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CASE REPORT

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Efgartigimod combined with steroids as a fast-acting therapy for myasthenic crisis: a case report

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Abstract

Background Generalized myasthenia gravis (gMG) can be managed with acetylcholinesterase inhibitors (AChEis; e.g., pyridostigmine), corticosteroids, other immunosuppressive drugs (e.g., tacrolimus), and their combinations. Intravenous immunoglobulin (IVIg) or plasmapheresis (PLEX) may be administered if symptoms persist. PLEX and IVIg are also mainstays of treatment for myasthenic crisis. Recently, efgartigimod was approved in Japan for treating adults with gMG (irrespective of the antibody status) who do not have a sufficient response to corticosteroids and nonsteroidal immunosuppressive therapies. Efgartigimod is generally safe and well tolerated. However, since phase III trials of efgartigimod excluded those with myasthenic crisis, the efficacy of efgartigimod in treating myasthenic crisis is still unclear. Moreover, there are no reports that efgartigimod therapy can reduce the dose of corticosteroids needed to achieve a minimal manifestation status.

Case presentation We report the case of a 70-yeat-old woman with gMG who developed a myasthenic crisis. After she was diagnosed with gMG, the patient had been treated with oral corticosteroids and tacrolimus for 1 year. However, she refused to continue taking the medication, and two weeks later, she developed ptosis, dysphagia and dyspnea. The patient was intubated and treated with efgartigimod in combination with steroid therapy, and she recovered without PLEX or IVIg. Afterward, when she experienced worsening of fatigue and increased levels of anti-acetylcholine receptor antibodies, efgartigimod therapy was effective. The patient achieved minimal manifestation status even after the reduction of corticosteroids and showed improvements in the Myasthenia Gravis Activities of Daily Living scales after 4 cycles of efgartigimod infusion.

Conclusions Our case suggests that efgartigimod can be an alternative drug for achieving minimal manifestation status in patients with myasthenic crisis. Considering its strong efficacy and safety, efgartigimod could be expanded to use as bridging therapy in the acute and chronic phases of gMG.

Keywords Efgartigimod, Neonatal Fc receptor inhibitor, FcRn inhibitor, Myasthenia gravis, Myasthenic crisis, Corticosteroid

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Background

Generalized myasthenia gravis (gMG) is an autoimmune disorder of the neuromuscular junction that is characterized mostly by the presence of anti-acetylcholine receptor antibodies (AChR-Abs) and the activation of complement, resulting in damage to the postsynaptic membrane. gMG can involve the upper airway muscles, respiratory muscles, or a combination of both muscle groups, resulting in respiratory failure that requires intubation and mechanical ventilation [1]. The exacerbation of respiration is clinically termed a myasthenic crisis, which is often associated with infections, surgery, adverse effects of medication, comorbidities, pregnancy or the tapering of immunosuppressive medication and can be life-threatening [2]. Despite therapeutic advances, 10–20% of MG patients experience a myasthenic crisis during their disease course [2].

gMG can be managed with acetylcholinesterase inhibitors (AChEis; pyridostigmine), corticosteroids, other immunosuppressive drugs (e.g., tacrolimus), and combinations of these drugs. In recent years, complement inhibitors (eculizumab, ravulizumab) have become available for the treatment of adults with gMG who do not have a sufficient response to these drugs. Intravenous immunoglobulin (IVIg) or plasmapheresis (PLEX) may be performed if symptoms persist [3]. PLEX and IVIg are also the mainstays of myasthenic crisis treatment [4]. Efgartigimod, a neonatal Fc receptor (FcRn) inhibitor, is a novel drug designed to promote catabolism of IgG, leading to reduced IgG levels [5]. In December 2021, efgartigimod was approved by the United States Food and Drug Administration for the treatment of adults with AChR-Ab-positive gMG. Efgartigimod was also approved in Japan in January 2022 for the treatment of adults with gMG who do not have a sufficient response to corticosteroids and nonsteroidal immunosuppressive therapies, irrespective of their antibody status [5]. In phase II clinical trials, efgartigimod has been found to be safe and well tolerated, and treated patients show improved scores on the Quantitative Myasthenia Gravis (QMG), Myasthenia Gravis Activities of Daily Living (MG-ADL), and Myasthenia Gravis Quality of Life 15-item revised (MG-QOL15r) scales [6]. The Japanese clinical guidelines 2022 for Myasthenia Gravis and Lambert–Eaton myasthenic syndrome state that efgartigimod is a promising biological drug for patients with moderate to severe gMG [7].

