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Single-Dose Nivolumab as a Trigger of Myocarditis, Myositis, and Myasthenia Gravis Overlap Syndrome With Late Cardiac Death Despite Initial Recovery: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF 1 Deepika Beereddy
ABCE 1 Durga Naga Malleswara Rao Jonnalagadda
CDE 2 Shobha Mandal
B 1 Jieyu Zhang

1 Department of Internal Medicine, Reading Hospital-Tower Health, Reading, PA, USA
2 Department of Hematology and Oncology, Reading Hospital-Tower Health, Reading, PA, USA

Corresponding Author: Deepika Beereddy, 420 S 5th Ave, West Reading, PA 19611, Phone: (484)628-8000, Fax: (484)659-0010, e-mail: deepika.beereddy@towerhealth.org

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Patient: Male, 73-year-old
Final Diagnosis: Immune checkpoint inhibitor-associated myocarditis, myositis, and myasthenia gravis overlap syndrome
Symptoms: Dark urine • exertional dyspnea • gait instability • general weakness • intermittent diplopia • intermittent dysphagia
Clinical Procedure: —
Specialty: Cardiology • Neurology • Oncology
Objective: Rare disease

Background: Immune checkpoint inhibitors (ICIs) have transformed the treatment of advanced malignancies but can cause life-threatening immune-related adverse events. Myocarditis, myositis, and myasthenia gravis (MMM) overlap syndrome is a rare, highly morbid complication with high mortality. Most cases develop early in therapy, sometimes after a single dose.

Case Report: A 73-year-old man with resected stage IIIC malignant melanoma presented 4 weeks after his first dose of adjuvant nivolumab with progressive weakness, gait instability, dyspnea, and dark-colored urine. Workup revealed markedly elevated troponin (11 162 ng/L), creatine kinase (5518 IU/L), and transaminases (aspartate transaminase 614 IU/L, alanine transaminase 497 IU/L), with new right bundle branch block on electrocardiography. ICI-associated myocarditis, myositis, and hepatitis were diagnosed; high-dose intravenous methylprednisolone was initiated. He subsequently developed diplopia, ptosis, bulbar weakness, and respiratory compromise from myasthenia gravis, prompting plasmapheresis and pyridostigmine. He improved with escalating immunosuppression and was discharged on oral prednisone and pyridostigmine after 16 days. Nivolumab was permanently discontinued. Two and a half weeks later, he had a fatal out-of-hospital cardiac arrest.

Conclusions: This case illustrates the severe and unpredictable course of MMM overlap syndrome after a single dose of nivolumab. Despite early aggressive immunosuppression and apparent recovery, the patient had a delayed fatal cardiac event. Given the risk of relapse during corticosteroid taper, structured post-discharge cardiac surveillance with serial troponin and ambulatory rhythm monitoring may help detect subclinical activity or arrhythmia. Prospective studies are needed to define optimal monitoring and identify predictors of late mortality.

Keywords: Case Reports • Immune Checkpoint Inhibitors • Melanoma • Myasthenia Gravis • Myocarditis • Myositis • Nivolumab
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/953173

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Introduction

Immune checkpoint inhibitors (ICIs) are widely used in the treatment of advanced malignancies, including melanoma, and have led to substantial improvements in survival [1,2]. These therapies enhance antitumor immune responses by inhibiting key regulatory pathways such as cytotoxic T-lymphocyte–associated protein 4 (CTLA-4), programmed cell death 1 (PD-1), and programmed death ligand 1 (PD-L1) [1,2]. Nivolumab, a PD-1 inhibitor, was the agent administered in this case. Although highly effective, disruption of immune tolerance by ICIs can lead to immune-related adverse events (irAEs) affecting multiple organ systems [1,2].

Myocarditis, myositis, and myasthenia gravis (MMM) overlap syndrome is a rare but highly morbid irAE. While immune checkpoint inhibitor-associated myocarditis and myositis each occur in fewer than 1% of treated patients, the concurrent development of myasthenia gravis is associated with markedly increased disease severity and mortality [3-5]. Reported mortality rates for MMM overlap syndrome range from approximately 35% to 60%, substantially exceeding those observed with isolated neuromuscular or cardiac irAEs [3,6]. Ventilatory failure and malignant cardiac arrhythmias are the most common causes of death, with many patients requiring pacemaker implantation or experiencing sudden cardiac events [3,6].

