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Complete Atrioventricular Block in Severe Guillain–Barré Syndrome: A Case Report

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Case Report

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Abstract

Background

Guillain–Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy that can progress to profound muscle weakness and respiratory failure. Cardiovascular autonomic dysfunction is another potentially life-threatening complication, manifesting as marked fluctuations in heart rate and blood pressure or severe arrhythmias. Among these manifestations, complete atrioventricular block (CAVB) is particularly severe and rarely reported in adults with GBS, potentially causing profound bradycardia and hemodynamic instability. Its unpredictable onset presents a diagnostic and management challenge, emphasizing the need for continuous cardiac monitoring, early recognition, and timely intervention.

Case presentation:

A 42-year-old woman with congenital right renal agenesis and end-stage renal disease was admitted with progressive limb weakness, followed by respiratory failure requiring invasive mechanical ventilation. Cerebrospinal fluid albuminocytologic dissociation and abnormal nerve conduction findings supported the diagnosis of GBS. Despite sinus rhythm on admission, bedside monitoring on Day 6 detected CAVB with bradycardia (43 beats/min) and hypotension (80/50 mmHg). An immediate 12-lead electrocardiogram (ECG) confirmed CAVB with a ventricular escape rhythm, prompting temporary transvenous pacing and subsequent hemodynamic stabilization. Serial high-sensitivity cardiac troponin I levels remained within the reference range, and a preceding echocardiogram showed preserved left ventricular ejection fraction (60%). She received plasma exchange and later intravenous immunoglobulin because severe weakness persisted. Subsequent cardiac monitoring documented no further CAVB, and the temporary pacemaker was removed on Day 19. Follow-up coronary computed tomography angiography showed atherosclerosis without significant stenosis. At three months, ECG confirmed normal atrioventricular conduction without reported recurrent bradycardia or syncope. The overall clinical course supported GBS-associated cardiovascular autonomic dysfunction as the primary explanation for CAVB.

Conclusions

This case describes severe GBS complicated by hemodynamically unstable CAVB, clinically attributed to GBS-associated cardiovascular autonomic dysfunction. Prompt recognition and temporary transvenous pacing were followed by hemodynamic stabilization, with subsequent pacemaker removal and normal atrioventricular conduction at three months. This case underscores the importance of continuous cardiac monitoring, careful etiological assessment, and individualized management of severe conduction disturbances during GBS.

Background

Guillain–Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by progressive limb weakness and reduced or absent tendon reflexes [1–3]. Beyond neuromuscular impairment, cardiovascular autonomic dysfunction may cause marked fluctuations in heart rate and blood pressure, with life-threatening rhythm disturbances in severe cases [4–7]. Such complications make sustained cardiac surveillance an important part of care in patients with severe GBS [8].

Complete atrioventricular block (CAVB) is a particularly severe but rarely reported manifestation in adults with GBS [9, 10]. Available cases differ in the onset of cardiac manifestations, the need for pacing, and subsequent conduction recovery. Distinguishing autonomic involvement from coexisting cardiac, metabolic, or medication-related factors may further complicate clinical assessment. Herein, we describe a patient who developed CAVB with hypotension after progression to respiratory failure, requiring temporary transvenous pacing and subsequently demonstrating recovery of atrioventricular conduction. This case emphasizes the importance of timely recognition, etiological assessment, and individualized management of severe conduction abnormalities in GBS.

Case presentation

A 42-year-old woman with congenital right renal agenesis and end-stage renal disease (ESRD) was admitted to the nephrology department on July 15, 2023, following approximately two weeks of progressive weakness in all four limbs. The day of admission was designated as Day 1. She had been receiving regular nephrology follow-up. Because of congenital hearing and speech impairments, her medical history was obtained primarily from family members. The clinical course and major interventions are summarized in Fig. 1.

