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Successful corticosteroid-free management of anti-HMG–CoA reductase immune-mediated necrotizing myopathy with subcutaneous immunoglobulins monotherapy: a case report

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Abstract

Background Given the high rate of cardiovascular comorbidities in anti-HMG–CoA reductase (HMGCR)–immune-mediated necrotizing myopathy (IMNM), a glucocorticosteroids (GC)-free treatment approach remains an appealing strategy. While intravenous immunoglobulins (IVIg) represents an effective therapy in most subgroups of idiopathic inflammatory myositis (IIM), there remains limited evidence supporting the use subcutaneous immunoglobulins (SCIg), a potentially safer, more convenient and cost-effective alternative.

Case presentation We present a case of a 68-year-old male of European descent with a 2-year history of progressive proximal bilateral lower extremity weakness. He had been treated for his dyslipidemia with atorvastatin for 2 years, then with rosuvastatin for 2 additional years until discontinued a year before our assessment. His other comorbidities included hypertension and diabetes mellitus type II. Physical examination demonstrated proximal weakness of his bilateral hip flexors and deltoids, and no rash. His creatine kinase (CK) was elevated at 1644 IU/L. Electromyography revealed a generalized myopathic disorder with proximal predominance and muscle biopsy of the right quadriceps showed typical findings of an IMNM. Serum HMG–CoA reductase antibody was positive. Due to accumulating evidence supporting the effectiveness of IVIg in anti-HMGCR–IMNM, even without concomitant GC or other immunosuppressive agent, we opted for a GC-free approach through shared-decision making in light of patient's preference regarding potential GC side effects with his comorbidities. As he lived a considerable distance from any center, where IVIg infusions could be offered, and given the patient was unable to drive himself due to his symptoms we opted for SCIg (0.5 g/kg/week). Within 1 month, his CK decreased and weakness resolved. SCIg monotherapy was tapered over 3 years and this patient remains in remission 7 years after SCIg initiation. The patient did not have any adverse effects related to SCIg use.

Conclusions To our knowledge, this is the first case report describing a successful corticosteroid-free management of anti-HMGCR immune-mediated necrotizing myopathy using SCIg as monotherapy. This case highlights the potential for steroids-free induction and maintenance strategies in IMNM. Larger studies are required to confirm

†Nancy Maltez and Catherine Ivory shared the co-senior author for this publication and contributed equally to this work.

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the effectiveness and safety of SCIg in IMNM and other subtypes of IIM, and to provide guidelines for dosing recommendations.

Keywords Idiopathic inflammatory myositis, HMG–CoA reductase, Immune-mediated necrotizing myopathy intravenous immunoglobulins, Subcutaneous immunoglobulins

Background

Idiopathic inflammatory myopathies (IIM) represent a rare subset of autoimmune disease characterized by symmetrical proximal muscle weakness, elevation of creatine kinase (CK) and may present with extra-muscular manifestations, such as dysphagia, arthritis, interstitial lung disease and cardiac disease, amongst others [1]. Combination therapy with high-dose intravenous immunoglobulins (IVIg) and glucocorticosteroids (GC) remains one of the only treatment modalities shown in controlled trials to be effective for the management of most subgroups of IIM [2, 3]. Although mechanisms of action are not fully understood, IVIg is thought to have immunomodulatory effects on various components of the immune system, including complement, cellular immunity, and humoral immunity [4–6].

There remain several prohibitive factors for the use of IVIg as first-line therapy, such as high cost and current supply shortage [7, 8]. In addition, potential adverse events associated with the use of IVIg include infusion-related symptoms, such as flushing, headache, malaise, fever, chills, fatigue and lethargy; however, more serious side effects have been reported, such as renal impairment, thrombosis, arrhythmia, anaphylactic reactions, aseptic meningitis, hemolytic anemia, and transfusion-related acute lung injury [9, 10]. Administration of IVIg for IIM requires two to five hospital visits per month (for up to many years) with monitoring by qualified health care staff given the potential complications described above.