MMM overlap syndrome most often develops early during ICI therapy, frequently within the first month and sometimes after a single dose [3,7]. Melanoma is the malignancy most commonly associated with this syndrome, accounting for approximately one-third of reported cases [3,6]. Neuromuscular involvement is often accompanied by markedly elevated creatine kinase levels, positivity for acetylcholine receptor antibodies in a substantial subset of patients, and concurrent myocardial injury [3,6].

Given its rarity, evidence guiding early diagnosis, risk stratification, and long-term management of MMM overlap syndrome remains limited [8]. We describe a case of MMM overlap syndrome with concurrent hepatitis after a single dose of nivolumab, characterized by initial clinical and biochemical improvement with aggressive inpatient therapy followed by delayed fatal cardiac arrest after hospital discharge.

Case Report

A 73-year-old man with stage IIIC malignant melanoma status after wide local excision of the left shoulder approximately 2 months earlier, currently receiving adjuvant nivolumab, presented with progressive generalized weakness, gait instability, and difficulty walking. Symptoms began 4 to 5 days prior to presentation and progressively worsened. At baseline, he was fully independent and capable of walking several miles; however, 1 week before admission, he became unable to walk independently, and he had dark-colored urine for 4 days.

The patient had received his first dose of monthly adjuvant nivolumab approximately 1 month earlier. He presented to the chemotherapy infusion center for his second scheduled dose but was referred to the emergency department because of worsening weakness. He denied recent infections, sick contacts, falls, trauma, fever, diarrhea, or dysuria. COVID-19 and influenza testing performed 1 week earlier for intermittent dyspnea were negative. He was not taking any medications other

Table 1. Serial trends in troponin, creatine kinase, aspartate transaminase (AST), and alanine transaminase (ALT) during the clinical course.

	Day 0 on presentation to ED	Day 0-1 repeat labs	Day 4 start of plasmapheresis	Day 12 completion of 5 sessions of plasmapheresis	Day 15 close to discharge	2 weeks after discharge
Troponin (ng/L)	11 162	15 353*	3372	439	N/A	N/A
Creatine Kinase (IU/L)	5518*	4563	391	353	N/A	N/A
Aspartate Transaminase (AST) (IU/L)	614*	518	55	55	62	53
Alanine Transaminase (ALT) (IU/L)	497*	493	139	226	212	162

Footnotes: Day 0 is the day of presentation to the emergency department. Peak values are marked with an asterisk; all peaks occurred on Day 0. Reference ranges: high-sensitivity troponin ≤ 53 ng/L; creatine kinase 46-171 IU/L; AST < 34 IU/L; ALT 10-49 IU/L.