To date, the efficacy of efgartigimod in treating myasthenic crisis is still unclear. In a phase III clinical trial of efgartigimod, severe patients in Class V (defined as intubation) according to the Myasthenia Gravis Foundation of America clinical classification of MG were excluded [7]. Moreover, there is no information on whether efgartigimod can reduce the dose of corticosteroids while achieving minimal manifestation (MM) status or minimal symptom expression.

Here, we report the case of an elderly patient with gMG who was successfully treated with efgartigimod in combination with corticosteroids through the acute phase of myasthenic crisis to the maintenance period and maintained MM status.

Case presentation

A 70-year-old Japanese woman who presented with gradually developing diplopia over approximately 1 month was evaluated. She had been in her normal healthy state without any significant medical history until this presentation. Her family history was unremarkable. On arrival, the patient complained of diplopia in all directions, photophobia, and blurred vision (Day 1). She subsequently experienced generalized fatigue, including respiratory symptoms, and subsequently diagnosed with gMG. During the disease course, she experienced a myasthenic crisis, and efgartigimod treatment was effective, including during the acute phase of respiratory failure. Thus, we thoroughly evaluated the associations among clinical symptoms, laboratory findings, and the effects of efgartigimod during the acute and maintenance phases.

This study was conducted according to the principles of the Declaration of Helsinki and this case report was written following the CARE guidelines. The Institutional Ethics Review Board of the Minaminara General Medical Center waived approval of the study design. Written informed consent was obtained from the patient and her daughter for publication of this case report.

The whole clinical course from disease onset to the latest follow-up visit is shown in Fig. 1. Neurological examination on Day 1 revealed impairment of extraocular movements and ptosis of the left eyelid. Laboratory tests revealed elevated blood levels of AChR-Ab (42 nmol/L, normal range: less than 0.2 nmol/L). Anti-thyroglobulin antibodies (32 IU/mL, normal range: less than 28 IU/mL) and anti-thyroid peroxidase antibodies (more than 600 IU/mL, normal range: less than 16 IU/mL) were also elevated. Repetitive stimulation studies revealed waning of the compound muscle action potentials by stimulation of the left median nerve (decrement by 19.0–20.5%, normal range: less than 10%) and of the left accessory nerve (decrement: 15.3%). Chest CT revealed a calcified cyst in the superior mediastinum without enhancement, suggesting a benign tumor, but a thymoma was not detected. The patient subsequently presented with a fluctuating course of neck pain, camptocormia, diplopia and fatigue, and she was diagnosed with gMG. Combination therapy using AChEi, oral prednisolone (1.0–10.0 mg/day), and tacrolimus (1.0–2.0 mg/day) was effective for maintaining her activities of daily living and to suppress the

