Journal of Geriatric Cardiology | JGC
LETTER TO THE EDITOR
J Geriatr Cardiol 2026; 23(9): 647–654

Immune checkpoint inhibitor-induced myocarditis with third-degree atrioventricular block: two case reports and literature review

Natasha Kalombo[1,*], Liang Xu[2,*], Ke Li[1,*], Yunjie Yin[2], Han Tang[2], Wei Zhong[1,✉], Ye Zhou[1,✉]

1. Department of Cardiology, Affiliated Hospital of Jiangsu University, Zhenjiang, Jiangsu, China; 2. Department of Cardiology, Affiliated Yi Xing Hospital of Jiangsu University, Wuxi, Jiangsu, China
[*]The authors contributed equally to this manuscript
✉ Correspondence to: zw8834314@sina.com (ZHONG W); njzy1088@163.com (ZHOU Y)
https://doi.org/10.26599/1671-5411.2026.09.004

Immune checkpoint inhibitors (ICIs), particularly those targeting the programmed cell death-1 (PD-1) and programmed death-ligand 1 (PD-L1) pathways, have revolutionized the landscape of modern oncology, offering unprecedented survival benefits across a variety of malignancies. However, this profound restoration of anti-tumor immunity comes with the inherent risk of disrupting peripheral immune tolerance, leading to a wide spectrum of immune-related adverse events (irAEs). With the rapidly expanding demographic of oncology patients, an increasingly substantial proportion of those receiving ICI therapies are older adults. This geriatric population is inherently vulnerable to cardiovascular toxicities due to a complex interplay of age-related immunosenescence, pre-existing subclinical cardiovascular diseases, and a significantly reduced physiological cardiac reserve. Among the various irAEs, ICI-associated myocarditis (ICIM) is relatively rare, with an estimated incidence of less than 1.5%, but it is notoriously characterized by a fulminant clinical course and alarmingly high mortality rates ranging from 25% to 50%.[1,2]

The clinical prognosis becomes particularly devastating when ICIM is complicated by severe cardiac conduction system abnormalities, specifically third-degree atrioventricular block (AVB). The exact pathophysiological mechanism is believed to involve a shared-antigen response, where activated T-cells erroneously target homologous antigens present in both the tumor microenvironment and the myocardial tissue, leading to intense focal inflammation, edema, and subsequent fibrosis within the fragile His-Purkinje system.[3-5] This profound electrical failure significantly increases the risk of fatal hemodynamic collapse. Here, we present two distinct and challenging clinical cases of fulminant ICIM complicated by third-degree AVB that required urgent pacemaker therapy, alongside a comprehensive review of the existing literature, to provide actionable, evidence-based guidance tailored specifically to the physiological realities of older adults.

Our first case involved a 66-year-old man with a history of gastric adenocarcinoma who was admitted to our cardiovascular department. He had been receiving immunotherapy with the PD-1 inhibitor sintilimab. Twenty-one days after the administration of his initial ICI dose, routine clinical monitoring revealed significantly elevated cardiac biomarkers, although the patient was notably asymptomatic at the time, underscoring the insidious onset of this complication. Upon admission to the intensive care unit, his vital signs were notable for severe and dangerous bradycardia: heart rate, 44 beats/min; blood pressure, 108/66 mmHg. Extensive laboratory investigations demonstrated a marked and acute elevation in myocardial injury markers: troponin I (cTnI), 3.6 ng/mL (0-0.056); myoglobin, 1000 ng/mL (0-70); creatine kinase (CK), 1962 U/L (55-170); and CK-MB, 49.44 ng/mL (0-16). The initial standard 12-lead electrocardiogram (ECG), which was explicitly obtained before any temporary pacing intervention was inserted, revealed at-

© 2026 JGC All rights reserved; www.jgc301.com

rial tachycardia, complete right bundle branch block (CRBBB), an accelerated ventricular escape rhythm, and complete third-degree AVB. To rule out ischemic etiologies, emergency coronary angiography was performed, which revealed normal coronary arteries with no obstructive disease. Given the patient's critical hemodynamic instability and absolute reliance on continuous cardiac monitoring, transferring the patient for a prolonged cardiovascular magnetic resonance (CMR) imaging session or performing an invasive endomyocardial biopsy was deemed clinically unsafe and practically unfeasible in the acute emergency setting. Consequently, sintilimab was permanently discontinued, and aggressive immunosuppression with intravenous pulse methylprednisolone (200 mg/day) was immediately initiated. Remarkably, the third-degree AVB resolved on the very same day of steroid administration, with the rhythm reverting to normal sinus rhythm. Unfortunately, on day 3 of his admission, the patient refused further intravenous therapy due to poor compliance. This premature cessation of corticosteroids led to a rapid and severe rebound in cardiac markers (cTnI soared to 5.38 ng/mL) and a swift progression back to complete AVB with a fragile junctional escape rhythm. High-dose intravenous methylprednisolone was urgently reinitiated to control the overwhelming inflammation, and a temporary transvenous pacemaker was placed to secure his hemodynamics. Continuous monitoring indicated a 100% pacing dependency despite intensive medical therapy. As a result, a dual-chamber permanent pacemaker was implanted 13 days later. The patient was ultimately discharged on day 34, with his underlying intrinsic cardiac rhythm remaining completely pacemaker-dependent (Figure 1).