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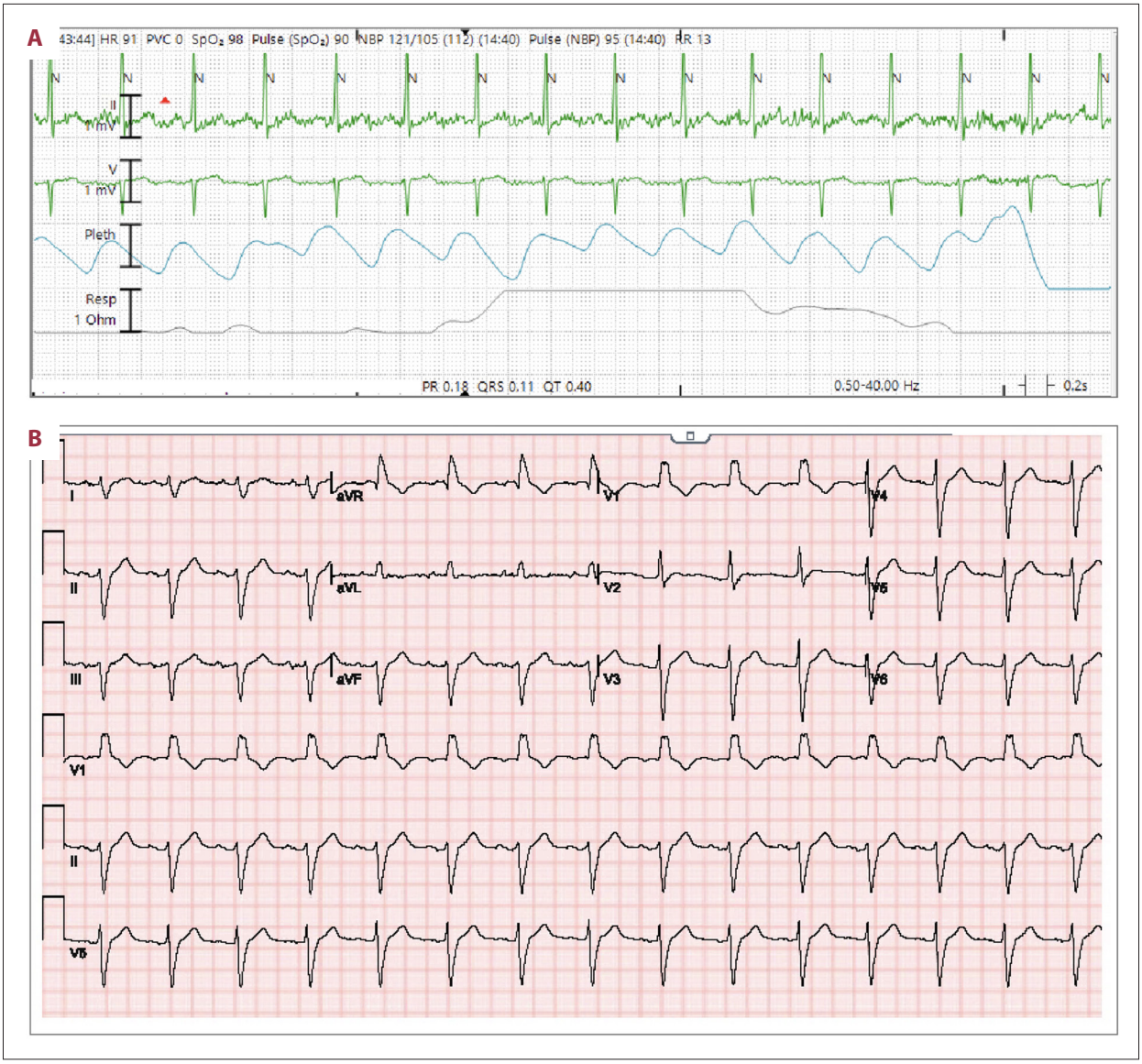

Figure 1. (**A**) Telemetry rhythm strip from approximately 2 months prior showing a narrow QRS complex. (**B**) Electrocardiogram (ECG) on admission showing normal sinus rhythm and new right bundle branch block.

than nivolumab. His medical history was notable for hypertriglyceridemia and autism spectrum disorder.

Initial laboratory evaluation demonstrated markedly elevated high-sensitivity troponin (11 162 ng/L; reference ≤ 53 ng/L), which peaked later the same day at 15 353 ng/L; creatine kinase (5518 IU/L; reference 46-171 IU/L); and transaminases (aspartate transaminase [AST] 614 IU/L, reference < 34 IU/L; alanine transaminase [ALT] 497 IU/L, reference 10-49 IU/L). Creatine kinase and transaminase levels peaked at presentation and subsequently trended downward. The serial trends in laboratory parameters are shown in **Table 1**. The initial electrocardiogram showed sinus rhythm with right bundle branch block and right axis deviation. A telemetry strip obtained about 2 months earlier demonstrated a narrow QRS complex (**Figure 1**). Cardiology evaluated the patient for suspected immune checkpoint inhibitor (ICI)-associated myocarditis, and high-dose intravenous methylprednisolone (250 mg every 6 hours) was initiated. Transthoracic echocardiography revealed normal left ventricular size and preserved systolic function, with an ejection fraction of 64% and no regional wall motion abnormalities (**Figure 2**).

