Cureus
Part of SPRINGER NATURE

Open Access Case Report

Probable Overlapping Myasthenia Gravis and Myositis: A Case Report

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Review began 10/06/2026
Review ended 10/28/2026
Published 10/30/2026

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DOI: 10.7759/cureus.118853

Abstract

Polymyositis and myasthenia gravis (MG) are both autoimmune disorders with different pathophysiological mechanisms but similar clinical features. Polymyositis is one of the idiopathic inflammatory myopathies, characterised by endomysial inflammation and presents with symmetrical proximal muscle weakness, fatigue, and elevated muscle enzyme levels. MG is a neuromuscular junction disorder with clinical findings of fatigable weakness with ocular and bulbar involvement. Their coexistence is rare and may lead to significant diagnostic delay, hence, presenting this case can raise awareness of the concomitant occurrence of polymyositis and MG.

We report a 68-year-old man who presented with progressive proximal muscle weakness and myalgia with markedly elevated creatine kinase levels of 6,435 U/L. He also developed fluctuating diplopia and evidence of left eye adduction failure on examination. These ocular features raised suspicion for neuromuscular junction involvement. Initial neurological, ophthalmological, and rheumatological assessments, together with imaging and negative serological testing, including acetylcholine receptor (AChR) and muscle-specific kinase antibodies (anti-MuSK), favoured polymyositis. Electromyography and MRI revealed classical myopathic changes in the pelvic girdle, thigh, and shoulder girdle musculature consistent with inflammatory myositis. Despite oral corticosteroids and methotrexate, the patient deteriorated with worsening ophthalmoplegia affecting both eyes, left eye blepharoptosis, and significant muscle weakness, requiring hospital admission, reflecting a suspected corticosteroid-induced exacerbation of MG. A repeat serological test subsequently revealed AChR antibody positivity, suggesting a probable diagnosis of MG with ocular predominance and myositis overlap. Following administration of intravenous methylprednisolone and intravenous immunoglobulin (IVIG), there was significant clinical improvement. After the initial response, rituximab was introduced as long-term immunosuppression.

Ocular involvement is rare in inflammatory myopathies and should prompt evaluation for coexisting MG. This case report adds to the limited existing literature on MG-myositis overlap, highlighting the value of a multidisciplinary team approach in avoiding misdiagnosis and guiding treatment.

Categories: Neurology, Rheumatology, Ophthalmology
Keywords: idiopathic inflammatory myositis, ivig, myasthenia gravis, myositis, ocular involvement, ophthalmoplegia, overlap, polymyositis, ptosis, rituximab

Introduction

Myasthenia gravis (MG) and polymyositis are both autoimmune disorders that can cause muscle weakness, but they differ fundamentally in their pathophysiology and presentation. Polymyositis is a T-cell-mediated disorder of the skeletal muscle, with several mechanisms proposed to explain its underlying pathophysiology. Firstly, abnormal activation of cytotoxic CD8+ T cells and macrophages can cause an immune-mediated response and direct damage to muscle fibres by releasing perforin-1 and granzyme B. Secondly, muscle injury may occur indirectly through the release of inflammatory mediators, including cytokines and interleukins (ILs). A T helper 1-mediated response is triggered via the release of cytokines such as tumour necrosis factor, interferon-gamma, and IL-12, while the T-helper 17 response is driven by pro-inflammatory mediators including IL-17, IL-22, and IL-6, both contributing to the sustained inflammatory cascade underlying muscle damage [1,2].

Clinically, patients with polymyositis usually present with symmetrical proximal muscle weakness, fatigue, and elevated muscle enzymes. Systemic features or involvement of multiple organ systems may also occur, including the joints, lungs, heart, and gastrointestinal tract. While extraocular muscle involvement has occasionally been described in dermatomyositis, it is particularly rare in polymyositis [3]. Population-based studies report a low incidence of idiopathic inflammatory myopathy (IIM), estimated at 17.6 per 1,000,000 person-years [4]. The diagnosis of polymyositis is based on clinical history alongside electromyography (EMG) findings (low amplitude polyphasic potentials, fibrillations at rest and with high-frequency repetitive discharges), raised muscle enzyme activity (creatine kinase (CK), alanine aminotransferase), evidence of systemic inflammation (fever, raised C-reactive protein (CRP) or erythrocyte sedimentation rate), and

muscle biopsy results of endomysial inflammation [2,3].