Our second case highlighted a different clinical presentation. A 54-year-old man undergoing treatment for metastatic nasopharyngeal carcinoma presented to the emergency department with acute-onset severe chest tightness and dizziness. These symptoms manifested 38 days after his eighth cycle of cadonilimab (a bispecific antibody targeting PD-1 and CTLA-4). On admission, he was in profound cardiogenic shock with a pulse of merely 31 beats/min. Laboratory evaluation showed a characteristic pattern of elevated cardiac enzymes indicative of acute myocardial necrosis: cTnI, 5.38 ng/mL (0-0.056); CK, 1261 U/L (55-170); CK-MB, 93.4 ng/mL (0-16) and elevated transaminases AST, 115 U/L (15-40); ALT, 79 U/L (7-40). The 12-lead ECG demonstrated profound third-degree AVB with a wide QRS ventricular escape rhythm. Notably, it also displayed prominent ST-segment elevations simulating an acute ST-elevation myocardial infarction (STEMI) (Figure 2). Emergent

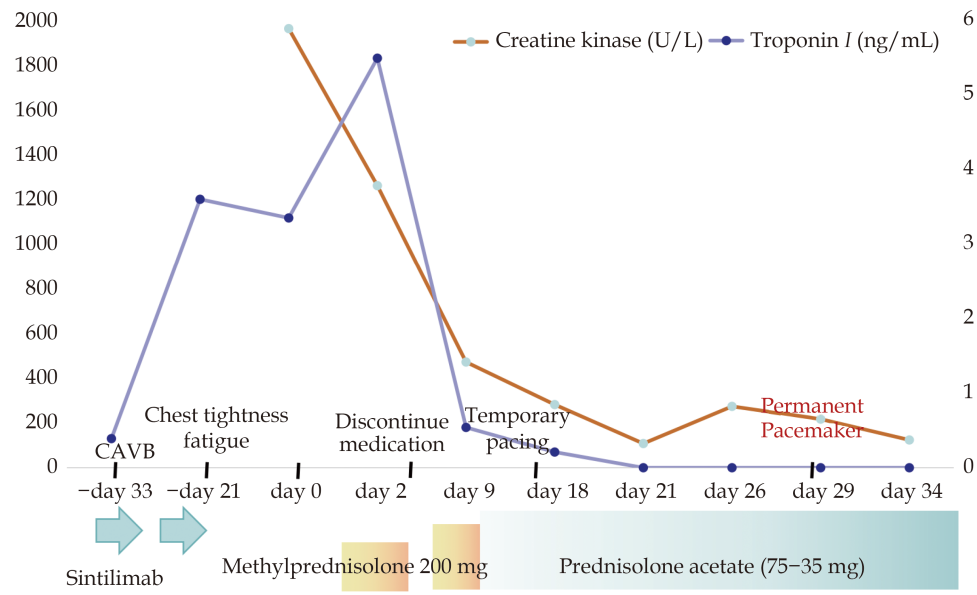


Figure 1 Timeline of clinical course and therapeutic interventions. A detailed timeline illustrating the sequence of events from the initiation of immune checkpoint inhibitor therapy to the onset of symptoms, diagnostic evaluations, dynamic fluctuations of cardiac biomarkers, and step-by-step therapeutic interventions (including specific corticosteroid regimens and the timing of temporary and permanent pacemaker implantations) for Case 1.

coronary angiography was performed simultaneously with the insertion of a temporary pacemaker, which successfully ruled out acute coronary syndrome (Figure 3). Crucially, a retrospective review of his baseline ECG obtained prior to the index episode was completely normal, confirming that he had no pre-existing intraventricular conduction delays or RBBB. Cadonilimab was immediately withdrawn, and immunosuppressive therapy was initiated with moderate-dose intravenous methylprednisolone (160 mg/day). Despite a steady improvement in his clinical symptoms and a decline in cardiac biomarkers, complete AVB stubbornly persisted on day 10 of admission. To ensure long-term hemodynamic stability, a dual-chamber permanent pacemaker was implanted. Interestingly, serial ECGs taken over the following days showed the gradual disappearance of the complete AVB, and the patient's intrinsic rhythm eventually recovered to a normal sinus rhythm prior to discharge. However, this recovery was accompanied by a newly developed intrinsic CRBBB, which likely represents permanent residual inflammatory damage to the fragile His-Purkinje conduction system. The detailed clinical characteristics, therapeutic regimens, and outcomes of both cases are meticulously summarized in Table 1.