Our patient met the International Cardio-Oncology Society (IC-OS) definition of a probable diagnosis of ICI-associated myocarditis, based on cardiac troponin elevation (above the 99th-percentile upper reference limit) in combination with 3 minor criteria: a compatible clinical syndrome (exertional

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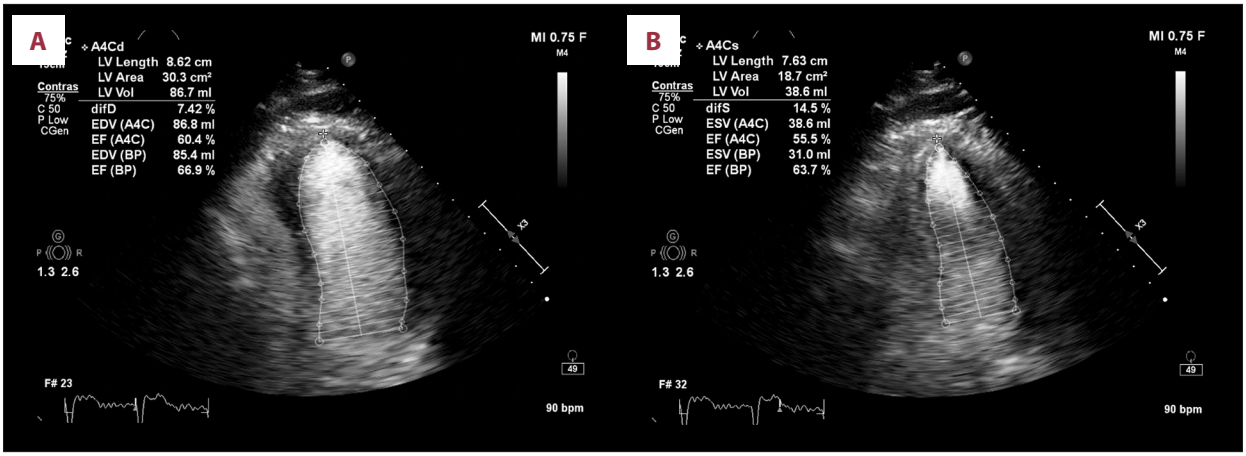

Figure 2. Apical 4-chamber view of transthoracic echocardiogram with Definity contrast (perflutren lipid microsphere) demonstrating preserved left ventricular ejection fraction (64%) in (**A**) end-diastole and (**B**) end-systole.

dyspnea), new conduction-system disease (a new right bundle branch block relative to a prior telemetry strip), and concurrent multiorgan immune-related adverse events (myositis, myasthenia gravis, and hepatitis). The major criteria—diagnostic cardiac magnetic resonance (CMR) by modified Lake Louise criteria and endomyocardial biopsy—could not be obtained due to the acuity of presentation and the rapid initiation of immunosuppression [9].

Hematology/Oncology was also consulted because of concern for immunotherapy-related myocarditis, myositis, and hepatitis, and continuation of corticosteroid therapy was recommended. Immune checkpoint inhibitor-induced myositis was diagnosed clinically based on proximal and generalized muscle weakness, markedly elevated creatine kinase, transaminitis with an aspartate aminotransferase-predominant pattern consistent with muscle origin, and recent nivolumab exposure in the absence of an alternative etiology. Following initiation of treatment, troponin, creatine kinase, and transaminase levels showed progressive improvement.

On hospital day 3, the patient reported intermittent diplopia that had begun approximately 1 week before admission. Physical examination demonstrated bilateral ptosis, raising suspicion for myasthenia gravis. Serologic testing for myasthenia gravis was performed. ICI-induced myasthenia gravis was diagnosed clinically based on fluctuating diplopia, bilateral ptosis, and bulbar involvement (hypophonia, dysphagia) in the setting of recent nivolumab exposure. Clinical improvement following plasmapheresis and pyridostigmine further supported the diagnosis. Serology subsequently returned negative, including acetylcholine receptor antibody (< 0.3 nmol/L; negative), muscle-specific kinase antibody (negative, < 1: 10), and low-density lipoprotein receptor–related protein 4 antibody (negative), consistent with seronegative myasthenia gravis. He subsequently developed a new oxygen requirement of 2 L/min via nasal cannula. Neurologic evaluation revealed bulbar involvement, including intermittent dysphagia, facial weakness, hypophonic speech, and bilateral ptosis, findings concerning for myasthenic crisis. The patient was placed on serial monitoring of negative inspiratory force and vital capacity, and plasmapheresis was initiated on hospital day 4.