Following admission, her limb weakness progressively worsened, accompanied by a weak cough. On Day 3, she developed respiratory failure requiring endotracheal intubation, invasive mechanical ventilation, and transfer to the intensive care unit (ICU). At ICU admission, muscle strength was graded 0–1 in all four limbs, although head and neck movements were preserved. Detailed sensory examination was limited by communication difficulties. The admission 12-lead electrocardiogram (ECG) showed sinus rhythm, while transthoracic echocardiography on Day 4 revealed a left ventricular ejection fraction (LVEF) of 60% with trace pericardial effusion and no other significant structural cardiac abnormalities reported. Cerebrospinal fluid analysis on Day 5 demonstrated albuminocytological dissociation, with a protein concentration of 72.5 mg/dL and a white blood cell count of 2 cells/μL. Nerve conduction studies revealed absent compound muscle action potentials in both common peroneal nerves, reduced motor conduction velocities in both tibial nerves, and abnormalities in sensory nerve responses. These findings, together with the rapidly progressive weakness and respiratory muscle involvement, supported the diagnosis of GBS. Nerve conduction study findings are presented in Supplementary Table S1 and laboratory findings in Supplementary Table S2 (Additional file 1).

On Day 6, bedside cardiac monitoring revealed CAVB, accompanied by marked bradycardia and hypotension. The heart rate decreased to 43 beats/min, with a blood pressure of 80/50 mmHg. An

immediate bedside 12-lead ECG confirmed CAVB with a ventricular escape rhythm. Given the associated hemodynamic instability, temporary transvenous cardiac pacing was initiated on the same day. A pacing lead was advanced through the right internal jugular vein to the right ventricular apex, with the pacing rate set at 80 beats/min. Appropriate sensing and capture were achieved, followed by hemodynamic stabilization. The patient had been receiving moxifloxacin and azithromycin since Day 4. Given concerns about potential cardiac adverse effects, both agents were discontinued on Day 6. Ventricular tachycardia (VT) was also documented on the same day. Serial ECGs obtained at admission, during CAVB and VT, and one day after temporary pacemaker removal (Day 20) are presented in Fig. 2.

Laboratory investigations around the cardiac event showed serum potassium levels of 3.5–3.6 mmol/L, within the institutional reference range of 3.5–5.1 mmol/L. Arterial blood gas analysis on Day 6 revealed a pH of 7.455, $PaCO_2$ of 28.6 mmHg, PaO_2 of 154 mmHg, ionized calcium of 1.04 mmol/L, and lactate of 2.9 mmol/L. The ionized calcium level was below the institutional reference range of 1.19–1.25 mmol/L. High-sensitivity cardiac troponin I (hs-cTnI) levels did not exceed the upper reference limit of 0.03 ng/mL on Days 6, 7, and 10. Creatine kinase-MB (CK-MB) was 15 U/L on Day 10, within the reference range of 0–16 U/L. The laboratory findings and changes in cardiac biomarkers are summarized in Supplementary Table S2 (Additional file 1) and Fig. 1B, C.

Given the severity of GBS, with profound limb weakness and respiratory muscle involvement requiring mechanical ventilation, the patient underwent three sessions of plasma exchange (PLEX) on Days 7–9. However, neurological improvement remained limited, with persistent severe muscle weakness. Intravenous immunoglobulin (IVIg) was therefore administered at 20 g/day on Days 18–22. Owing to prolonged ventilatory requirements and persistent neuromuscular weakness, tracheostomy was performed on Day 18.

During the subsequent hospital course, no further episodes of CAVB were documented. Holter monitoring on Days 17–18 showed sinus tachycardia, ventricular pacing, premature ventricular contractions, and ventricular escape beats, without recorded recurrence of CAVB. With stable hemodynamics, heart rates remaining above 75 beats/min, and no further high-grade atrioventricular block detected on continuous monitoring, the temporary pacemaker was removed on Day 19.

The patient's limb strength gradually improved, and she was discharged on Day 35. At discharge, she was conscious and remained tracheostomized, receiving supplemental oxygen through a Venturi device. Upper-limb strength improved to grade 3, whereas lower-limb strength remained severely impaired at grade 1.

On Day 68, 33 days after discharge, coronary computed tomography angiography (CTA) performed at our hospital demonstrated coronary atherosclerosis without significant luminal stenosis. The coronary artery calcium score was 0, although calcification was noted in the aortic valve region. At the three-month follow-up, further improvement in motor function was observed. A 12-lead ECG showed normal atrioventricular conduction, with no reported recurrence of bradycardia or syncope. Representative coronary CTA images are presented in Supplementary Fig. S1 (Additional file 1).

