Cureus
Part of SPRINGER NATURE

Open Access Case Report

Autoimmune Overlap of Systemic Lupus Erythematosus and Myasthenia Gravis: A Case Report and Emerging Therapeutic Strategies

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Review began 10/08/2026
Review ended 10/15/2026
Published 10/20/2026

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DOI: 10.7759/cureus.118264

Abstract

Systemic lupus erythematosus (SLE) and myasthenia gravis (MG) are autoimmune disorders that predominantly affect women and may coexist, though their overlap remains rare. Shared mechanisms, including loss of immune tolerance, B-cell hyperactivity, and dysregulated cytokine signaling, are thought to underlie this association. We report the case of a 56-year-old woman diagnosed with seronegative MG after presenting with progressive proximal weakness, ocular involvement, and electrophysiologic evidence of neuromuscular junction dysfunction. She was stabilized on IVIG, pyridostigmine, and azathioprine. Four years later, she developed a malar rash, polyarthralgia, and bilateral upper-extremity numbness; serologic evaluation confirmed SLE and revealed newly detectable acetylcholine receptor binding antibodies, representing her first positive MG serologic marker. Hydroxychloroquine (HCQ) and methotrexate were added to her regimen. Efgartigimod alfa, a neonatal Fc receptor (FcRn) antagonist, was subsequently initiated for persistent MG and produced marked improvement in MG Activities of Daily Living (MG-ADL) scores. Her SLE-related joint symptoms also improved, although SLE-directed therapy had been adjusted during the same period. However, cyclic dosing resulted in consistent symptomatic wear-off in the days preceding each treatment cycle. Transition to nipocalimab, a fixed-schedule anti-FcRn monoclonal antibody providing continuous IgG suppression, is under multidisciplinary consideration. This case highlights the diagnostic and therapeutic complexity of SLE-MG overlap and suggests that FcRn inhibition may offer a mechanistically rational approach to IgG-mediated disease in both conditions, a possibility that warrants further study.

Categories: Neurology, Rheumatology, Internal Medicine
Keywords: autoimmune, auto immune overlap syndrome, fcrn inhibitor, myasthenia, sle

Introduction

Systemic lupus erythematosus (SLE) and myasthenia gravis (MG) are distinct autoimmune diseases that predominantly affect women and frequently present during young to middle adulthood. The Lupus Foundation of America estimates that approximately 1.5 million Americans live with some form of lupus, encompassing systemic and cutaneous variants, while recent epidemiologic analyses using stricter case definitions suggest that about 200,000 to 300,000 individuals in the United States have SLE [1]. In contrast, MG is less common, affecting an estimated 80,000 Americans, with prevalence steadily rising due to improved diagnostic recognition and longer survival [2]. Autoimmune diseases often cluster, and patients with one autoimmune disorder have an increased likelihood of developing another. Although SLE and MG are individually well characterized, their coexistence remains rare, with most evidence limited to case reports and small series. Systemic lupus erythematosus is a systemic autoimmune disease characterized by multisystem inflammation and the production of pathogenic autoantibodies [3], whereas MG is an organ-specific autoimmune disorder targeting the neuromuscular junction. Despite their differing pathophysiology, both conditions may share underlying immune dysregulation involving B-cell hyperactivity, impaired immune tolerance, and abnormal cytokine signaling [4]. Because overlap occurs infrequently, the clinical presentation, optimal management, and long-term outcomes of patients with coexisting SLE and MG are not well defined. Moreover, therapies used to treat one disease may influence the course of the other, adding complexity to treatment decisions. Here, we describe a middle-aged woman initially diagnosed with seronegative MG who subsequently developed SLE four years later. We also review the available literature on SLE-MG overlap and discuss emerging therapeutic options, including neonatal Fc receptor (FcRn) antagonists that may provide targeted benefit in patients with dual IgG-mediated autoimmune disease.