MRI of the cervical, thoracic, and lumbar spine demonstrated multilevel degenerative changes without evidence of acute pathology. The patient completed 6 days of high-dose intravenous methylprednisolone (1 g/day) and was transitioned to weight-based dosing. Pyridostigmine was initiated and gradually up-titrated. After 2 plasmapheresis sessions, proximal muscle strength improved sufficiently to allow assisted ambulation. Ocular symptoms improved after the third session.

After completion of 5 plasmapheresis sessions, laboratory abnormalities continued to improve, with troponin decreasing to 439 ng/L, creatine kinase to 353 IU/L, and transaminases to AST 55 IU/L and ALT 226 IU/L. Muscle weakness and diplopia improved substantially compared with presentation. Corticosteroids were tapered cautiously, and the patient was discharged on hospital day 16 with oral prednisone 70 mg daily (1 mg/kg/day) and pyridostigmine 90 mg every 8 hours. The prednisone dose was decreased to 60 mg daily after 10 days, with a planned subsequent taper of 10 mg every 2 weeks.

Given the extent of immune-mediated toxicity—including myocarditis, hepatitis, and myasthenia gravis—nivolumab was permanently discontinued at outpatient oncology follow-up, and the patient was transitioned to melanoma surveillance. Approximately 2.5 weeks after discharge, he had an out-of-hospital cardiac arrest at the rehabilitation facility, with a total downtime of approximately 34 minutes; he received an initial 4 minutes of cardiopulmonary resuscitation with return of spontaneous circulation, followed by a second arrest requiring 30 minutes of

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Table 2. Chronological summary of clinical presentation, management, and outcome in a patient with immune checkpoint inhibitor-associated myocarditis, myositis, and myasthenia gravis overlap syndrome.

Time point	Clinical event
~2 months before admission	Wide local excision of left shoulder for stage IIIC malignant melanoma
~4 weeks before admission	First dose of adjuvant nivolumab administered
7 days before admission	Progressive generalized weakness and gait instability began
4 days before admission	Dark-colored urine noted
Day 0 (admission)	Presented with weakness and gait impairment; markedly elevated troponin, CK, and transaminases; ECG showed right bundle branch block; high-dose IV methylprednisolone initiated for suspected ICI-associated myocarditis and myositis
Hospital Day 1-2	Troponin, CK, and liver enzymes began to improve
Hospital Day 3	Development of diplopia and bilateral ptosis; concern for myasthenia gravis; new oxygen requirement
Hospital Day 4	Neurologic evaluation demonstrated bulbar weakness; plasmapheresis initiated
Hospital Day 6	Completed 6 days of high-dose IV methylprednisolone (1 g/day); transitioned to weight-based dosing of IV methylprednisolone (2 mg/kg/day)
Hospital Day 9	Completed 3 sessions of plasmapheresis; Pyridostigmine 60 mg every 8 hours was started
Hospital Day 12	Completed 5 sessions of plasmapheresis with improvement in muscle strength and ocular symptoms
Hospital Day 16	Discharged on oral prednisone (1 mg/kg/day) and pyridostigmine (90 mg every 8 hours)
12 days after discharge	Nivolumab permanently discontinued at outpatient follow-up with oncology
~2.5 weeks after discharge	Out-of-hospital cardiac arrest with prolonged downtime (~34 minutes); death following transition to comfort-focused care

ICI, immune checkpoint inhibitor; ECG, electrocardiogram; CK, creatine kinase; IV, intravenous. Hospital Day 0 denotes the day of hospital admission; subsequent hospital days are referenced relative to admission. All medication doses are reported as administered during hospitalization unless otherwise specified.

cardiopulmonary resuscitation before circulation was again restored. He subsequently died following transition to comfort-focused care. The mechanism of his cardiac arrest could not be confirmed, as he was not on cardiac monitoring at the time of the event. A malignant arrhythmia is the most likely explanation given his recent diagnosis of MMM overlap syndrome. The clinical course and key interventions are summarized in **Table 2**.