The use of subcutaneous immunoglobulins (SCIg) as an alternative to IVIg has been studied in a variety of conditions, such as immunodeficiency diseases, multifocal motor neuropathy, chronic inflammatory demyelinating polyneuropathy and myasthenia gravis, with lower rates of adverse events when compared to IVIg despite small sample sizes [11, 12]. There remains paucity of data regarding the use of SCIg in immune-mediated necrotizing myopathy (IMNM), such as anti-HMG–CoA reductase (HMGCR)–IMNM. One previous case report reported successful treatment of this entity with SCIg after the patient failed multiple lines of treatment, such as mycophenolate, cyclophosphamide, and rituximab, and could not tolerate IVIg [13]. Zuppa et al. have also described three cases of anti-HMGCR–IMNM treated with SCIg with a favorable clinical course, albeit after having been initiated on IVIg initially for remission of disease [14].

We hereby describe a case of successful corticosteroid-free management of anti-HMGCR–IMNM with SCIg monotherapy for induction and maintenance and summarize the current evidence that supports the use of SCIg in anti-HMGCR–IMNM. To the best of our knowledge, no similar case has previously been described in the literature. This case highlights the potential for GC-free induction and maintenance strategies in anti-HMGCR–IMNM using SCIg, an alternative which offers the potential advantages of home administration, improving convenience and adherence.

Case presentation

We present the case of a 68-year-old male of European descent who was referred to our Rheumatology clinic in April 2018 with a 2-year history of progressive proximal bilateral lower extremity weakness. He had a past medical history of hypertension, dyslipidemia and diabetes mellitus type II and was prescribed amlodipine 5 mg daily, perindopril–indapamide 8–2.5 mg daily and aspirin 81 mg po daily when he was seen in our clinic. The patient had been on atorvastatin 20 mg since Sept 2012, which was increased to 40 mg in February 2014, then change to rosuvastatin 10 mg in September 2014 and discontinued only in January 2017 when the patient's proximal lower extremity muscle weakness came to medical attention. Despite the discontinuation of his statins, his symptoms persisted.

He was referred to the rheumatology service and first seen in April 2018 for a suspected autoimmune myopathy. His weakness had progressed, and he had difficulty getting up from a seated position. He denied upper extremity symptoms, visual changes or diplopia, dysphagia, respiratory symptoms, new dermatological findings, Raynaud's phenomenon and constitutional symptoms. Physical examination revealed no rash, proximal weakness with a Medical Research Council Manual Muscle Testing (MRC) score [15] of 4-/5 of his bilateral hip flexors and deltoids.

Laboratory investigations revealed an elevated CK (1644 IU/L, 3304 IU/L in 2016; normal range 52–256 IU/L). Anti-nuclear antibodies were positive at 1:160 titers with negative extractable nuclear antigens panel, myositis panel and double-stranded DNA.

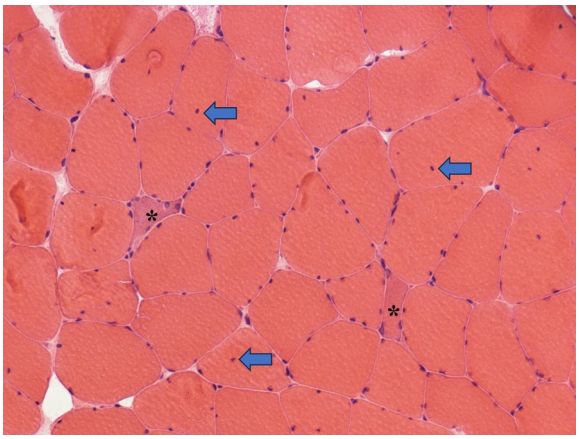

Fig. 1 Muscle biopsy, right vastus lateralis muscle. Hematoxylin and eosin stain (magnification power × 100). *: Regenerating muscle fibers; ←: Internal nuclei

Needle electromyography (EMG) showed generalized irritative myopathic units with proximal predominance. Nerve conduction studies were normal. Muscle biopsy of the right vastus lateralis showed myopathic features with regenerating fibers, rare necrotic fibers and increased internal nuclei (Fig. 1). Genetic panel for muscular dystrophy was requested and came back negative. In our institution at this time, anti-HMGCR testing was referred to a reference laboratory for analysis (Mitogen Advanced Diagnostics, Calgary, AB, Canada, performed using an addressable laser bead immunoassay) and was reported as negative, low, moderate or high positive. Serum HMGCR antibody for this patient was eventually reported as high positive. Cancer screening, including computed tomography of chest, abdomen and pelvis, was negative. Other causes of myopathy were excluded, including endocrine and metabolic disorders, as evidenced by normal thyroid-stimulating hormone, parathyroid hormone, serum calcium, and 25-hydroxyvitamin D levels.