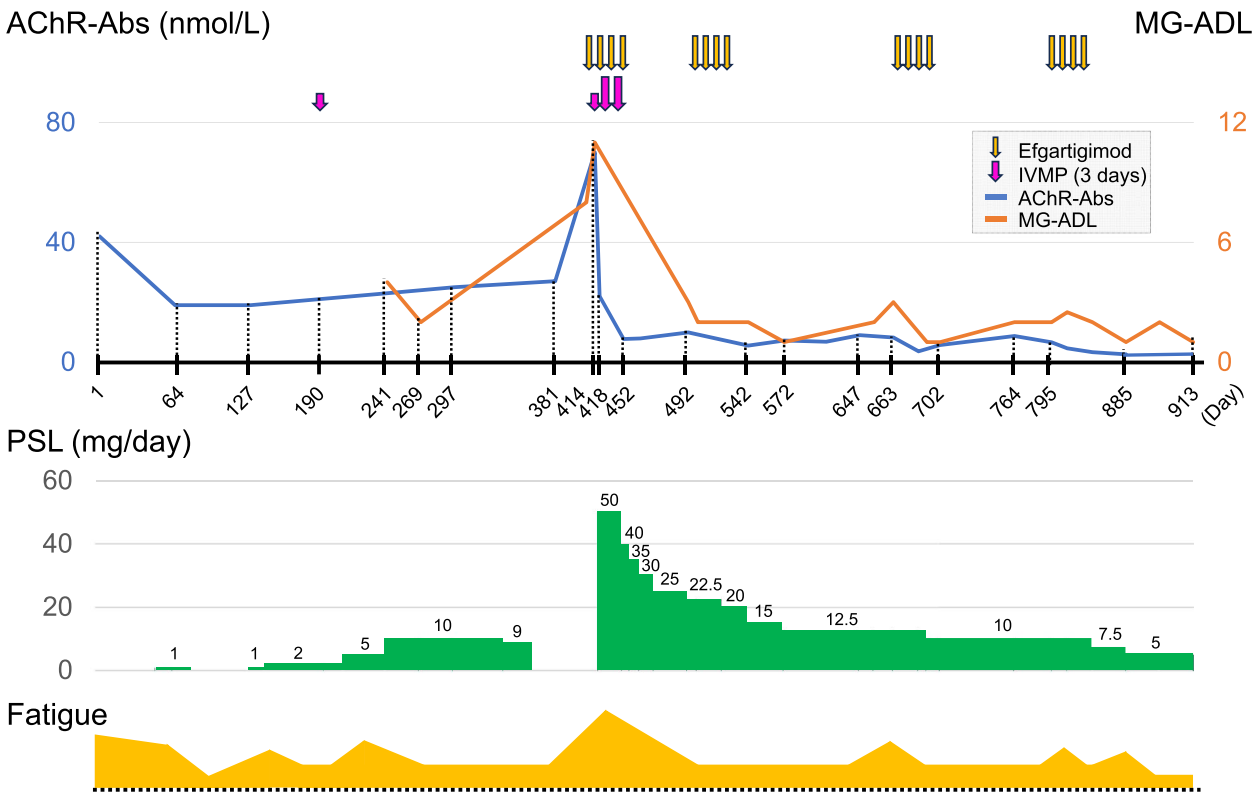

Fig. 1 Clinical course from onset to infusion of efgartigimod. The patient developed a myasthenic crisis due to refusal to take medication and was treated with efgartigimod from Day 414 (yellow arrows). In total, four cycles of efgartigimod were administered. AChR-Abs: Anti-acetylcholine receptor antibodies; MG-ADL: Myasthenia Gravis Activities of Daily Living; IVMP: Intravenous methylprednisolone pulse therapy; PSL: prednisolone

AChR-Ab titer. An additional cycle of half-dose intravenous methylprednisolone pulse therapy (IVMP, 500 mg/day for 3 consecutive days, Days 190–192) was also effective when her symptoms worsened.

However, the patient suddenly experienced deterioration of fatigue, severe camptocormia, and ptosis on Day 381 because she refused to continue taking the medications. These symptoms rapidly progressed to dysphasia and dyspnea, requiring intubation despite increased doses of AChEis and oral corticosteroids. Figure 2 shows the specific clinical course of the myasthenic crisis. We considered standard treatment for myasthenic crisis; however, IVIg was unavailable because of a supply shortage, and we could not secure specialists to perform emergent blood access for PLEX. Thus, after providing informed consent, the patient was treated with a cycle of efgartigimod (500 mg/day, weekly for 4 weeks, Days 414, 421, 428, and 436) in combination with repeated IVMP (500 mg/day for 3 consecutive days, Days 415–417, 1000 mg/day for 3 consecutive days, 422–424 and 430–432), followed by oral corticosteroids, which were gradually tapered. The patient also suffered from complicating aspiration pneumonia and was treated with ampicillin/sulbactam twice. She also had insomnia due to sputum retention and needed sedatives. In addition, steroid-induced hyperglycemia occurred, for which she was treated with insulin. On Day 429, the patient's arterial blood gas improved, and she was extubated. Afterward, she recovered and was able to walk without feeling any dyspnea or fatigue. Her AChR-Ab titer, which was 70 nmol/L on Day 414, decreased to 7.7 nmol/L on Day 439. Her IgG level also decreased from 1067 mg/dL on Day 414 to 358 mg/dL on Day 452 (Figs. 2 and 3). During intubation, the patient was confused and unaware of her situation, but after decannulation, her cognitive dysfunction became more apparent. She could not remember or write her name, tell her age, or read simple Kanji (Chinese characters). The patient scored 21/30 on the Mini-Mental State Examination-Japanese (authorized translation, purchased from Nihon Bunka Kagakusha Co., Ltd., Tokyo, with a license to use) on Day 443 and 59/100 on the Addenbrooke's Cognitive Examination-III on Day 449. She also complained of mild headache. The findings from magnetic resonance imaging of her brain on Day 437 were unremarkable. Cerebrospinal fluid examination on Day 452 revealed no pleocytosis or oligoclonal band, and