Case Presentation

A 56-year-old woman with a history of obstructive sleep apnea, persistent atrial fibrillation on anticoagulation, morbid obesity, and heart failure with preserved ejection fraction presented with progressive lower-extremity fatigue first noted while driving in late 2017. By early 2018, she had developed upper-extremity weakness described as a sense of heaviness in the arms during routine activities of daily

living, accompanied by intermittent diplopia. Neurological examination revealed extraocular motility deficits, right-sided ptosis, and proximal shoulder and hip girdle weakness. The ice-pack test was positive. Serologic testing was negative for acetylcholine receptor (AChR) binding, muscle-specific tyrosine kinase (MuSK), and lipoprotein receptor-related protein 4 (LRP4) antibodies. Single-fiber electromyography was borderline abnormal. Repetitive nerve stimulation at 3 Hz showed a reproducible decrement in the right trapezius. The amplitude decrement was 17.4% at baseline and 10.1% to 15.5% after exercise, and the area decrement was 18.1% to 25.7%. The right abductor pollicis brevis showed no significant decrement (≤3.5% amplitude). Using the conventional threshold of ≥10%, these findings indicate impaired neuromuscular transmission with a proximal-predominant pattern, shown below in Table *1*. The combined clinical and electrophysiological findings supported a diagnosis of seronegative MG defined as clinically and electrophysiologically confirmed MG without detectable AChR, MuSK, or LRP4 antibodies. The patient was treated with IVIG, prednisone, and pyridostigmine with symptomatic improvement. Azathioprine was added as a steroid-sparing agent, and chronic IVIG infusions enabled eventual discontinuation of prednisone. In mid-2020, an inadvertent lapse in IVIG following surgery for diverticulitis precipitated a myasthenic exacerbation requiring hospitalization

Region	Trial	Label	Amplitude 1 (mV) onset-to-peak	Amplitude 5 (mV) onset-to-peak	Amplitude % difference	Area 1 (mV·ms)	Area 5 (mV·ms)	Area % difference	Rep rate (Hz)	Train length	Pause time (minutes:seconds)
Right abductor pollicis brevis	1	Baseline	14.18	13.74	-3.1	37.86	34.45	-9	3	10	0:30
	2	Post exercise	14.37	14.32	-0.2	34.65	33.4	-3.6	3	10	1:00
	3	1 min post-exercise	13.62	13.14	-3.5	36.07	33.33	-7.6	3	10	1:00
	4	2 mins post-exercise	13.24	13.14	-0.7	36.7	33.95	-7.5	3	10	1:00
	Voluntary activation (exercise)	3 mins post-exercise							3	10	0:00
Right trapezius	1	Baseline	5.8	4.79	-17.4	54.08	40.16	-25.7	3	10	0:30
	2	Post exercise	5.05	4.53	-10.1	43.39	35.48	-18.2	3	10	1:00
	3	1 min post-exercise	5.41	4.7	-13.2	48.38	38.67	-20.1	3	10	1:00
	4	2 mins post-exercise	5.11	4.57	-10.5	45.67	37.4	-18.1	3	10	1:00
	5	3 mins post-exercise	5.19	4.38	-15.5	47.89	36.5	-23.8	3	10	0:00

TABLE 1: Repetitive nerve stimulation (RNS) study showing greater decremental response in trapezius than abductor pollicis brevis suggesting a proximal predominant pattern of neuromuscular junction transmission failure

Amplitude 1/Amplitude 5: Amplitude of the 1st and 5th compound muscle action potential in each train; Area 1/Area 5: Area under the waveform of the 1st and 5th response; % Difference: Percentage change between the 1st and 5th responses (negative values indicate decrement); Rep rate: Stimulation repetition rate; Train length: Number of stimuli per train; Pause time: Rest interval between trains; Voluntary activation: Exercise performed before the post-exercise trials