The patient was classified as having an anti-HMGCR–IMNM, supported by 224th ENMC International Workshop criteria, namely, a positive HMGCR antibody result, elevated CK, proximal muscle weakness and further supported by typical pathological changes of anti-HMGCR–IMNM including paucilymphocytic infiltrates and the presence of necrotic fibers with scattered distribution [16]. Magnetic resonance imaging was not obtained during the initial evaluation, as it was deemed unlikely to provide additional diagnostic value in the context of an already established diagnosis. Statin therapy had already been discontinued, and the patient was switched to evolocumab.

Given the increasing evidence that corticosteroids-free induction and maintenance therapy could be successful anti-HMGCR–IMNM patients, we opted for a GC-free approach through shared-decision making in light of patient's preference regarding potential GC side effects. The choice of IVIg as induction therapy over other GC-sparing alternatives (e.g., methotrexate, azathioprine) was based on accumulating evidence of its effectiveness in anti-HMGCR–IMNM, even without concomitant GC or other immunosuppressive agent [17].

The patient lived in a rural community which was at a considerable distance from any center, where IVIg infusions could be offered. Given his weakness, the patient was unable to safely drive himself and had no other alternative transportation available to him. The patient was referred to the immunoglobulin treatment program at our hospital, where expertise in teaching and managing SCIg in immunodeficient patients has been established. Self-administered 20% SCIg product (Cuvitru^{R} manufactured by Takeda) using a pump-assisted method was initiated at 0.5 g/kg/week or 56 g based on a weight of 113.8 kg. The dose was divided (20 g, 20 g, and 16 g) to be administered three times a week. Dose setting of SCIg was extrapolated from previous studies [18], as there exists no current recommendations for SCIg dosage in the use of IIM, its recommended duration and tapering.

Within a month, the patient's CK decreased to 520 U/L (Fig. 2) and the proximal weakness improved with MRC score 5/5 in shoulder abductors, hip flexors, and other muscle groups. His CK normalized 5 months after SCIg initiation and has remained normal ever since. SCIg was well-tolerated without any reported adverse effects. SCIg monotherapy continued at 56 g/week for 12 months, and was tapered to 40 g/week until February 2020. The dose was then reduced further to 20 g/week, and the

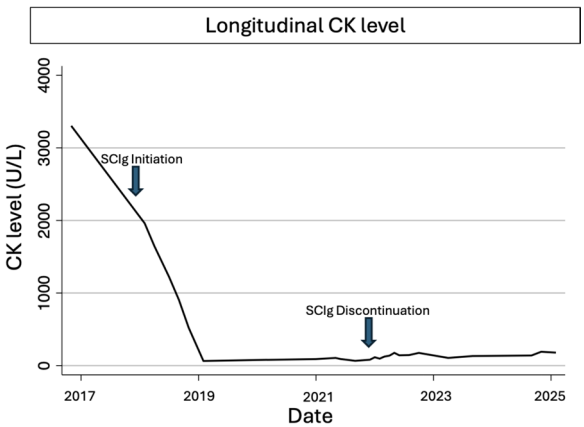

Fig. 2 Longitudinal CK level. *CK* creatine kinase, *SCIg* subcutaneous immunoglobulins

treatment was discontinued in September 2021. The patient had repeat anti-HMGCR antibodies in May 2025 due to reported leg fatigue, this time detected by chemiluminescent immunoassay (Werfen Bioflash, San Diago, USA), which resulted at 121 CU (equivalent to moderate positive). His strength remained unchanged, his CK normal and a repeat EMG was unremarkable. At his last follow-up, the patient remained in remission while off any immunosuppressive therapy.