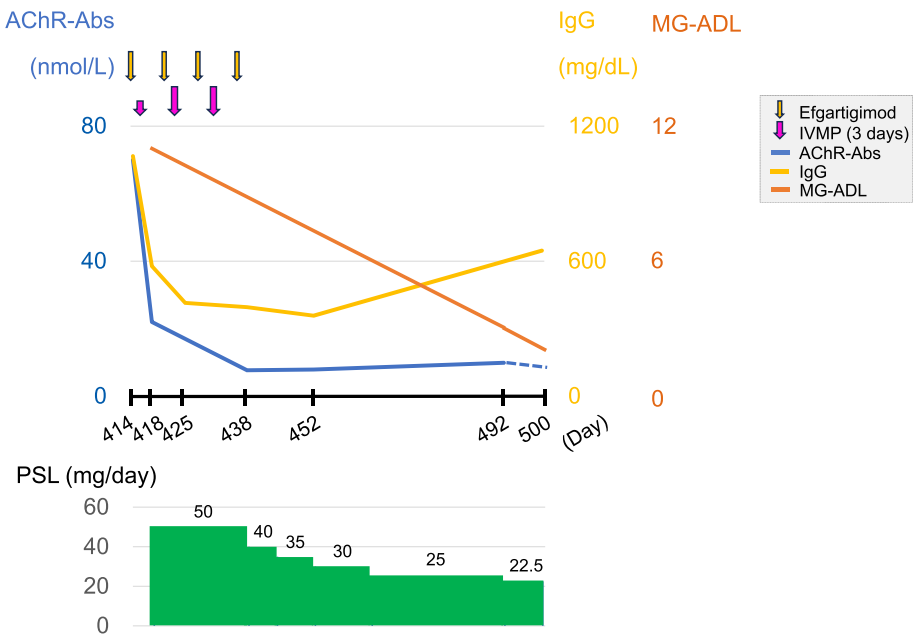

Fig. 2 Precise clinical course of the myasthenic crisis. The patient's dyspnea and fatigue worsened despite intravenous steroid therapy (500 mg/day for 3 days); she was intubated and treated with a high dose of oral prednisolone (50 mg/day), intravenous efgartigimod (500 mg/day, weekly, 4 times), and intravenous methylprednisolone pulse therapy (1000 mg/day for 3 days, 2 cycles). The patient was extubated on the 15th day. AChR-Abs: Anti-acetylcholine receptor antibodies; MG-ADL: Myasthenia Gravis Activities of Daily Living; IVMP: Intravenous methylprednisolone pulse therapy; PSL: prednisolone