In late 2021, the patient developed bilateral upper-extremity numbness, polyarthralgia, and a malar rash. Notably, antinuclear antibody (ANA) had been negative in 2018 but was positive at a titer of 1:320 in 2020, preceding clinical SLE. Rheumatologic evaluation demonstrated positive anti-double-stranded DNA (anti-dsDNA; 24 IU/mL) and anti-ribonucleoprotein (anti-RNP; 3.7) antibodies, with negative anti-Smith and SSA/SSB antibodies. The AChR-binding antibodies were detected for the first time (0.68 nmol/L), representing the first positive serologic marker for her MG. Anti-thyroid peroxidase and anti-thyroglobulin antibodies were also positive with normal thyroid function. A diagnosis of SLE was established; the patient met the 2019 European Alliance of Associations for Rheumatology and the American College of Rheumatology (EULAR/ACR) classification criteria with a total score of 18 (ANA ≥1:80; acute cutaneous

lupus, 6; joint involvement, 6; anti-dsDNA, 6). Hydroxychloroquine (HCQ) and methotrexate were initiated, and the azathioprine dose was increased. In 2022, after maintenance IVIG became unavailable, efgartigimod alfa, an FcRn antagonist, was initiated for MG. The patient's MG Activities of Daily Living (MG-ADL) score decreased from 16 to 5 within weeks of initiation. During an interruption in efgartigimod therapy in 2023, her MG-ADL score rose to 10 and improved again after re-treatment. She also reported substantial improvement in joint symptoms, with no flares documented at subsequent rheumatology visits. Anti-dsDNA titers decreased from 24 IU/mL at SLE diagnosis to 12 IU/mL in 2023 during efgartigimod therapy, with normal complement levels (C3 143 mg/dL; C4 35.4 mg/dL). The trajectory of MG-ADL scores across maintenance IVIG and efgartigimod therapy is shown below in Figure *1*.

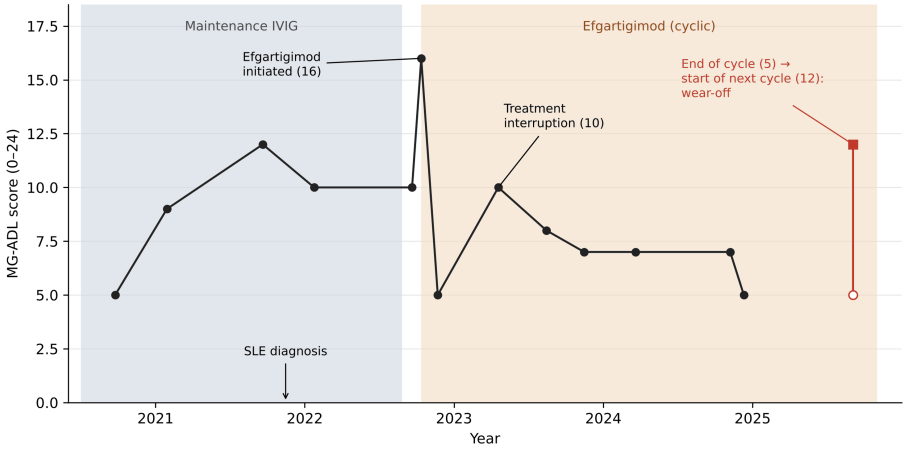


FIGURE 1: The MG-ADL scores during maintenance IVIG and cyclic efgartigimod therapy

The MG-ADL score ranges from 0 to 24; higher scores indicate greater functional impairment.

MG-ADL: Myasthenia Gravis Activities of Daily Living; SLE: Systemic lupus erythematosus

The patient was maintained on efgartigimod, azathioprine, pyridostigmine, and glycopyrrolate for MG, with HCQ and methotrexate continued for SLE. Despite meaningful disease control, she consistently experienced symptomatic deterioration in the final week before each efgartigimod infusion cycle, with MG-ADL scores ranging from 5 at the end of a cycle to 12 at the start of the next. Following multidisciplinary discussion between neurology and rheumatology, the transition to nipocalimab, a fully human anti-FcRn monoclonal antibody administered on a fixed continuous dosing schedule, is currently under consideration.

Discussion

Autoimmune overlap and shared pathogenesis

Systemic lupus erythematosus is a chronic autoimmune disorder that affects multiple organ systems and is linked to considerable morbidity and mortality. A combination of genetic, immunologic, hormonal, and environmental factors contributes to the breakdown of immune tolerance to self-antigens, resulting in the production of pathogenic autoantibodies that mediate tissue injury. Myasthenia gravis is an organ-specific autoimmune disorder of the neuromuscular junction characterized by autoantibodies targeting the nicotinic acetylcholine receptor, muscle-specific tyrosine kinase, or lipoprotein receptor-related protein 4 [4]. These antibodies impair neuromuscular transmission, leading to fluctuating muscle weakness that typically improves with rest.

