of interest to declare.

Ethical approval. All human and animal studies have been approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. The study was approved by the appropriate Ethics Committee "Palermo I" protocol code 11/2022, approved on 12 December 2022, and it was conducted in conformity with the Declaration of Helsinki principles. All participating centers adhered to the study protocol. Patient informed consent was obtained and signed for the study and publication.

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