To systematically address the prevailing knowledge gaps regarding the true reversibility of high-grade conduction blocks and the optimal timing for pacing strategies, we conducted a rigorous systematic literature search. We queried PubMed, EMBASE, and the Cochrane Library for relevant cases published from inception up to March 2026, utilizing MeSH terms and free-text keywords including "immune checkpoint inhibitors," "myocarditis," and "complete heart block." Following a strict inclusion criterion, we identified 35 previously reported cases with definitive clinical outcomes and detailed pacing data.[6-34] Together with our two current cases, this formed a robust cohort of 37 patients for pooled quantitative analysis (Tables 2 and 3). An age-stratified analysis of this cohort revealed a striking and highly clinically relevant demographic disparity: an overwhelming majority (70.3%) of severe ICIM-AVB cases occurred in older adults aged 65 years or above. More alarmingly, the mortality rate was disproportionately higher in this geriatric subgroup, reaching a staggering 57.7%, compared to a 36.4% mortality rate in younger patients. Furthermore, a stark contrast in survival was observed based on the implementation of pacing interventions. The overall mortality rate among patients who received adequate pacing support (temporary or permanent) was 43%, whereas the mortality rate skyrocketed to 85% for those who did not receive a pacemaker.

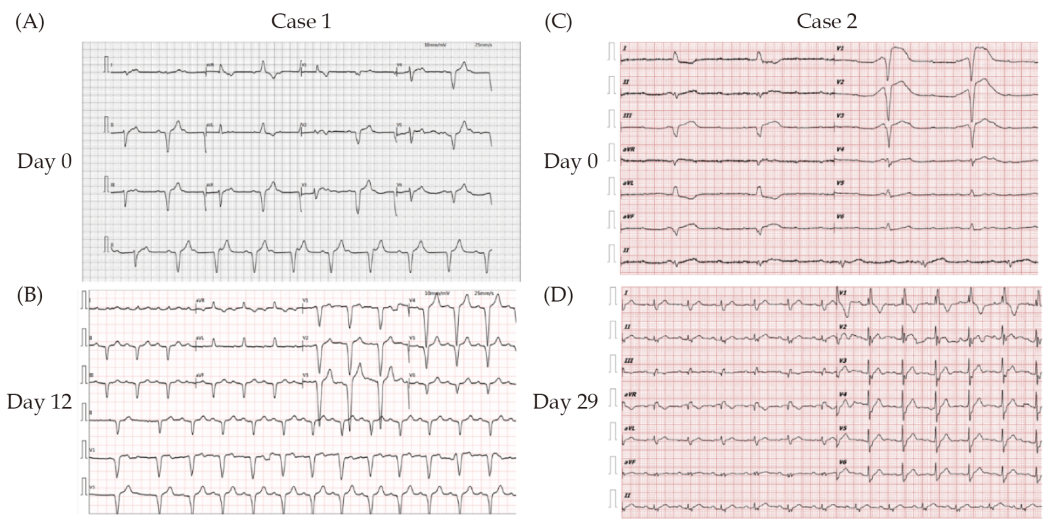


Figure 2 Electrocardiographic (ECG) evolution in both cases. Representative ECGs demonstrating the progression and resolution of cardiac conduction abnormalities. (A): Initial ECG of Case 1 upon admission showing third-degree atrioventricular block (AVB) with an accelerated ventricular escape rhythm and a complete right bundle branch block. (B): Postoperative ECG of Case 1 prior to discharge, demonstrating a 100% pacemaker-dependent rhythm following dual-chamber permanent pacemaker implantation. (C): Admission ECG of Case 2 revealing profound third-degree AVB accompanied by striking ST-segment elevations in the inferior (II, III, aVF) and anterior (V1-V3) leads, mimicking an acute myocardial infarction. (D): Follow-up ECG of Case 2 showing complete resolution of the AV block and restoration of the underlying normal sinus rhythm after combined corticosteroid and pacemaker therapy.