MG is an antibody-mediated disorder of the neuromuscular junction, presenting as fatigable weakness due to antibodies against the postsynaptic membrane acetylcholine receptor (AChR), impairing synaptic transmission [5,6]. In contrast to polymyositis, MG classically presents with fatigable weakness of skeletal muscles that worsens with repeated activity and improves with rest. The extraocular muscles are commonly affected, manifesting as ptosis and ophthalmoplegia, while bulbar and respiratory muscle involvement may develop as the disease progresses. MG has an annual incidence of approximately 10 per million and a prevalence of 150-300 per million [5]. Diagnosis is guided by this clinical pattern together with confirmatory serological and electrodiagnostic testing. AChR antibody positivity is the most prevalent finding, present in 85% of patients, while approximately 6% of patients have anti-MuSK antibodies and up to 10% remain seronegative [5]. Electrodiagnostic studies, such as repetitive nerve stimulation (RNS) and/or single-fibre EMG, together with ice-pack testing and thymic imaging to identify a thymoma, form the remainder of the diagnostic pathway.

The presence of ocular symptoms in a patient with suspected inflammatory myopathy presents a diagnostic challenge and should raise suspicion for MG-myositis overlap. Both disorders are acquired autoimmune conditions and share similar presenting features, including limb weakness and fatigue. Symptoms may therefore be attributed to a single diagnosis, with each condition potentially obscuring the other, resulting in delayed diagnosis or suboptimal treatment. The coexistence of MG and IIM is rare, with only 69 cases reported to date [7]. Early recognition is important, as corticosteroid therapy may precipitate transient worsening of MG, and bulbar or respiratory involvement necessitates close monitoring. We describe a patient with progressive proximal myopathy and ocular involvement, in which extensive investigation ultimately led to the probable diagnosis of polymyositis-MG overlap syndrome. Diagnosis was challenging, as initial serological and electrodiagnostic investigations were not supportive of MG, with delayed seroconversion to anti-AChR antibody positivity. This case highlights the importance of maintaining a high index of suspicion for MG in patients with myositis and ocular signs, and of repeating investigations when initial results are negative.

Case Presentation

A 68-year-old man with well-controlled hypertension and asthma presented initially with proximal symmetrical weakness in the upper and lower limbs, mild myalgia, and intermittent diplopia. He reported progressive difficulty rising from chairs and raising his arms above his head. Over the following weeks, he experienced further episodes of intermittent diplopia, lasting for a few hours every day, with each flare-up self-resolving within four to five days. Neurological examination revealed left-eye deviation with adduction failure and no fatigability, proximal weakness of both shoulders, and mild bilateral proximal lower limb weakness, with normal reflexes and downgoing plantars. There was no facial weakness, with preserved speech and cough, and no evidence of rash, photosensitivity, Raynaud's phenomenon, or sicca symptoms. Clinical signs for MG screening, including sustained upgaze test, ice pack testing, and Cogan's lid twitch, were negative.

His regular medications include asthma inhalers, lisinopril, and atorvastatin, the latter having been commenced four years before this presentation. CK measured by the patient's GP at the time of presentation was markedly elevated at 3618 U/L. Atorvastatin was discontinued immediately, and he was referred for specialist assessment.

His CK rose further to 6435 U/L despite cessation of atorvastatin. The remaining laboratory blood results revealed deranged transaminases and a mildly raised CRP, with normal renal function, full blood count, thyroid function, and HbA1c. In line with current British Society for Rheumatology (BSR) guidance that all patients with suspected IIM should be tested for myositis autoantibodies, a broad autoimmune and myositis-specific antibody panel was sent [8]. All myositis-specific antibodies were negative, arguing against antisynthetase syndrome, statin-induced necrotising myopathy, dermatomyositis, and scleroderma-polymyositis overlap. Anti-Ro (SS-A) was positive with a negative antinuclear antibody and elevated rheumatoid factor; however, in isolation, this pattern was not considered specific for an underlying connective tissue disease such as Sjogren syndrome and more likely reflected a non-specific immune activation. The antineutrophil cytoplasmic antibody (ANCA) screen was negative, and thyroid function tests showed mildly elevated thyroid-stimulating hormone with normal free T4 (Table *1*). In view of the normal extraocular muscles on orbital MRI, thyroid eye disease was considered unlikely (Table *2*).