Discussion

We report the first described case of anti-HMGCR–IMNM successfully managed with corticosteroid-free induction and maintenance using SCIg monotherapy. While current recommendations for the treatment of IIM include high-dose glucocorticoids in combination with disease modifying anti-rheumatic drugs, our case highlights a feasible GC-sparing strategy with the added advantages of home administration, convenience, and improved adherence [19].

Statin-induced IMNM remains a rare complication of HMG–CoA inhibitors and is characterized by the presence of HMGCR antibodies, with subsequent cascade of autoimmune responses resulting in necrotizing myopathy [20]. Though the precise pathophysiology of statin-induced IMNM remains poorly understood, it is hypothesized that statins trigger an overexpression of HMGCR in genetically susceptible patients, which in turn is thought to cause the development of autoimmunity against HMGCR [21].

Beyond discontinuation of statins, current treatment recommendations for the treatment of anti-HMGCR–IMNM from the 224th European Neuromuscular Centre International Workshop include the use of corticosteroids in conjunction with a steroid-sparing immunosuppressant (SSI), such as methotrexate, IVIg or eventually rituximab [16]. A recent retrospective multi-site cohort study of anti-HMGCR–IMNM discovered a high rate of diabetes mellitus at 52.8% in this patient population [22]. Considering that anti-HMGCR–IMNM typically affects patients already at increased risk of cardiovascular comorbidity, a GC-free treatment approach remains an appealing strategy.

The evidence supporting the effectiveness of IVIg as monotherapy or in conjunction with other immunosuppressive agents is accumulating. Mammen et al. first described three anti-HMGCR–IMNM patients with diabetes who had declined GCs because of concerns about potential side effects. These patients were subsequently successfully treated with IVIg monotherapy (2 g/kg per month), with normalization of strength examination in two patients [17]. In another study, the clinical course of a total of 16 consecutive patients anti-HMGCR–IMNM was analyzed retrospectively. GCs were usually employed in induction therapy, but most patients (62.5%) remained on GCs only for a short time (i.e., the first 3 months). IVIg and MTX were used very frequently also to allow a quicker GC tapering [23]. A retrospective study by Meyer et al. described a successful GC-free induction strategy in 14 selected patients, with initial induction strategies being steroid-sparing immunosuppressant (SSI) monotherapy ($n=7$) or dual IVIg/SSI monotherapy ($n=7$). In the same cohort, of the anti-HMGCR–IMNM cohort treated with GC at induction, 73% of patients had a successful steroid-free maintenance. Finally, a retrospective longitudinal cohort study by Suh et al. described 15 patients that received IVIg without any other SSI, with none of them that had any contraindications to GCs or other immunosuppressants and the most frequently documented reason for IVIg being physician preference [24]. With IVIg monotherapy, CK levels declined significantly from baseline to follow-up at 3 months and 6 months with a median percent decrease in CK from baseline of 84.5% (79.5–91.1%) at 3 months and 95.7% (76.6–98.3%) at 6 months [24]. These patients also demonstrated a statistically significant improvement in their Kendall score (a grading system that scores clinical muscle strength) at 3 and 6 months, and 76.9% of these patients showing normalization of their strength at 6 months [24] However, 5 patients eventually required additional immunosuppressive therapy for disease control [24], highlighting the need for careful selection of patients when deciding for a corticosteroids-free induction therapy, and the need for close monitoring even with a successful initial response.

Given the increasing evidence that corticosteroids-free induction and maintenance therapy could be successful anti-HMGCR–IMNM patients, we opted for a GC-free approach through shared-decision making in light of patient's preference regarding potential GC side effects. Immunoglobulin therapy was favored due to its recognized efficacy in anti-HMGCR–IMNM, and the subcutaneous route was favored due to the patient's inability to travel to an infusion center. Although he was symptomatic in 2016 with an elevated CK, he was only referred to our service in 2018. This long delay highlights the common diagnostic delays for this rare condition, with an overall mean diagnostic delay for IIM reported at 27.91 months in a systematic review [25]. Improvement of the patient's CK (without normalization) may be explained by chronic damage of viable muscle fibers resulting in fewer sources for CK leakage, resulting in lower serum levels even as muscle function deteriorates [26]. It is important to note that our patient had relatively mild symptoms, and that this therapeutic approach may not be generalizable to more severe anti-HMGCR–IMNM patients, or other IIM subtypes.