The prevalence of a second autoimmune disease is higher among patients with a primary diagnosis of autoimmune disease than in the general population. In a study involving 215 patients with SLE, 30% were found to have an additional autoimmune disorder, with Sjogren's syndrome being the most frequently associated condition [5]. The prevalence of SLE among patients with MG, and vice versa, is higher than in the general population. Several mechanisms have been proposed to explain this association, including loss of central immune tolerance following thymectomy, regulatory T-cell dysfunction, altered Fas receptor signaling, and the potential neuromuscular effects of antimalarial medications. Additionally, dysregulation of chemokines such as C-X-C motif chemokine ligand 13 and cytokines like granulocyte-macrophage colony-stimulating factor may play a role in the shared pathogenesis [4].

The coexistence of SLE and MG has been particularly noted among patients who have undergone thymectomy for the latter, with loss of central tolerance proposed as a possible mechanism [6]. Long-term

thymectomized MG patients demonstrate mild T-cell lymphopenia accompanied by hypergammaglobulinemia, B-cell hyperreactivity, and high titers of autoantibodies. However, the role of thymectomy remains uncertain. In a series of 17 patients with both diseases, combined with 53 cases from the literature, MG preceded SLE in 62% of cases, and 27 of 59 patients with available data developed SLE after thymectomy. The authors nevertheless concluded that a shared genetic and immunologic predisposition may better explain the association [6]. A large population-based national study found a significantly increased risk of SLE among MG patients, with an odds ratio exceeding 10 [7]. This association is particularly strong in women, African Americans, and younger patients. Familial aggregation studies support a shared genetic predisposition, showing that first-degree relatives of SLE patients have nearly a threefold higher risk of developing MG [8].

Clinical descriptions of SLE-MG overlap cases reveal characteristic manifestations: SLE often presents with arthritis, cytopenias, and cutaneous involvement, while MG typically manifests as ocular, bulbar, or limb weakness. In one case series of five women with both conditions, SLE preceded MG in four patients, while MG and thymectomy occurred first in one. Systemic lupus erythematosus was generally mild to moderate, with systemic, cutaneous, or articular features; only one patient developed severe cardiac and renal involvement leading to death. Hydroxychloroquine, the cornerstone of SLE therapy, was well tolerated and did not generally worsen myasthenia gravis, though it was discontinued in one patient due to ocular toxicity [9]. In a cohort of 132 MG patients followed between 2000 and 2010, five were also diagnosed with systemic lupus erythematosus. All had generalized MG, and four experienced at least one myasthenic crisis. They showed poor response to corticosteroids, requiring escalation to immunosuppressants, intravenous immunoglobulin, or plasmapheresis. Notably, in contrast to earlier observations, SLE developed before thymectomy in these cases [10].

In the same series of 17 women with SLE-MG overlap, the mean ages at MG onset and SLE diagnosis were 34.5 and 37.8 years, respectively. The most frequent MG symptoms were limb weakness (94%), ocular involvement (88%), and bulbar symptoms (53%), with acetylcholine receptor antibodies positive in 94%. Systemic lupus erythematosus manifestations included arthritis (88%), cytopenias (53%), and skin rash (41%). Hydroxychloroquine was prescribed in 94% of cases, and despite concerns about neuromuscular effects, only one patient developed possible HCQ-induced MG and one experienced a mild exacerbation after starting the drug [6]. Overall, HCQ appeared to be safe and well tolerated in patients with overlapping SLE and MG [6,9]. These findings are summarized below in Table *2*.