Test	Result	Reference range
Acetylcholine receptor (AChR) antibody, 1st sample	Negative	<2.5×10⁻¹⁰ mol/L
Acetylcholine receptor (AChR) antibody, 2nd sample	Positive	5.8×10⁻¹⁰ mol/L
Anti-MuSK antibody (both samples)	Negative	-
Myositis-specific antibody panel (Ro-52, Jo-1, PL-7, PL-12, OJ, EJ, SRP, Mi-2a, Mi-2b, TIF1g, MDA5, NXP2, SAE1, PM-Scl75, PM-Scl100, Ku)	Negative	-
Anti-HMGCR antibody	Negative	<1.5 CU/mL
Anti-Ro (SS-A)	61.9 U	<30 U
Extractable nuclear antibodies (anti-La (SS-B), anti-U1RNP, anti-SM, anti-Scl-70, anti-Jo1, anti-CENP)	Negative	-
ANCA (PR3, MPO)	Negative	-
Rheumatoid factor (RF)	35.9 IU/mL	<20.1 IU/mL
CRP	11 mg/L	<5 mg/L
Immunoglobulin G	17.49 g/L	7.00-16.00 g/L
Immunoglobulin A	5.45 g/L	0.70-4.00 g/L
Immunoglobulin M	0.53 g/L	0.42-2.30 g/L
TSH	5.53 mU/L	0.27-4.20 mU/L
Free T4	18.6 pmol/L	11.9-21.6 pmol/L
Treponemal total antibody (syphilis screen)	Non-reactive	-
HIV, hepatitis B, and hepatitis C serology	Negative	-

TABLE 1: Summary of laboratory investigations, including myositis autoantibody and autoimmune and infection screen

ANCA, antineutrophil cytoplasmic antibody; CRP, C-reactive protein; HIV, human immunodeficiency virus; TSH, thyroid-stimulating hormone.

Investigation	Key findings
MRI head (non-contrast)	No structural, ischaemic, or space-occupying abnormality
CT angiogram, aortic arch, and carotids	Patent, unremarkable major and extracranial arteries
CT chest, abdomen, pelvis	No malignancy. No thymoma present
Electromyography (EMG)/nerve conduction studies (NCS)	Fibrillations and positive sharp waves were observed in several sites; motor unit action potentials were short in duration, polyphasic, and low in amplitude, mostly 2-4 mV. Findings are suggestive of myopathic changes in the proximal shoulder girdle, hand and thigh muscles, and orbicularis oris. Normal nerve conduction with no decrement/increment on repetitive stimulation
MRI pelvis and proximal femur	Symmetrical multicompartmental intramuscular oedema (psoas, gluteal, quadriceps, hamstrings, adductors); fatty atrophy of hamstrings; pattern compatible with polymyositis
PET-CT	No FDG-avid malignancy; widespread striated muscle uptake (shoulder girdles, gluteus medius) in keeping with active myositis
MRI brain and orbits with contrast	Normal optic nerves and extraocular muscles; no retro-orbital mass or chiasmal compression

TABLE 2: Imaging and neurophysiological investigations

MRI, magnetic resonance imaging; PET-CT, positron emission tomography-computed tomography.

Magnetic resonance imaging (MRI) of the head and computed tomography (CT) angiogram of the aortic arch and carotid arteries were unremarkable, while CT imaging of the thorax, abdomen, and pelvis revealed no evidence of malignancy or paraneoplastic causes of his presenting features. MRI of the pelvis and thighs demonstrated a symmetrical, multicompartmental pattern of muscle oedema strongly supportive of polymyositis (Figure *1*, Table *2*).