Discussion

This case illustrates the severe and unpredictable clinical course of immune checkpoint inhibitor-associated myocarditis, myositis, and myasthenia gravis overlap syndrome with concurrent hepatitis. Despite early recognition and timely initiation of high-dose immunosuppressive therapy, the patient had a fatal out-of-hospital cardiac arrest after apparent clinical stabilization, highlighting the persistent and frequently underappreciated risk associated with this condition [6,8,10].

The pathophysiology of MMM overlap syndrome is not fully elucidated but is believed to involve shared immune-mediated mechanisms affecting skeletal muscle, myocardium, and the neuromuscular junction. Dysregulated T-cell activation, breakdown of immune tolerance, and shared antigenic targets likely contribute to simultaneous multiorgan involvement [2,10]. Histopathologic analyses of immune checkpoint inhibitor-associated myopathy have demonstrated necrotic myofibers, macrophage-predominant inflammatory infiltrates, and increased major histocompatibility complex class I expression, features that help distinguish these conditions from idiopathic inflammatory myopathies [11].

The diagnosis of immune checkpoint inhibitor-related myasthenia gravis remains challenging. Expert consensus criteria recommend the presence of both serologic antibody positivity and electrodiagnostic evidence of impaired neuromuscular transmission to establish a definite diagnosis [12]. However, many patients present with overlapping or incomplete clinical

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features, and antibody-negative cases have been reported, further complicating timely identification [3,11]. In our patient, the clinical diagnosis of myasthenia gravis despite a negative serologic panel is consistent with this seronegative presentation.

Clinical outcomes in MMM overlap syndrome are generally poor. Pooled analyses and systematic reviews have reported in-hospital mortality rates approaching 60%, with arrhythmias, respiratory failure, and early onset of immune-related adverse events identified as indicators of worse prognosis [3,6]. Importantly, improvement in laboratory markers and apparent neurologic recovery do not reliably predict favorable long-term outcomes. Delayed cardiac complications, including fatal arrhythmias, have been described even after clinical stabilization, suggesting ongoing myocardial vulnerability or delayed immune-mediated remodeling [6,10].

Beyond these broad observations, more granular predictors of outcome have emerged from recent case series and registries. Outcomes appear to depend heavily on the timing and intensity of immunosuppression. Early initiation of high-dose corticosteroids has been associated with substantially lower mortality, while severe and steroid-refractory cases have demonstrated benefit from escalation to additional immunomodulatory agents, including intravenous immunoglobulin, plasmapheresis, mycophenolate, antithymocyte globulin, abatacept, and rituximab [13-15]. Biomarker and clinical features further inform prognosis, with higher peak troponin levels, earlier symptom onset after ICI initiation, and the presence of multiorgan immune-related adverse events all correlating with greater disease severity and worse outcomes [16]. Our patient's markedly elevated peak troponin level of 15 353 ng/L on presentation is consistent with this high-risk profile. Electrocardiographic findings provide additional risk stratification; in a large international registry, pathological Q waves, low QRS voltage, complete heart block, and life-threatening ventricular arrhythmias were each independently associated with myocarditis-related mortality [17].