Study	Study design/population	Key findings
McDonagh and Isenberg, 2000 [5]	Cohort of 215 patients with SLE	30% had an additional autoimmune disorder; Sjögren's syndrome was the most frequently associated condition
Jallouli et al., 2012 [6]	Series of 17 women with SLE and MG	Mean age 34.5 years at MG onset and 37.8 years at SLE diagnosis. MG: limb weakness 94%, ocular involvement 88%, bulbar symptoms 53%; AChR antibodies positive in 94%; SLE: arthritis 88%, cytopenias 53%, skin rash 41%. HCQ prescribed in 94%; 1 possible HCQ-induced MG and 1 mild exacerbation after starting HCQ; HCQ considered safe and well tolerated
Igoe et al., 2024 [7]	Population-based national study	Significantly increased risk of SLE in MG patients (odds ratio >10); association strongest in women, African Americans, and younger patients
Kuo et al., 2015 [8]	Familial aggregation study	First-degree relatives of SLE patients had nearly threefold higher risk of MG
Jiménez-Alonso et al., 2021 [9]	Case series of five women with SLE and MG	SLE preceded MG in 4; MG and thymectomy occurred first in 1. SLE mild to moderate (systemic, cutaneous, or articular); one patient developed severe cardiac and renal involvement leading to death. HCQ was well tolerated and did not worsen MG; discontinued in one patient for ocular toxicity
Bekircan-Kurt et al., 2014 [10]	Cohort of 132 MG patients (2000-2010); five with SLE	All five had generalized MG; four had ≥1 myasthenic crisis. Poor response to corticosteroids, requiring immunosuppressants, IVIG, or plasmapheresis. SLE developed before thymectomy

TABLE 2: Summary of published studies on SLE-MG overlap

AChR: Acetylcholine receptor; HCQ: Hydroxychloroquine; MG: Myasthenia gravis; SLE: Systemic lupus erythematosus

Treatment options and emerging therapies

Managing patients with coexisting SLE and MG requires an individualized, multidisciplinary approach due to the complexity of addressing both autoimmune diseases simultaneously. Treatment options include acetylcholinesterase inhibitors, corticosteroids, steroid-sparing immunosuppressants, biologics for refractory disease, IVIG or plasmapheresis for myasthenic crisis, HCQ for SLE, and thymectomy in select cases, with emerging therapies reserved for refractory presentations [11-13].

In patients with overlapping SLE and MG, biological therapies are often required for refractory disease or specific organ involvement. Commonly used biologics in SLE include rituximab (anti-CD20) and belimumab (anti-B-cell activating factor), while in MG, eculizumab (anti-C5 complement) and rituximab are used for severe or refractory cases. These agents act via distinct mechanisms: B-cell depletion, complement inhibition, or modulation of autoantibody production. Neonatal Fc receptor antagonists such as efgartigimod and rozanolixizumab are approved for refractory MG and may be considered in SLE-myasthenia gravis overlap [14]. The FcRn is responsible for recycling IgG and prolonging its half-life. Neonatal Fc receptor antagonists block this receptor, leading to accelerated catabolism of IgG and a reduction in circulating pathogenic autoantibodies, which is the primary mechanism for their efficacy in MG. Importantly, FcRn blockade selectively lowers IgG levels without broadly suppressing other Ig classes or cellular immunity, distinguishing these agents from conventional immunosuppressants. Immunoglobulin G autoantibody levels often reflect disease activity in SLE; for example, increased anti-dsDNA titers commonly signal disease flare-ups or the progression of nephritis. Consequently, therapeutic approaches aimed at eliminating or decreasing pathogenic IgG antibodies through high-dose intravenous Ig therapy or FcRn inhibitors are viewed as promising strategies to lessen immune complex-driven tissue injury. The rationale for use of FcRn antagonists in overlap syndromes is to reduce circulating pathogenic IgG autoantibodies, which are central to both SLE and MG pathogenesis [15].