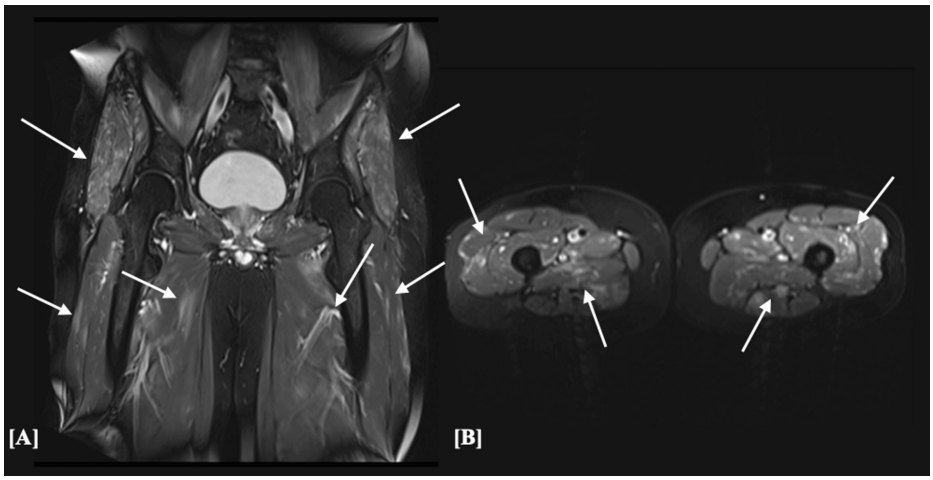


FIGURE 1: MRI pelvis and femur demonstrating inflammatory changes consistent with myositis

[A] Coronal and [B] axial STIR images showing symmetrical, patchy, hyperintense signal within the gluteal, adductor, and thigh musculature bilaterally (arrows), consistent with muscle oedema and active myositis.

Inflammatory myopathy was favoured over other differentials, including MG, given the raised CK, negative AChR and anti-MuSK antibodies, absence of fatigability on examination, and supportive MRI femur/pelvis imaging. Ophthalmoplegia was noted to be atypical for polymyositis, though it has occasionally been

described in the literature [3]. A tapering course of oral prednisolone, starting at 50 mg daily, was commenced, with methotrexate 15mg weekly to be introduced shortly afterwards while awaiting further confirmatory investigations.

Four weeks into the oral treatment, the patient acutely deteriorated with significantly reduced mobility due to muscle weakness, bilateral complex ophthalmoplegia, and left eye ptosis requiring hospital admission. This deterioration possibly reflected a corticosteroid-induced exacerbation of MG. Formal neuro-ophthalmology assessment during admission found visual acuities of 6/4 (right) and 6/6 (left); impairment of right adduction and supraduction; marked limitation of left infraduction, adduction and abduction; left blepharoptosis with only minimal improvement on ice-pack testing and no obvious fatigability on formal testing; and pupillary reactions were normal (Figure *2*). Mild hypophonia was present, but no dysarthria. The patient reported no dysphagia. Serial forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) measurements were monitored closely, with results suggestive of preserved respiratory reserve throughout; therefore, he did not require any ventilatory support.



FIGURE 2: Clinical photograph taken during neuro-ophthalmology assessment in primary gaze, at hospital admission, showing right exotropia (outward deviation of the right eye) and left blepharoptosis

During admission, an extensive investigation workup was performed, including imaging and neurophysiology studies. Notably, EMG confirmed a myopathic pattern in the proximal shoulder girdle, thigh and hand muscles, and in the orbicularis oris, while nerve conduction studies and RNS were normal, with no decrement or increment to suggest a superimposed neuromuscular junction disorder. PET-CT excluded an underlying malignancy but showed prominent striated muscle uptake in the shoulder and gluteal muscles, supportive of polymyositis, while MRI of the brain and orbits was unremarkable, arguing against a structural or compressive cause for his ophthalmoplegia (Table *2*, Figure *3*).