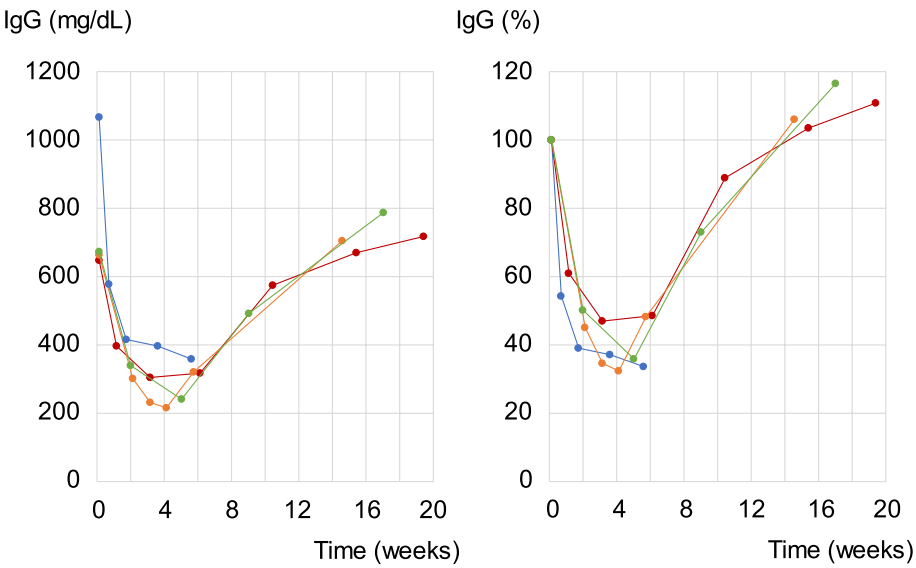

Fig. 3 Changes in the IgG level and its ratio in the first (blue), second (red), third (orange), and fourth (green) cycles of efgartigimod. In all cycles, more than 50% IgG reduction was observed, which lasted for 6 weeks

the IgG index was 0.53 (normal upper limit: 0.73). These symptoms spontaneously improved over 2 weeks.

The patient was able to manage her daily life on her own but then started to experience shortness of breath and fatigue on Day 492. As her AChR-Ab concentration had increased to 9.9 nmol/L, the patient underwent a second cycle of efgartigimod (500 mg/day, Days 500, 507, 521, and 528). On Day 528, her symptoms improved, and her AChR-Ab titer was 5.5 nmol/L. The dose of oral corticosteroid was successfully tapered. When the

patient showed worsening of fatigue and slight elevation of the AChR-Ab level, efgartigimod therapy was effective (500 mg/day; the third cycle: Days 663, 670, 677, and 684; the fourth cycle: Days 795, 802, 809, and 816). During every cycle of efgartigimod, the patient's IgG level decreased by more than 50%, which lasted for 6 weeks (Fig. 3). Since then, the patient has remained in the MM state as of the most recent follow-up visit on Day 913.

Discussion and Conclusions

To the best of our knowledge, this is the first case of gMG to show the efficacy of efgartigimod during the acute phase of a myasthenic crisis without using IVIg or PLEX. Recent case studies demonstrate the efficacy of efgartigimod as an add-on therapy for myasthenic crisis in combination with IVIg [8], PLEX [9, 10], or both [11]. A multicenter cohort study of efgartigimod for gMG in China showed clinically meaningful improvement in 4 out of 9 (44.4%) of patients with myasthenic crisis, but information on combination therapies was not described [12]. The current data suggest a promising, possibly independent, role of efgartigimod in the treatment of MG exacerbation and myasthenic crisis as a new option in addition to therapy with IVIg or PLEX [4]. In addition, efgartigimod was safe and well tolerated in both phase II and phase III clinical trials [6, 13]. In our case, the patient complained of only a mild headache.

In our patient, the IgG level decreased by more than 50%, which lasted for 6 weeks in all cycles of efgartigimod (Fig. 3). Compared with PLEX, efgartigimod can remove lgG more selectively while sparing albumin with a lower patient burden and can be more easily administered in general hospitals without intensive care units [14, 15]. Compared with IVIg, efgartigimod poses a lower risk of thrombosis and hemolysis [14]. Tran MH reported that 4 doses of efgartigimod could reduce IgG levels by 61–85% and that this reduction was comparable to a median of 6 plasma exchanges [16]. Moreover, efgartigimod reduces anti-AChR autoantibodies by 40–70%, which is equivalent to the effect of IVIg (29%), plasma exchange (63%), or immunoadsorption (55%) [17], and results in favorable clinical improvement and a short hospital stay [14]. Notably, the reduction in IgG and anti-AChR antibodies paralleled the improvement in symptoms [14]. Considering its ability to reduce anti-AChR autoantibodies comparable to IVIg or PLEX, efgartigimod may be effective in treating myasthenic crisis. In our patient, the myasthenic crisis was treated with corticosteroids and efgartigimod without IVIg or PLEX, but it took three weeks to withdraw the ventilator —a result that was not inferior to that of conventional therapies [18]. After the use of efgartigimod, the MG-ADL score improved by 9 points, which was consistent with the"good responder" score reported in the ADAPT III trial [6, 13]. Although an intravenous methylprednisolone pulse had no demonstrable effect on the total (polyclonal) anti-AChR antibody titer, the onset of improvement after intravenous infusion of methylprednisolone for MG exacerbation was rapid (3.0 ± 1.1 days) [19]. Add-on therapy of efgartigimod to methylprednisolone may be effective because efgartigimod maintains its effect for several weeks [13].