The CARE reporting checklist is provided in Additional file 2.

Discussion

This case highlights CAVB as a life-threatening cardiovascular complication of severe GBS. Despite sinus rhythm on admission, CAVB with hypotension developed approximately 18 days after the onset of limb weakness, after respiratory failure had necessitated mechanical ventilation. Temporary transvenous pacing was followed by hemodynamic stabilization, and normal atrioventricular conduction was documented at the three-month follow-up. Based on the neurological course and evaluation of alternative etiologies, CAVB was clinically attributed to GBS-associated cardiovascular autonomic dysfunction.

Published reports of CAVB in adults with GBS remain limited, particularly regarding its temporal relationship with neurological deterioration. Wani et al. described CAVB with Stokes–Adams attacks requiring demand pacing before limb weakness became apparent 48 hours later [9]. Similarly, Kuruppuarachchi et al. reported CAVB preceding limb weakness, with sinus rhythm restored within 24 hours of IVIg initiation without cardiac pacing [10]. In contrast, electrocardiographically confirmed CAVB accompanied by hypotension developed in our patient after profound weakness had progressed to respiratory failure. These differences illustrate that severe atrioventricular conduction abnormalities may occur before recognizable neurological symptoms or later during advanced GBS, even when the initial ECG is normal.

Determining the need for pacing and when it can be safely withdrawn presents another clinical challenge. Our patient underwent temporary transvenous pacing for CAVB with hypotension; subsequent ECG showed sinus rhythm, and Holter monitoring predominantly documented sinus tachycardia without further recorded CAVB. With sustained clinical and hemodynamic stability, the temporary pacemaker was removed on Day 19, and normal atrioventricular conduction was confirmed at three months. Earlier reports described demand pacing for GBS-related profound bradycardia, idioventricular rhythms, or occasional asystole [11, 12]. Kuroiwa et al. reported temporary pacemaker removal after three weeks without recurrent bradycardia or asystole [13], whereas Patel et al. described permanent pacemaker implantation for persistent pacing requirements, with pacing still needed at three months [14]. Other reports document recurrent asystole or prolonged pauses requiring vigilance and, in selected cases, pacing [15, 16]. Although these cases did not all involve confirmed CAVB, their differing outcomes support prompt pacing when hemodynamically indicated and reassessment of intrinsic rhythm during recovery rather than assuming a fixed duration of pacing or inevitable permanent implantation [17, 18]. Selected reports of GBS-associated CAVB and other severe bradyarrhythmias are summarized in Table 1, with comparisons of their timing relative to weakness, pacing management, and outcomes.

Table 1
Selected reports of complete atrioventricular block and pacing-requiring bradyarrhythmias in Guillain–Barré syndrome

Study	Cardiac event	Timing relative to weakness	Pacing	Outcome
Wani, 1989 [9]	CAVB; Stokes–Adams attacks	48 h before limb weakness	Demand pacemaker	Long-term conduction outcome not specified
Kuruppuarachchi, 2018 [10]	CAVB	Before limb weakness	No cardiac pacing; IVIg	Sinus rhythm within 24 h of IVIg initiation
Tonelli, 2007 [16]	Sinus pauses; paroxysmal AV block	During severe, ventilated GBS	Temporary → permanent pacemaker	Rhythm variability decreased by week 6
Patel, 2009 [14]	Recurrent bradycardia and asystole	During severe GBS	Temporary → permanent pacemaker	Pacing still required at 3 months
Kuroiwa, 2023 [13]	Recurrent asystole during airway clearance	During ventilated GBS	Temporary pacemaker	Removed after 3 weeks; no further bradyarrhythmia reported
Present case	**CAVB; hypotension**	**18 days after weakness onset**	**Temporary transvenous pacing**	**Removed on Day 19; normal AV conduction at 3 months**

Note. Only Wani, Kuruppuarachchi, and the present case were classified as confirmed CAVB. Other reports were included for comparison of pacing management, not as CAVB cases. Abbreviations: AV, atrioventricular; CAVB, complete atrioventricular block; GBS, Guillain–Barré syndrome; IVIg, intravenous immunoglobulin.