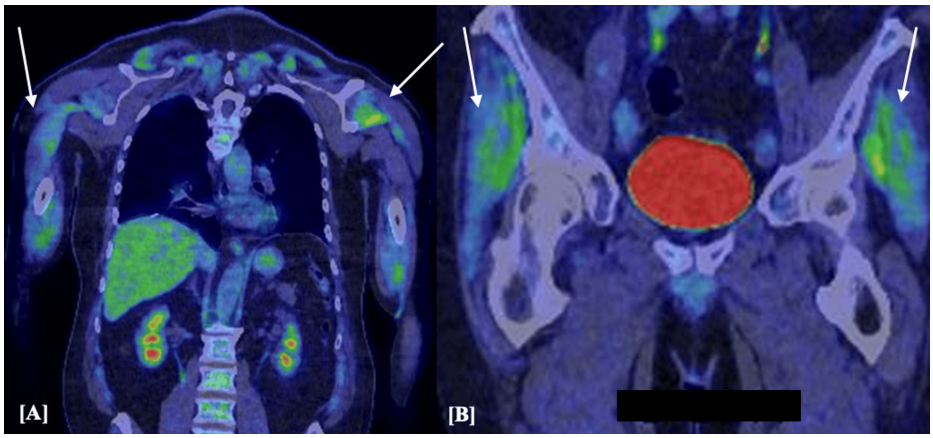


FIGURE 3: PET CT scan coronal views

Increased FDG uptake in [A] the shoulder girdle and [B] the gluteal muscles bilaterally (arrows), consistent with active myositis. Physiological uptake is observed in the liver, kidneys, and bladder.

On admission, he was treated empirically with intravenous methylprednisolone 500 mg daily for three days and intravenous immunoglobulin (IVIG) at a total of 75 g (approximately 1 g/kg), administered as 25 g daily for three days. Daily physiotherapy, including active cycle of breathing technique, was provided to support

respiratory function. He was discharged on a tapering course of oral corticosteroids starting from 50 mg prednisolone and 20 mg methotrexate. Rituximab 1 g was introduced a few weeks following discharge, aimed at reducing the risk of future relapse. A formal fragility fracture risk assessment was also undertaken in view of his cumulative glucocorticoid exposure, consistent with recommended bone health surveillance in IIM. Oral bisphosphonates and vitamin D maintenance therapy were introduced.

The patient showed a sustained clinical and biochemical response to combined immunosuppression. Four weeks after discharge, he reported no muscle pain, substantially improved limb strength and an increase in his daily step count from around 2000 to 4000 steps. His CK fell from a peak of 6435 U/L to 706 U/L, with normalisation of CRP and improvement in transaminases (Table *3*). Serial ophthalmological review documented gradual improvement in ocular motility. By two months post-discharge, ductions were intact with no nystagmus, though an exotropia of approximately 60 prism diopters persisted, along with slightly hypermetric horizontal saccades. By five months post-discharge, ocular motility was full, with residual exophoria of 35 prism diopters at distance and 40 prism diopters at near, and binocular single vision was maintained with normal saccades. Ptosis had fully resolved (Figure *4*).

Timeline (days from initial presentation)	CK, U/L (40-320)	CRP, mg/L (<5)	ALT, U/L (0-40)	Actions taken
Day 0: Initial presentation	3618	7	113	Statin discontinued
Day 44: Approximately 6 weeks since presentation and statin discontinuation	6435	11	-	Steroids started
Day 65: Approximately 3 weeks following steroid initiation	4262	4	188	Methotrexate started
Day 74: Approximately 4 weeks following steroid initiation	5550	23	207	Acute clinical deterioration and hospital admission. IV methylprednisolone and IVIG started
Day 77: Three days following IVIG and IV methylprednisolone	1577	3	-	Ongoing physiotherapy and serial FVC/FEV1 monitoring
Day 81: 1 week since hospital admission	811	2	153	Hospital discharge
Day 110: Approximately 4 weeks post-discharge at outpatient's follow-up	706	1	62	Patient remains on methotrexate and weaning course of steroids. Consented for rituximab
Day 131	444	8	68	Rituximab initiation
Day 154	475	10	48	Patient remains on methotrexate and a weaning course of prednisolone. Ongoing follow-up

TABLE 3: CK, ALT, and CRP trajectory during patient's journey

Numbers correlate with active disease, with peak numbers during the time of maximal clinical deterioration, followed by a sustained fall with escalation of immunosuppression.

ALT, alanine aminotransferase; CK, creatine kinase; CRP, C-reactive protein; IV, Intravenous; IVIG, intravenous immunoglobulin; FEV1, forced expiratory volume in 1 second; FVC, forced vital capacity.