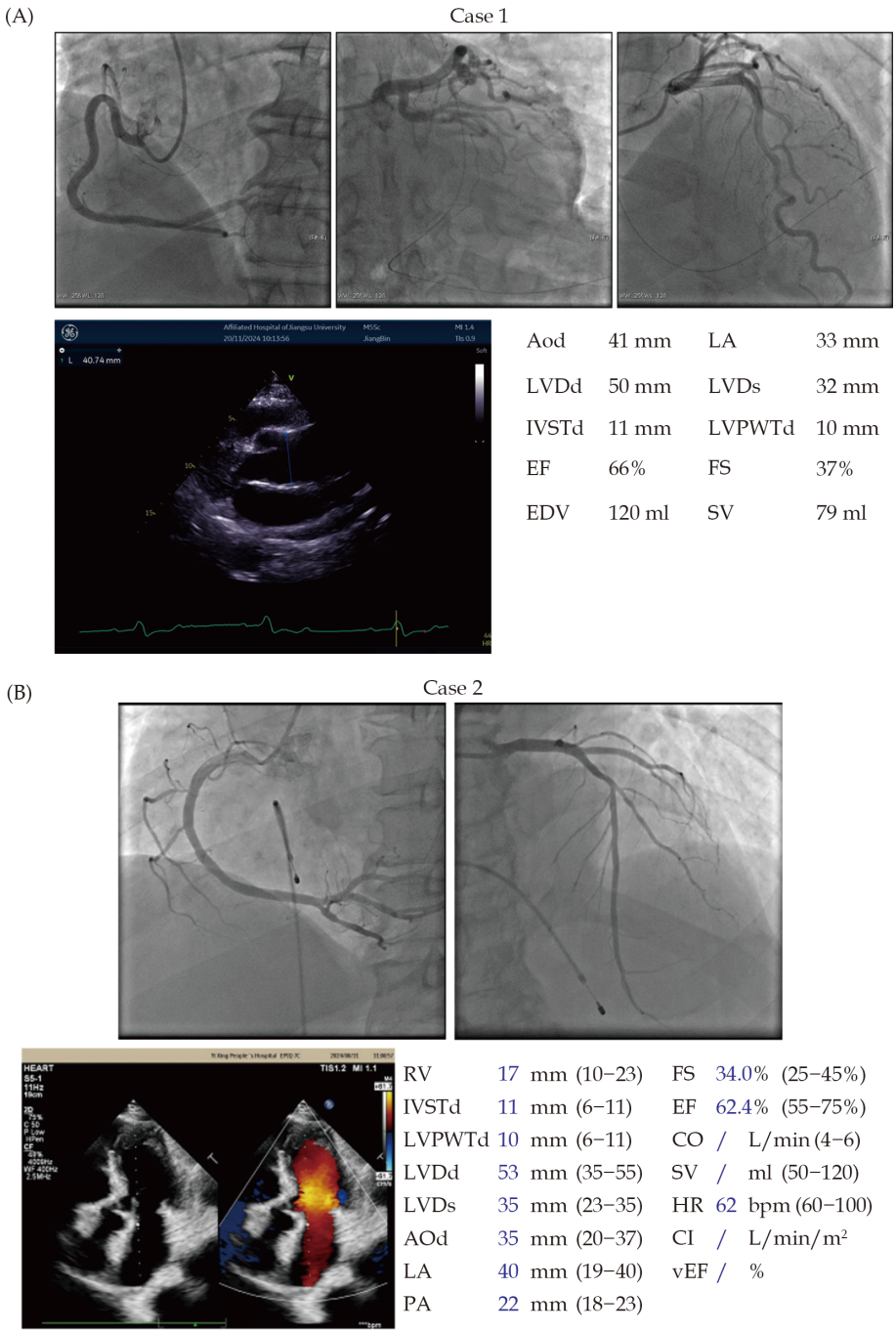


Figure 3 Cardiovascular imaging findings excluding acute ischemic etiologies. (A): Coronary angiography and transthoracic echocardiography results for Case 1; (B): Coronary angiography and transthoracic echocardiography results for Case 2. Both demonstrated the absence of obstructive coronary artery disease, thereby definitively ruling out acute coronary syndrome despite the presence of severe arrhythmias and/or ST-segment elevation. Representative transthoracic echocardiography showing preserved ejection fraction and the absence of significant regional wall motion abnormalities, further supporting the diagnosis of immune-mediated myocarditis rather than an ischemic event.

Diagnosing fulminant ICIM in emergency scenarios remains exceptionally challenging. In highly unstable older patients, clinicians must frequently rely on a "clinically suspected" diagnostic pathway guided by rapid ECG changes and escalating biomarker profiles. While we previously considered CMR imaging unfeasible and

Table 1 Conclusion of the information in two cases.