Cardiovascular autonomic dysfunction in GBS can involve both sympathetic and parasympathetic regulation [19, 20]. Autonomic abnormalities may persist after the acute phase of GBS, including in patients without overt autonomic symptoms [21]. In the present case, autonomic involvement provides a plausible explanation for CAVB. Nevertheless, other causes warranted consideration, particularly in the setting of ESRD. Serial hs-cTnI levels were within the reference range, CK-MB was normal on Day 10, and echocardiography before CAVB showed preserved LVEF (60%). Follow-up coronary CTA revealed no significant stenosis. These findings offered little support for overt myocardial injury or obstructive coronary disease, although myocarditis and transient ischemia could not be entirely excluded [22, 23]. Chronic kidney disease may itself impair cardiovascular autonomic regulation, but its relative contribution in this patient remains uncertain [24, 25]. Serum potassium was within the reference range, arguing against hyperkalemia. Ionized calcium was mildly reduced (1.04 mmol/L) in the presence of alkalemia and hypocapnia; respiratory alkalosis can lower ionized calcium by increasing protein binding

[26]. Hypocalcemia can alter cardiac repolarization [27], but its contribution to CAVB in this patient remains uncertain.

Recent exposure to moxifloxacin and azithromycin also required consideration. Altin et al. reported torsades de pointes on the fourth day of moxifloxacin treatment, with normalization of the corrected QT interval (QTc) seven days after withdrawal [28]; Koide et al. described QTc prolongation after six days of moxifloxacin, resolving six days after discontinuation [29]. Abdelazeem et al. reported symptomatic bradycardia approximately two days after azithromycin initiation, with symptom improvement following withdrawal [30]. CAVB has also been described after massive azithromycin overdose in an infant, a substantially different exposure from therapeutic adult use [31]. Our patient developed CAVB approximately two days after both antibiotics were started; both were stopped on Day 6, when VT was also documented. Although a medication-related contribution to electrical instability cannot be excluded, these reported arrhythmias and exposure settings differ from the CAVB observed here, and the available evidence does not establish antibiotic-induced CAVB.

Despite stabilization of the patient's cardiovascular condition, profound neuromuscular weakness and ventilator dependence persisted, complicating subsequent treatment decisions. Three sessions of PLEX were performed on Days 7–9, followed by IVIg on Days 18–22 because neurological improvement remained limited. Algahtani et al. described a similarly ventilator-dependent patient who received IVIg several weeks after PLEX, although more substantial recovery occurred only after additional IVIg courses [32]. In our patient, upper-limb strength improved from grade 0–1 in the acute phase to grade 3 at discharge, while lower-limb strength remained at grade 1. Both PLEX and IVIg are established treatments for GBS [33, 34], but a randomized trial found no significant added benefit from PLEX immediately followed by IVIg [35]. The European Academy of Neurology/Peripheral Nerve Society guideline also advises against routinely switching to the alternative therapy after an inadequate initial response because trial evidence is lacking [1]. In this context, the later IVIg course was undertaken as an individualized attempt to promote further neurological recovery in the setting of persistent severe neuromuscular deficits, rather than as a routine immediate sequential regimen.

Several limitations should be acknowledged. Formal cardiovascular autonomic testing was not performed, and available monitoring records could not establish the exact duration of CAVB or when intrinsic atrioventricular conduction recovered. Coexisting renal disease, electrolyte abnormalities, and recent antibiotic exposure also remain potential contributors, despite the clinical assessment. Furthermore, the absence of standardized serial muscle strength measurements around IVIg initiation precludes attribution of neurological improvement to delayed sequential immunotherapy. Nevertheless, the documented cardiac course, pacing management, and follow-up add clinically relevant longitudinal information to the limited reports of CAVB in severe GBS.

Conclusions

CAVB is a rare but potentially life-threatening complication of severe GBS that may occur despite an initially normal ECG. Prompt recognition, temporary transvenous pacing when hemodynamically indicated, and continued rhythm monitoring can support acute stabilization and guide subsequent pacing decisions. This case underscores the value of integrating neurological progression, cardiac findings, and alternative etiologies to inform diagnosis and individualized management.