FIGURE 4: Clinical photograph from a neuro-ophthalmological assessment five months after hospital treatment, demonstrating clinical improvement and good ocular motility

Repeat serological testing undertaken on admission, prompted by persisting clinical suspicion of MG, revealed AChR antibody positivity at $5.8x10^{-10}$ mol/L (reference range $<2.5x10^{-10}$ mol/L), in contrast to the negative result obtained on the initial sample taken at first presentation. Anti-MuSK antibody remained negative on both occasions. This seroconversion, together with blepharoptosis and ophthalmoplegia, supports a possible diagnosis of an overlap between ocular MG and polymyositis.

Discussion

IIMs and MG are individually well-characterised conditions, but their coexistence is rare and likely underestimated. Ophthalmoplegia and blepharoptosis are rarely observed in IIM and should prompt screening for an alternative, co-existing pathology. In this patient, the diagnosis continues to rest on a combination of clinical, imaging (MRI/PET), and electromyographic findings favouring polymyositis, alongside ocular involvement attributable to MG and raised AChR antibody titres. This atypical ocular picture was the strongest clinical clue to a second neuromuscular junction process rather than muscle involvement from polymyositis alone.

This case reinforces the importance of a multidisciplinary approach. Neurology, rheumatology, and ophthalmology input, together with an extensive diagnostic work-up, was required to establish a complete clinical picture and exclude numerous differentials. The absence of dermatological involvement, including heliotrope rash or Gottron's papules, argued against dermatomyositis, a diagnosis further excluded by negative myositis-specific antibodies (MSAs) (anti-Mi-2, anti-TIF1γ, anti-NXP2, and anti-SAE antibodies). Necrotising myopathy was considered unlikely given negative anti-SRP and anti-HMGCR antibodies. Patients with myositis may also have an overlapping autoimmune disease such as systemic lupus erythematosus, Sjögren's syndrome, or systemic sclerosis, none of which were supported by the autoimmune screen or clinical presentation in this case. Immune-mediated inflammatory infiltration of the extraocular muscles was excluded given the unremarkable MRI of the orbits, while CT/PET imaging excluded malignancy and paraneoplastic causes. Thyroid eye disease was considered unlikely, given normal extraocular muscles on orbital MRI. Vasculitis and mononeuritis multiplex diagnoses were not supported by normal ANCA levels. Infection screen, including syphilis, human immunodeficiency virus (HIV), hepatitis B, and hepatitis C, was unremarkable.

A key diagnostic learning point from this case is the limitation of AChR antibody testing due to limited sensitivity, particularly in ocular disease. Sensitivity is reported at approximately 85% in generalised MG but only around 50% in purely ocular MG. In selected seronegative cases, particularly where clinical suspicion for MG remains high, repeat testing may be useful, as delayed seroconversion can occur, although it is rare [5]. It is also worth noting that RNS, although highly specific for a neuromuscular junction disorder, is markedly insensitive at 10% in ocular MG; therefore, the normal study performed in this patient did not exclude a coexisting diagnosis at that stage [9].

This report adds to the limited but growing literature describing the co-occurrence of MG and IIM, most often polymyositis, as part of an overlapping autoimmune process. Based on a review of published case reports, 69 patients with concurrent MG and IIM have been reported, with polymyositis accounting for the majority of myositis subtypes (44/69) [7]. Anti-AChR antibodies were commonly present, while MSAs were largely absent. Onset was simultaneous in a substantial proportion of cases (46%), whereas in others, one condition typically precedes the other by months to years [7]. There was a strong association between thymoma and the MG-IIM overlap, with more than 60% of individuals having an underlying thymoma [7]. These findings are further supported by a retrospective cohort study, in which Garibaldi et al. identified 13 patients with MG-IIM association among 441 individuals with MG (2.9%) [10]. This study also confirms AChR antibody positivity in all cases and the absence of MuSK, LRP4, and MSAs. Thymic pathology was present in 10 of 13 (77%) patients, and this subgroup was additionally positive for anti-titin and anti-RyR1 antibodies, regardless of their clinical phenotype. Importantly, Garibaldi et al. proposed that focal or milder forms of

myositis may be underrecognised in myasthenic patients, as overlapping fatigability can obscure the non-fluctuating weakness characteristic of IIM, and suggested a low threshold for CK testing to screen for IIM overlap [10].