SCIg has demonstrated similar effectiveness in several case series in IIM patients [18, 27, 28]. A review of the current evidence of SCIg in IIM has previously been described by our group [29]. However, previous experiences with the use of SCIg in IMNM have yielded conflicting results, as a previous case series demonstrated worsening in median CK and worsening of symptoms [30], while another case report in a patient who had previous failed mycophenolate mofetil, IVIg, Rituximab and cyclophosphamide achieved clinical remission when started on SCIg [13]. There is increasing evidence that early recognition of the disease and early treatment of anti-HMGCR myopathy may increase the efficacy of SSI monotherapy in maintaining remission and allow a successful corticosteroid-free induction strategy [31].

Side effects, accessibility and associated high cost remain prohibiting factors for the wider use of IVIg in IIM. Although the cost remains elevated, SCIg can be administered in smaller doses more frequently, and has been purported to be safer, because it results in sustained, steady serum immunoglobulin levels, reducing the risk of volume overload [18]. Use of SCIg may lead to better health-related quality of life and treatment satisfaction as seen in other patient populations such as primary immunodeficiencies when compared to IVIg treatment in hospital or clinics in addition to significant cost savings [32, 33]. SCIg, in turn, eliminates the need for intravenous access and monitoring, allowing patients to receive treatment at home. A 2023 patient survey by Ma et al. of patients with IIM who were transitioned from IVIg to SCIg therapy for > 12 months did demonstrate that the vast majority of respondents (78.9%) preferred SCIg over IVIg, with an overall high satisfaction with SCIg therapy [34].

Dosing, monitoring parameters and tapering recommendations for the use of SCIg in anti-HMGCR–IMNM, and more broadly in IIM, have not been established. There remains significant heterogeneity in dosage and duration of treatment as evidenced in various studies, and dosage used for this patient was similar to best evidence [4, 18, 35]. The duration and tapering of SCIg in this case were determined by the patient's primary physician due to the absence of established guidance. Its prolonged use was largely driven by its role as monotherapy, with gradual dose reductions implemented, while CK levels and muscle strength remained stable. Serum HMGCR antibody titers may decline over time as strength and CK improves, although most HMGCR antibody titers may not normalize [36]. Though his repeat serum HMGCR antibody titers had fallen when repeated in May 2025 due to reported leg fatigue, serum HMGCR antibody titers were not used to guide tapering decisions in this case. It remains uncertain whether SCIg could have been tapered more rapidly. More than 4 years after his SCIg was discontinued, the patient did not have any clinical or subclinical relapses.

Conclusion

To the best of our knowledge, we report the first case of anti-HMGCR–IMNM successfully treated with SCIg as monotherapy for induction and maintenance. GC-free management of anti-HMGCR–IMNM remains appealing given the high rate of cardiovascular comorbidities in this patient population. Further studies in anti-HMGCR–IMNM, anti-signal recognition particle IMNM and other subtypes of IIM, are required in order to confirm the efficacy of SCIg, which could represent a safe and convenient alternative to IVIg. There remain important gaps in terms of guidelines for dosing recommendation, monitoring parameters and tapering.

Abbreviations

IIM	Idiopathic inflammatory myositis
CK	Creatine kinase
IVIg	Intravenous immunoglobulins
GC	Glucocorticosteroids
SCIg	Subcutaneous immunoglobulins
HMGCR	HMG–CoA reductase
IMNM	Immune-mediated necrotizing myopathy
MRC	Medical Research Council Manual Muscle Testing
SSI	Steroid-sparing immunosuppressant

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Author contributions

NW wrote the manuscript and performed a literature search; JC, RB, JWC, NM and CI performed a literature search, proofread and edited the manuscript.

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Data availability

Not applicable.

Declarations

Ethics approval and consent to participate

Ethical approval was not required for this single case report, in accordance with the Ottawa Hospital Research Institute for Ottawa Health Science Network Research Ethics Board policy.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interest

The authors declare that they have no competing interest. JC received consultation fees or honorarium for educational presentations from Takeda, CSL Behring. JC is the medical lead for the Ontario Immunoglobulin Treatment program funded by the Ministry of Health, Government of Ontario.

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