Efgartigimod is a humanized Fc fragment-based IgG derivative that binds the FcRn at both acidic and physiological pH. In a first-in-human study of 62 healthy volunteers, a single dose of efgartigimod reduced circulating IgG levels to approximately 50% of baseline, while repeated dosing further decreased IgG concentrations to about 25%. Immunoglobulin G levels gradually recovered to baseline roughly eight weeks after the final dose. In a phase three trial involving 167 patients with MG, efgartigimod demonstrated superior efficacy compared with placebo, with a significantly greater proportion of treated patients achieving a response on the MG-ADL scale during the first treatment cycle (odds ratio 4.95; 95% CI 2.21-11.53; $p < 0.0001$) [16]. Rozanolixizumab is a humanized Ig G4 monoclonal antibody directed against the IgG-binding domain of the FcRn, thereby preventing FcRn-IgG interactions. In a phase three trial enrolling 200 patients with MG, rozanolixizumab was assessed using multiple clinical endpoints, including the MG-ADL scale, MG composite, quantitative MG, and patient-reported outcome measures. Treatment resulted in clinically meaningful and statistically significant improvements across these measures compared with placebo (ClinicalTrials.gov: NCT03971422) [16]. Nipocalimab is currently being evaluated in an ongoing phase two trial in adults with active SLE. The primary endpoint is the proportion of participants achieving an SLE responder index-4 response at week 24, with secondary endpoints assessing improvements in cutaneous and musculoskeletal disease activity, as well as pharmacokinetic, pharmacodynamic, and immunogenicity outcomes. This study by Yang et al. aimed to establish whether nipocalimab can safely and effectively reduce disease activity across multiple clinical domains in SLE [16].

When FcRn inhibitors are administered in combination with other biologic therapies, particularly IgG-based monoclonal antibodies, there is a theoretical concern regarding reduced efficacy of the co-administered biologic. Neonatal Fc receptor blockade decreases total IgG levels, encompassing both endogenous Ig and therapeutic monoclonal antibodies, which may lead to lower serum concentrations and shortened half-lives of agents such as rituximab, belimumab, or eculizumab. Although this interaction is supported by pharmacokinetic modeling and preclinical studies, clinical evidence in patients with overlapping SLE and MG remains limited. A clinical trial is currently underway to investigate the combined use of efgartigimod and telitacicept, a recombinant fusion protein that inhibits B-cell activating factor and a proliferation-inducing ligand to suppress B-cell maturation in patients with MG (ClinicalTrials.gov: NCT06827587). Importantly, this study uses a sequential treatment strategy, with efgartigimod administered first to rapidly reduce pathogenic circulating antibodies, followed by telitacicept to inhibit ongoing antibody production, thereby supporting sustained immune modulation and longer-term disease control [15,16].

Several observations in this case support a treatment effect of efgartigimod on MG: rapid improvement in MG-ADL after initiation, worsening during a treatment interruption with recovery after re-treatment, and a reproducible wear-off between cycles. The contribution of efgartigimod to SLE activity is less certain. Hydroxychloroquine and methotrexate had been started, and azathioprine was increased after the SLE diagnosis; methotrexate was changed from oral to parenteral administration around the time efgartigimod was initiated, and maintenance IVIG, which may itself modulate SLE activity, was discontinued during the same period. The improvement in joint symptoms therefore cannot be attributed to FcRn inhibition alone. Similarly, the decline in anti-dsDNA titer during efgartigimod therapy is consistent with reduced pathogenic IgG burden, but because FcRn blockade lowers all circulating IgG, this reduction is an expected pharmacodynamic effect rather than independent evidence of reduced SLE activity.

Transition to nipocalimab, with its fixed continuous dosing schedule and emerging evidence in SLE, represents a rational next step and may offer more sustained disease control across both conditions. Whether it also benefits SLE activity remains to be determined, including in the ongoing phase 2 trial in SLE. As FcRn antagonists become more widely used, cases such as this will be important in defining their role in the management of complex, overlapping autoimmune disease.

Conclusions

The coexistence of SLE and MG is uncommon but clinically significant, reflecting shared immune dysregulation driven largely by pathogenic IgG autoantibodies. This case highlights the challenges posed by overlapping autoimmune diseases, as well as the need for coordinated, multidisciplinary management. Emerging targeted therapies, particularly FcRn antagonists, offer a promising approach by directly reducing circulating IgG without broad immunosuppression and may provide benefit in patients with dual IgG-mediated disease. Further clinical studies are needed to better define optimal treatment strategies, long-term outcomes, and the safety of combining FcRn inhibitors with other biologic therapies in SLE-MG overlap syndromes.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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