The mechanism underlying MG-myositis overlap remains incompletely understood. A proposed explanation is dysregulation of the immune checkpoint pathway involving programmed cell death 1 (PD-1), programmed cell death ligand 1 (PD-L1), and cytotoxic T-lymphocyte-associated protein 4 (CTLA-4), resulting in T-cell activation and autoimmunity, affecting both the neuromuscular junction and muscle fibres [7,11]. This overlap has also been reported as an immune-related adverse event in patients treated with immune checkpoint inhibitors targeting either PD-1 or CTLA-4 [7,11].

Furthermore, this case was characterised by clinical deterioration despite conventional oral immunosuppressive therapy with prednisolone and methotrexate. A corticosteroid-induced exacerbation of MG is one possible explanation, as this is recognised particularly in the early weeks of treatment, typically within the first one to two weeks. However, clinical deterioration in this case was recognised at four weeks, which is later than would usually be expected. Natural progression of the underlying MG-myositis overlap and an inadequate response to oral therapy cannot be excluded, and the available evidence does not allow the cause to be established definitively. This presentation raises the possibility that overlap syndromes involving MG and IIM may exhibit a more treatment-resistant course than either condition alone, which may warrant an earlier escalation to IV therapy, such as IVIG and IV methylprednisolone. Earlier consideration of IV treatment in overlap syndrome may help to prevent clinical deterioration and to achieve adequate disease control. IVIG is part of the first-line treatment of MG exacerbations, but it can also benefit refractory myositis. Respiratory monitoring with serial FVC/FEV1 was instituted in view of the recognised risk of respiratory compromise in both MG and severe myositis, and because high-dose corticosteroids can, paradoxically, worsen weakness in MG. The overall treatment sequence in this case, comprising high-dose glucocorticoids at induction, early introduction of the steroid-sparing disease-modifying antirheumatic drug methotrexate, and escalation to IVIG followed by rituximab for severe and/or refractory disease, mirrors the stepwise approach set out in the 2022 BSR guideline on management of IIM [8].

Limitations of this report include its retrospective, single-patient design and the absence of a muscle biopsy, which was deferred in favour of clinical, imaging, and electrophysiological findings. A biopsy would have added further histopathological confirmation of the diagnosis. Without histopathological confirmation, seronegative immune-mediated necrotising myopathy and inclusion body myositis cannot be fully excluded. In addition, single-fibre EMG was not performed, and the diagnosis of MG therefore rests on the clinical phenotype, AChR antibody positivity, and response to treatment.

Written informed consent was obtained from the patient for the publication and presentation of this case report, including clinical details and images.

Conclusions

This case describes a probable rare overlap syndrome of MG and polymyositis, presenting atypically with prominent ocular involvement in the context of an inflammatory myopathy. Ocular involvement in polymyositis is rare and should prompt active reconsideration of an overlapping neuromuscular junction disorder, even when the overall clinical and biochemical picture otherwise strongly favours myositis. RNS and a single negative AChR antibody do not exclude MG, particularly in ocular disease, where sensitivity is substantially lower than in generalised MG.

Existing evidence of this overlap between IIM and MG is limited, and this case report increases awareness of this pattern of clinical presentation. The presence of these co-existing conditions may lead to a more severe presentation and require early escalation to intravenous treatments. A multidisciplinary team approach is crucial to avoid misdiagnosis and exclude mimics.

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.

Concept and design: Marios Chrysostomou, Shiza Chaudhry, Michael Green

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Drafting of the manuscript: Marios Chrysostomou, Shiza Chaudhry, Michael Green

Critical review of the manuscript for important intellectual content: Marios Chrysostomou, Syed-Masood Ahmed, Michael Green

Supervision: Michael Green

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.

Acknowledgements

The authors would like to thank the patient for their cooperation and for granting consent to publish the details of this case, including clinical images. The authors also acknowledge the contributions of the neurophysiology, radiology, rheumatology, neurology, and ophthalmology teams at York Teaching Hospital NHS Foundation Trust for their input into the diagnostic workup of this case.

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