Our patient was treated with three cycles of efgartigimod in the outpatient clinic, achieved the MM state without the use of tacrolimus, and successfully maintained this state despite tapering the corticosteroid dose. Thus far, tacrolimus is known to be effective at reducing the corticosteroid dose [20], but IVIg is not effective [21]. The ADAPT III trial revealed that while the terminal half-life of efgartigimod is relatively short (4.89 days), its clinical effects last long throughout the follow-up period, i.e., 8 weeks after the last efgartigimod administration [13]. The levels of antibodies returned to within 20% of baseline within 3 weeks after PLEX; however, this value was reached 9 weeks after efgartigimod [13]. The clinical benefit of efgartigimod was initially correlated with the IgG reduction but persisted even after the IgG level returned close to baseline. This long-lasting effect of efgartigimod possibly resulted in a reduction in the dose of corticosteroids.

No publications have been published on the use of other biological drugs for the treatment of myasthenic crisis. Eculizumab was reported to be effective in patients with a history of myasthenic crisis [22].

Our patient exhibited transient cognitive decline after efgartigimod treatment, but she also suffered from insomnia and steroid-induced hyperglycemia and needed symptomatic treatments. Therefore, the combination of these factors could have caused her reversible cognitive dysfunction. On the basis of the results of phase II and III clinical trials, which revealed no adverse events related to cognition, it is reasonable to consider that the patient's cognitive symptoms were unrelated to efgartigimod treatment.

In conclusion, the results of the present study suggest that efgartigimod may be an alternative drug for achieving MM status in a myasthenic crisis. The Japanese Myasthenia Gravis and Lambert–Eaton myasthenic syndrome guidelines of 2022 recommend that efgartigimod should be used in patients with refractory MG [7]. The ADAPT phase III trial revealed that 78% of patients sustained more than a 2-point improvement on the MG-ADL scale for ≥ 4 weeks after 2 cycles [13]. However, data on efgartigimod use not only in patients with myasthenic crisis but also in patients with

refractory MG are still limited. Considering its strong efficacy and safety, efgartigimod could be expanded as bridging therapy in the acute and chronic phases of MG.

Abbreviations

gMG	Generalized myasthenia gravis
AChR-Abs	Anti-acetylcholine receptor antibodies
AChEis	Acetylcholinesterase inhibitors
IVIg	Intravenous immunoglobulin
PLEX	Plasmapheresis
FcRn	Neonatal Fc receptor
QMG	Quantitative Myasthenia Gravis
MG-ADL	Myasthenia Gravis Activities of Daily Living
MG-QOL15r	Myasthenia Gravis Quality of Life 15-item revised
MM	Minimal manifestation
IVMP	Intravenous methylprednisolone pulse therapy

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Authors' contributions
HO: conceptualization, investigation, resources, data curation, and writing – original draft. NK: conceptualization, investigation, resources, and data curation. NI: conceptualization, investigation, resources, and data curation. MK: conceptualization, writing – review and editing, visualization, supervision, and funding acquisition. All the authors contributed to the article and approved the submitted version.

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Availability of data and materials
The data that support the findings of this study are available on request from the corresponding author [MK]. The data are not publicly available due to information that could compromise research participant privacy.

Declarations

Ethics approval and consent to participate
The requirement for ethical approval was waived by the Ethics Committee of Minaminara General Medical Center. This study was conducted in accordance with local legislation and institutional requirements.

Consent for publication
Written informed consent was obtained from the patient and her daughter for the publication of any potentially identifiable images or data included in this article.

Competing interests
HO received contracts and honoraria from Otsuka Co., Ltd., Alexion Co., Ltd., UCB Japan Co., Ltd., Daiichi Sankyo Co., Ltd., Kyowa Kirin Co., Ltd., Tanabe Mitsubishi Co. Ltd., Sumitomo Pharma Co., Ltd, FP Pharma Co. Ltd, and Argenix Co. Ltd. MK received contracts and honoraria from Eisai Co., Ltd., Daiichi Sankyo Co., Ltd., and UCB Japan Co., Ltd. MK is an Editorial Board Member for BMC Neurology. The remaining authors declare that this study was conducted in the absence of any commercial or financial relationships that could be construed as potential conflicts of interest.

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