	Case 1	Case 2
Age, yrs	66	54
Sex	Male	Male
Primary disease	Cardia tumor	Nasopharyngeal cancer with cervical lymph node metastasis
Cancer therapy	Sintilimab	Cadonilimab
Cancer treatment cycle	1	8
Time from the cancer therapy initiation to admission	28 days	228 days
Form of onset	Abnormal cardiac markers	chest tightness and dizziness
Time from the symptom onset of cardiomyopathy toadmission	21 days	3 days
Cardiogenic shock	-	-
Cardiac rhythm	Complete atrioventricular block Accelerated ventricular escape rhythm	complete atrioventricular block
Findings of ultrasound examination	Ejection fraction of 67% without ventricular hypertrophy or segmental ventricular wall motion abnormalities	-
Initial troponin I value, ng/dL	3.6	5.32
Max troponin I value, ng/dL	5.49	5.32
Treatment for myocarditis	Days 2-4: mPSL 200 mg/day, discontinued on day 5 when the patient refused to cooperate with treatment, and Days 6-7: mPSL 200 mg/day, days 8-10: PAT 75 mg/day, days 11-17: 60 mg/day (patient did not take medication as prescribed during this period), days 18-25: 70 mg/day, days 26-30: 40 mg/day, days 31-36: 35 mg/day	Days 0-3: mPSL 160 mg/day, days 4-7: 120 mg/day, days 8-11: 40 mg/day, days 12-15: PSL 20 mg/day, day 16: 10 mg/day
Treatment of complete atrioventricular block	Day 17: implantation of a temporary pacemaker, Day 29: implantation of a permanent pacemaker.	Day 0: implantation of a temporary pacemaker, Day 10: implantation of a permanent pacemaker.
Ejection fraction after treatment	67%	62.40%
Heart rhythm after treatment	First degree atrioventricular block, DDD pacing pattern electrocardiography	Complete right bundle branch block

mPSL: Methylprednisolone; PSL: Prednisolone; PAT: Prednisone Acetate Tablets.

contraindicated in pacing-dependent patients due to the risks of lead heating and displacement, recent institutional advancements have demonstrated that CMR can indeed be safely and effectively performed. This requires a highly specialized technical setup, specifically utilizing active fixation leads inserted via the jugular vein and connecting them to externalized MRI-conditional generators. Furthermore, overcoming the severe metallic susceptibility artifacts requires precise modifications to standard MRI pulse sequences. For instance, utilizing short-tau inversion recovery (STIR) and wide-band phase-sensitive inversion recovery (PSIR) with wide-band pre-pulses ensures uniform inversion recovery and accurate assessment of myocardial edema and late gadolinium enhancement. Conversely, conventional steady-state free precession (SSFP) sequences must be actively avoided for rest perfusion due to their extreme sensitivity to field inhomogeneities caused by the metallic leads. However, we strongly emphasize that when such advanced hardware and imaging expertise are unavailable in acute local settings, the lack of definitive imaging or biopsy must never delay the administration of life-saving corticosteroids and pacing interventions.

In conclusion, ICI-associated myocarditis complicated by third-degree AVB is a catastrophic oncologic emergency that demands rapid clinical identification. While aggressive, individualized corticosteroid therapy remains the primary disease-modifying intervention, our extensive literature review and case observations confirm that the reversibility of cardiac conduction block is

Table 2 Eighteen cases of ICIM complicated by third-degree atrioventricular block with recovery.

No	Ref.	Age/Sex	Myocarditis onset time	CVD	Tumor type	ICI target	EF%	Pulse GC	OIT	Pacing	Myocarditis Outcome	CAVB recovery time, day
1	[6]	68/Male	14	-	Sarcoma	IP+NI	35	+	MYCO	PP	Recovery	-
2	[7]	76/Female	98	-	Lung cancer	NI	15	+	IVIG+PLAS+ INFI	CRT-D	Recovery	-
3	[8]	69/Female	38	-	Lung cancer	NI	normal	+	-	TP	Recovery	7
4	[9]	64/Female	22	-	Glioblastoma	NI	37	+	INFI+ATG+ MMF	TP	Recovery	5
5	[10]	73/Male	16	-	Lung cancer	PE	70	+	-	PP	Recovery	13
6	[11]	70/Female	16	-	Thymoma	PE	normal	+	PLAS	PP	Recovery	-
7	[12]	47/Female	28	-	Thymoma	TO	not mention	+	-	TP	Recovery	12
8	[13]	57/Male	12	-	Renal cancer	IP+NI	50	+	ABAT+MMF	PP	Recovery	-
9	[14]	57/Male	60	-	Lung cancer	IP+NI	normal	+	TCZ	P	Recovery	-
10	[15]	66/Male	25	-	Lung cancer	SI	normal	+	IVIG	PP	Recovery	2
11	[16]	59/Male	21	-	Renal cancer	IP+NI	normal	+	IVIG+PLAS	-	Recovery	2
12	[2]	80/Female	21	-	HNSCC	PE	65	-	-	PP	Recovery	5
13	[17]	65/Male	50	HTN	Esophageal cancer	PE	50	+	-	PP	Recovery	-
14	[17]	54/Male	35	-	Melanoma	PE+IP	35	+	-	TP	Recovery	2
15	[18]	82/Female	46	-	Bladder cancer	IP	normal	+	-	P	Recovery	3
16	[19]	70/Male	19	-	Renal cancer	IP+NI	normal	+	IVIG	TP	Recovery	3
17	ours	54/Male	228	HTN	HNSCC	ZI/CA	62	+	-	PP	Recovery	12
18	ours	66/Male	28	-	Gastric cancer	SI	66	+	-	PP	Recovery	-