Abbreviations

AV Atrioventricular

CAVB Complete atrioventricular block

CK-MB Creatine kinase-MB

CRRT Continuous renal replacement therapy

CTA Computed tomography angiography

ECG Electrocardiogram

ESRD End-stage renal disease

GBS Guillain–Barré syndrome

hs-cTnI High-sensitivity cardiac troponin I

ICU Intensive care unit

IVIg Intravenous immunoglobulin

LVEF Left ventricular ejection fraction

PLEX Plasma exchange

QTc Corrected QT interval

VT Ventricular tachycardia

Declarations

Ethics approval and consent to participate

The study was conducted under ethical principles (Declaration of Helsinki). Written consent was obtained from each patient or her authorized representatives.

Consent for publication

Written informed consent was obtained from the patient for all the manuscripts that included images and details.

Competing interests

The authors declare no competing interests.

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Author Contribution

Q.C.X., W.X. and J.J.C. conceptualized the study. W.X. and J.J.C. collected and curated the clinical data. W.X. wrote the original manuscript. Q.C.X. reviewed and edited the manuscript. Q.C.X., W.H.L. and L.J. supervised the work. Q.C.X. and Y.Y.C. acquired funding. All authors read and approved the final manuscript.

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Not applicable.

Data availability

No datasets were generated or analysed during the current study.

References

1. van Doorn PA, Van den Bergh PYK, Hadden RDM, et al. European Academy of Neurology/Peripheral Nerve Society Guideline on diagnosis and treatment of Guillain-Barré syndrome. Eur J Neurol. 2023;30(12):3646–74.
2. Leonhard SE, Mandarakas MR, Gondim FAA, et al. Diagnosis and management of Guillain-Barré syndrome in ten steps. Nat Rev Neurol. 2019;15(11):671–83.
3. Bellanti R, Rinaldi S. Guillain-Barré syndrome: a comprehensive review. Eur J Neurol. 2024;31(8):e16365.

4. Weiss N, Marois C, Le Guennec L, Rohaut B, Demeret S. Critical insights for intensivists on Guillain-Barré syndrome. Ann Intensive Care. 2025;15(1):67.
5. Pagaling GT, Dichoso LPC, Reyes NGD, Prado MB Jr. Association of dysautonomia and different risk factors in patients with Guillain-Barré syndrome in a tertiary hospital in the Philippines. BMC Neurol. 2025;25(1):29.
6. Bazán-Rodríguez L, Ruiz-Avalos JA, de Saráchaga AJ, Martinez-Jimenez E, López-Hernández JC. Vargas-Cañas S. Dysautonomia and related outcomes in Guillain-Barre syndrome. Auton Neurosci. 2023;245:103070.
7. Greenland P, Griggs RC. Arrhythmic complications in the Guillain-Barré syndrome. Arch Intern Med. 1980;140(8):1053–5.
8. Flachenecker P, Lem K, Müllges W, Reiners K. Detection of serious bradyarrhythmias in Guillain-Barré syndrome: sensitivity and specificity of the 24-hour heart rate power spectrum. Clin Auton Res. 2000;10(4):185–91.
9. Wani BA, Misra M, Shah M, Mufti S. Acute inflammatory demyelinating polyradiculoneuropathy presenting as complete heart block and Stoke-Adams attacks. Postgrad Med J. 1989;65(760):103–4.
10. Kuruppuarachchi AN, Sundaresan KT, Thivakaran T. Rare presentation of Guillain-Barre syndrome with complete heart block and neuropathic chest pain mimicking acute coronary syndrome: a case report. Ceylon Med J. 2018;63(3):149–50.
11. Emmons PR, Blume WT, DuShane JW. Cardiac monitoring and demand pacemaker in Guillain-Barré syndrome. Arch Neurol. 1975;32(1):59–61.
12. Maytal J, Eviatar L, Brunson SC, Gootman N. Use of demand pacemaker in children with Guillain-Barré syndrome and cardiac arrhythmias. Pediatr Neurol. 1989;5(5):303–5.
13. Kuroiwa R, Tateishi Y, Oshima T, et al. Cardiovascular autonomic dysfunction induced by mechanical insufflation-exsufflation in Guillain-Barré syndrome. Respirol Case Rep. 2023;11(5):e01135.
14. Patel MB, Goyal SK, Punnam SR, Pandya K, Khetarpal V, Thakur RK. Guillain-Barré Syndrome with asystole requiring permanent pacemaker: a case report. J Med Case Rep. 2009;3:5.
15. Abbas Z, Sardar Z. Use of pacemaker in GBS dysautonomia. BMJ Case Rep. 2021;14(10):e242464.
16. Tonelli A, Khasnis A, Abela GS. Peaked T-waves and sinus arrhythmia before prolonged sinus pauses and atrioventricular block in Guillain-Barre syndrome. Indian Pacing Electrophysiol J. 2007;7(4):249–52.
17. Glikson M, Nielsen JC, Kronborg MB, et al. 2021 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy. Eur Heart J. 2021;42(35):3427–520.
18. Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay. Circulation. 2019;140(8):e382–482.