Elevated troponin at the time of discharge has been associated with a higher risk of subsequent major adverse cardiac events [18], and because relapse during corticosteroid taper has been described, continued troponin surveillance after discharge is a reasonable strategy, although prospective data are lacking. Current guidelines, including those from the National Comprehensive Cancer Network, rely on Common Terminology Criteria for Adverse Events severity grading to direct acute management but do not define a structured approach to post-discharge monitoring, representing an important gap given the potential for delayed cardiac deterioration [19]. Notably, ongoing cardiotoxicity can persist despite discontinuation of the offending agent and apparent clinical recovery, with cases of refractory or recurrent myocarditis reported months after the initial event, underscoring the need for sustained cardiac surveillance after hospital discharge [20]. Our patient exhibited several features associated with adverse outcomes in this syndrome, including a new conduction abnormality (right bundle branch block), markedly elevated troponin at presentation, and concurrent multiorgan involvement, although the contribution of these factors to his delayed fatal cardiac event cannot be established with certainty from a single case. Because malignant ventricular arrhythmia and conduction disease are among the leading causes of death in patients with this syndrome, and because such events can occur after apparent clinical recovery, extended ambulatory rhythm monitoring—using a wearable external monitor or, in selected high-risk patients, an implantable cardiac monitor—may be a complementary strategy to biomarker surveillance for detecting electrical instability before catastrophic deterioration, although this approach has not been formally evaluated in this population [17].

Published case reports demonstrate a consistent pattern: patients who recovered typically received early escalation beyond corticosteroids, with plasma exchange and intravenous immunoglobulin initiated within 24 to 72 hours of recognition [3,14,21]. In contrast, fatal cases have most often involved either delayed escalation, an inability to deliver second-line therapy due to hemodynamic instability, or progression to refractory multiorgan failure despite early high-dose corticosteroids [14,20]. This pattern suggests that timely escalation of immunosuppression is an important determinant of survival, although the retrospective and heterogeneous nature of the available evidence precludes firm causal conclusions.

Current management approaches emphasize prompt discontinuation of immune checkpoint inhibitor therapy and early initiation of high-dose corticosteroids, with escalation to plasmapheresis, intravenous immunoglobulin, or other immunomodulatory therapies in refractory cases [8,19]. Although many patients have initial clinical improvement, treatment-refractory disease has been reported, leading to the use of agents such as rituximab, eculizumab, or other targeted immunotherapies based on limited case reports and small series [22]. Evidence guiding the management of steroid-refractory MMM overlap syndrome remains limited, and standardized treatment algorithms are not yet established [3,6].

Data regarding relapse after initial recovery are scarce but suggest that recurrence can occur during corticosteroid tapering or weeks to months following hospital discharge [6]. Emerging evidence also indicates that thymic abnormalities may increase susceptibility to severe immune checkpoint inhibitor-related myotoxicities, raising the possibility that clinico-radiologic assessment of the thymus could aid future risk stratification [23].

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This case reinforces the importance of thorough neuromuscular and cardiac evaluation in patients presenting with immune checkpoint inhibitor-associated myocarditis or myositis, as myasthenic manifestations can be subtle or under-recognized.

Further studies are needed to identify reliable predictors of disease severity, relapse, and delayed cardiac risk, and to develop evidence-based management and monitoring strategies aimed at reducing both early and late mortality associated with this rare but life-threatening immune-related adverse event [6,8,10].

Conclusions

Myocarditis, myositis, and myasthenia gravis overlap syndrome is a severe and unpredictable complication of immune checkpoint inhibitor therapy with high associated mortality. This case demonstrates that, despite prompt recognition and aggressive inpatient management resulting in initial clinical improvement, patients remain at risk for delayed fatal outcomes after discharge. Because the risk of relapse persists during corticosteroid taper, our case suggests a potential role for structured post-discharge cardiac surveillance—including serial troponin monitoring and prolonged ambulatory rhythm monitoring (eg, with a wearable or implantable cardiac monitor)—to detect subclinical disease activity or arrhythmia before catastrophic deterioration. Prospective studies are needed to define the optimal modality, intensity, and duration of such monitoring and to identify predictors of persistent cardiac risk in this rare but devastating immune-related adverse event.

Department and Institution Where Work Was Done

Department of Internal medicine and Department of Hematology and Oncology, Reading Hospital-Tower Health, Reading, PA, USA.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying information.

AI Statement

The AI models ChatGPT (OpenAI) and Claude Opus 4.7 (Anthropic) were used during preparation of this manuscript, primarily for English language editing and formatting assistance.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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