Table 3 19 cases of ICIM complicated by third-degree atrioventricular block with lethal outcome.

No	Ref.	Age/Sex	myocarditis onset time	CVD	Tumor type	ICI target	EF%	Pulse GC	OIT	Pacing	Myocarditis Outcome	Treatment to death time, day
1	[3]	63/Male	15	-	Melanoma	IP+NI	50	+	INFI	TP	Death	-
2	[3]	65/Female	12	-	Melanoma	IP+NI	73	-	-	-	Death	-
3	[5]	63/Male	21	HTN	Melanoma	NI	N	-	-	TP	Death	26
4	[20]	66/Male	7	-	CMML	IP	70	+	-	-	Death	1
5	[21]	79/Male	35	-	Gastric cancer	PE	N	+	IVIG+PLAS+ MTX	TP	Death	-
6	[22]	67/Male	16	-	Melanoma	IP+NI	20	+	ATG	PP	Death	-
7	[23]	67/Female	16	-	Myeloma	PE	30	-	INFI	-	Death	10
8	[24]	67/Male	16	HTN	Melanoma	IP+NI	N	+	IVIG+INFI	-	Death	18
9	[25]	33/Male	150	-	Lymphoma	NI	NM	-	MMF+IVIG	-	Death	-
10	[26]	81/Male	12	-	Renal cancer	IP+NI	62	+	PLAS	TP	Death	-
11	[27]	88/Male	22	-	Melanoma	NI	N	+	INFI+MMF	PP	Death	15
12	[28]	48/Female	13	-	Thymoma	PE	45	+	INFI	PP	Death	10
13	[29]	66/Male	24	-	Gastric cancer	NI	NM	+	IVIG+PLAS	TP	Death	103
14	[30]	79/Female	25	-	Melanoma	IP+NI	NM	NM	-	P	Death	-
15	[31]	73/Female	18	-	Melanoma	NI	N	-	-	PP	Death	35
16	[32]	70/Male	14	-	Renal cancer	IP+NI	N	+	MMF+PLAS	TP	Death	-
17	[33]	78/Male	15	CHD	Lung cancer	IP+NI	45	-	-	-	Death	3
18	[34]	74/Male	240	-	Gastric cancer	NI	10	+	IVIG+PLAS	TP	Death	19
19	[1]	68/Female	7	CHD+HTN	Urothelial carcinoma	IP+NI	65	+	MYCO	TP	Death	9

CVD: cardiovascular disease; GC: glucocorticoids; HTN: hypertension; CMML: chronic myelomonocytic leukemia; HNSCC: head and neck squamous cell cancer; CHD: coronary heart disease; IP: ipilimumab; NI: nivolumab; PE: pembrolizumab; SI: sintilimab; TO: toripalimab; ZI: zimberelimab; CA:Cadonilimab; INFI: infliximab; MMF: mycophenolate mofetil; PLAS: plasmapheresis; IVIG: intravenous immunoglobulins; MTX: methotrexate; ATG: anti-thymocyte globulin; ABAT: abatacept; TP: temporary pacemaker; PP: permanent pacemaker; P: pacemaker; CRT-D: cardiac resynchronization therapy defibrillator; TCZ: tocilizumab; N: normal; NM: not mention.

highly unpredictable, especially in the aging heart affected by pre-existing degenerative changes. Therefore, the early initiation of temporary pacing, coupled with a notably low threshold for transitioning to permanent pacemaker implantation, significantly improves survival. For frail elderly patients requiring prolonged immunosuppression, the utilization of leadless pacemakers presents a compelling alternative to mitigate the elevated risks of pocket infections and lead-related complications. Ultimately, stringent geriatric cardio-oncology monitoring protocols and proactive rhythm management are paramount in navigating these devastating arrhythmic events in the vulnerable older demographic.

DISCLOSURE

Ethics Approval and Consent to Participate

This case report was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Written informed consent was obtained from the patient for the publication of this case, including all clinical details and any accompanying images or data. All patient identifiers have been removed or anonymized to protect privacy and confidentiality. The study protocol was reviewed and approved by the Ethics Committee of the Affiliated Hospital of Jiangsu University (clinical trial number: KY2025K0702).

Availability of Data and Materials

All data generated or analyzed during this study are included in this article.

Competing Interests

None.

Fundings

This study was supported by the Natural Science Foundation of China (82000261), Natural Science Research in Universities of Jiangsu Province (BE2021694), and the Social Development Foundation of Zhenjiang (SH2024033, SH2024086).

Authors' Contributions

Natasha K., KL, and LX conceptualized the study and drafted the manuscript. YY and HT collected clinical data and performed a literature review. WZ and YZ supervised the clinical management, critically reviewed the manuscript for important intellectual content, and finalized the submission of the manuscript. All the authors have read and approved the final manuscript.

REFERENCES

[1] Koelmeyer H, Buckley K, Feradov D, Kotch N. Complete heart block in a patient undergoing combination immune checkpoint inhibitor therapy. *Cureus* 2024; 16: e66776.
[2] Su L, Liu C, Wu W, *et al.* Successful therapy for myocarditis concomitant with complete heart block after pembrolizumab treatment for head and neck squamous cell carcinoma: a case report with literature review. *Front Cardiovasc Med* 2022; 9: 898756.
[3] Johnson DB, Balko JM, Compton ML, *et al.* Fulminant myocarditis with combination immune checkpoint blockade. *N Engl J Med* 2016; 375: 1749–1755.
[4] Varricchi G, Galdiero MR, Tocchetti CG. Cardiac toxicity of immune checkpoint inhibitors: cardio-oncology meets immunology. *Circulation* 2017; 136: 1989–1992.
[5] Behling J, Kaes J, Munzel T, *et al.* New-onset third-degree atrioventricular block because of autoimmune-induced myositis under treatment with anti-programmed cell death-1 (nivolumab) for metastatic melanoma. *Melanoma Res* 2017; 27: 155–158.
[6] Reddy N, Moudgil R, Lopez-Mattei JC, *et al.* Progressive and reversible conduction disease with checkpoint inhibitors. *Can J Cardiol* 2017; 33: 1335.e13–e15.
[7] Frigeri M, Meyer P, Banfi C, *et al.* Immune checkpoint inhibitor-associated myocarditis: a new challenge for cardiologists. *Can J Cardiol* 2018; 34: 92.e1–e3.
[8] Fukasawa Y, Sasaki K, Natsume M, *et al.* Nivolumab-induced myocarditis concomitant with myasthenia gravis. *Case Rep Oncol* 2017; 10: 809–812.
[9] Tay RY, Blackley E, McLean C, Moore M, Bergin P, Gill S. Successful use of equine anti-thymocyte globulin (ATGAM) for fulminant myocarditis secondary to nivolumab therapy. *Br J Cancer* 2017; 117: 921–924.
[10] Katsume Y, Isawa T, Toi Y, *et al.* Complete atrioventricular block associated with pembrolizumab-induced acute myocarditis: the need for close cardiac monitoring. *Intern Med* 2018; 57: 3157–3162.
[11] Szuchan C, Elson L, Alley E, *et al.* Checkpoint inhibitor-induced myocarditis and myasthenia gravis in a recurrent/metastatic thymic carcinoma patient: a case report. *Eur Heart J Case Rep* 2020; 4: 1–8.
[12] Luo YB, Tang W, Zeng Q, *et al.* The neuromusclar triad of immune checkpoint inhibitors: a case report of myositis, myocarditis, and myasthenia gravis overlap following toripalimab treatment. *Front Cardiovasc Med* 2021; 8: 714460.
[13] Jespersen MS, Stenor C, Moller AK. A case report of immune checkpoint inhibitor-related steroid-refractory myocarditis and myasthenia gravis-like myositis treated with abatacept and mycophenolate mofetil. *Eur Heart J Case Rep* 2021; 5: ytab342.
[14] Doms J, Prior JO, Peters S, Obeid M. Tocilizumab for refractory severe immune checkpoint inhibitor-associated

myocarditis. *Ann Oncol* 2020; 31: 1273–1275.
[15] Xing Q, Zhang ZW, Lin QH, *et al*. Myositis-myasthenia gravis overlap syndrome complicated with myasthenia crisis and myocarditis associated with anti-programmed cell death-1 (sintilimab) therapy for lung adenocarcinoma. *Ann Transl Med* 2020; 8: 250.
[16] Yanase T, Moritoki Y, Kondo H, *et al*. Myocarditis and myasthenia gravis by combined nivolumab and ipilimumab immunotherapy for renal cell carcinoma: a case report of successful management. *Urol Case Rep* 2021; 34: 101508.
[17] Kondo H, Kirigaya J, Matsuzawa Y, Hibi K. Two cases of immune checkpoint inhibitor-induced myocarditis with complete atrioventricular block. *Cureus* 2023; 15: e36446.
[18] Saad R, Ghaddar A, Zeenny RM. Pembrolizumab-induced myocarditis with complete atrioventricular block and concomitant myositis in a metastatic bladder cancer patient: a case report and review of the literature. *J Med Case Rep* 2024; 18: 107.
[19] Lorente-Ros A, Rajjoub-Al-Mahdi EA, Monteagudo Ruiz JM, *et al*. Checkpoint immunotherapy-induced myocarditis and encephalitis complicated with complete AV block. *JACC Case Rep* 2022; 4: 1032–1036.
[20] Berg DD, Vaduganathan M, Nohria A, *et al*. Immune-related fulminant myocarditis in a patient receiving ipilimumab therapy for relapsed chronic myelomonocytic leukaemia. *Eur J Heart Fail* 2017; 19: 682–685.
[21] Nasr F, El Rassy E, Maalouf G, *et al*. Severe ophthalmoplegia and myocarditis following the administration of pembrolizumab. *Eur J Cancer* 2018; 91: 171–173.
[22] Jain V, Mohebtash M, Rodrigo M, *et al*. Autoimmune myocarditis caused by immune checkpoint inhibitors treated with anti-thymocyte globulin. *J Immunother* 2018; 41: 332–335.
[23] Martinez-Calle N, Rodriguez-Otero P, Villar S, *et al*. Anti-PD1 associated fulminant myocarditis after a single pembrolizumab dose: the role of occult pre-existing autoimmunity. *Haematologica* 2018; 103: e318–e321.
[24] Saibil SD, Bonilla L, Majeed H, *et al*. Fatal myocarditis and rhabdomyositis in a patient with stage IV melanoma treated with combined ipilimumab and nivolumab. *Curr Oncol* 2019; 26: e418–e421.
[25] Charles J, Giovannini D, Terzi N, *et al*. Multi-organ failure induced by nivolumab in the context of allo-stem cell transplantation. *Exp Hematol Oncol* 2019; 8: 8.
[26] Hardy T, Yin M, Chavez JA, *et al*. Acute fatal myocarditis after a single dose of anti-PD-1 immunotherapy, autopsy findings: a case report. *Cardiovasc Pathol* 2020; 46: 107202.
[27] Giancaterino S, Abushamat F, Duran J, *et al*. Complete heart block and subsequent sudden cardiac death from immune checkpoint inhibitor-associated myocarditis. *HeartRhythm Case Rep* 2020; 6: 761–764.
[28] Portoles Hernandez A, Blanco Clemente M, Escribano Garcia D, *et al*. Checkpoint inhibitor-induced fulminant myocarditis, complete atrioventricular block and myasthenia gravis-a case report. *Cardiovasc Diagn Ther* 2021; 11: 1013–1019.
[29] Komatsu M, Hirai M, Kobayashi K, *et al*. A rare case of nivolumab-related myasthenia gravis and myocarditis in a patient with metastatic gastric cancer. *BMC Gastroenterol* 2021; 21: 333.
[30] Barham W, Guo R, Park SS, *et al*. Case report: simultaneous hyper-progression and fulminant myocarditis in a patient with advanced melanoma following treatment with immune checkpoint inhibitor therapy. *Front Immunol* 2020; 11: 561083.
[31] Wang F, Sun X, Qin S, *et al*. A retrospective study of immune checkpoint inhibitor-associated myocarditis in a single center in China. *Chin Clin Oncol* 2020; 9: 16.
[32] González-Ferrero T, Vargas-Osorio K, González-Juanatey JR. Fulminant myocarditis with myositis after treatment with immune checkpoint inhibitors. *Med Clin (Barc)* 2022; 158: 140–141.
[33] Vartanov A, Kalotra A, Varughese J, Gautam S, Kandel S, Hosmer W. Immunotherapy-associated complete heart block in a patient with NSCLC: a case report and literature review. *Respir Med Case Rep* 2021; 33: 101390.
[34] Naganuma K, Horita Y, Matsuo K, *et al*. An autopsy case of late-onset fulminant myocarditis induced by nivolumab in gastric cancer. *Intern Med* 2022; 61: 2867–2871.

Please cite this article as: Kalombo N, Xu L, Li K, Yin YJ, Tang H, Zhong W, Zhou Y. Immune checkpoint inhibitor-induced myocarditis with third-degree atrioventricular block: two case reports and literature review. *J Geriatr Cardiol* 2026; 23(9): 647–654. DOI: 10.26599/1671-5411.2026.09.004