19. Chakraborty T, Kramer CL, Wijdicks EFM, Rabinstein AA. Dysautonomia in Guillain-Barré syndrome: prevalence, clinical spectrum, and outcomes. Neurocrit Care. 2020;32(1):113–20.
20. Majumder P, Tripathi M, Srivastava AK, et al. Characterization of autonomic involvement in various subtypes of Guillain-Barré syndrome and association with electrophysiological parameters. Muscle Nerve. 2025;72(6):1235–41.
21. Theologou R, Barkoula T, Ioannou C, et al. Autonomic dysfunction after Guillain-Barré syndrome: evidence from comprehensive autonomic testing. J Clin Med. 2026;15(15):5963.
22. Schulz-Menger J, Collini V, Gröschel J, et al. 2025 ESC Guidelines for the management of myocarditis and pericarditis. Eur Heart J. 2025;46(40):3952–4041.
23. Hokimoto S, Kaikita K, Yasuda S, et al. JCS/CVIT/JCC 2023 guideline focused update on diagnosis and treatment of vasospastic angina (coronary spastic angina) and coronary microvascular dysfunction. Circ J. 2023;87(6):879–936.
24. Celik A, Melek M, Yuksel S, Onrat E, Avsar A. Cardiac autonomic dysfunction in hemodialysis patients: the value of heart rate turbulence. Hemodial Int. 2011;15(2):193–9.
25. Dursun B, Demircioglu F, Varan HI, et al. Effects of different dialysis modalities on cardiac autonomic dysfunctions in end-stage renal disease patients: one year prospective study. Ren Fail. 2004;26(1):35–8.
26. Moe SM. Disorders involving calcium, phosphorus, and magnesium. Prim Care. 2008;35(2):215–37.
27. Gardner JD, Calkins JB Jr, Garrison GE. ECG diagnosis: the effect of ionized serum calcium levels on electrocardiogram. Perm J. 2014;18(1):e119–20.
28. Altin T, Ozcan O, Turhan S, et al. Torsade de pointes associated with moxifloxacin: a rare but potentially fatal adverse event. Can J Cardiol. 2007;23(11):907–8.
29. Koide T, Shiba M, Tanaka K, et al. Severe QT interval prolongation associated with moxifloxacin: a case report. Cases J. 2008;1(1):409.
30. Abdelazeem B, Hollander RM, Ayad S, Gergis K, Gismalla ME. Azithromycin-induced bradycardia. Cureus. 2021;13(8):e16995.
31. Tilelli JA, Smith KM, Pettignano R. Life-threatening bradyarrhythmia after massive azithromycin overdose. Pharmacotherapy. 2006;26(1):147–50.
32. Algahtani H, Shirah B, Alrefaei K, Albassam M, Abdelghaffar N. Are repeated cycles of intravenous immunoglobulin justified in patients with poorly responsive Guillain-Barré. syndrome? Neurohospitalist. 2020;10(3):224–8.
33. Chevret S, Hughes RAC, Annane D. Plasma exchange for Guillain-Barré syndrome. Cochrane Database Syst Rev. 2017;2017(2):CD001798.
34. Hughes RAC, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain-Barré syndrome. Cochrane Database Syst Rev. 2014;2014(9):CD002063.
35. Plasma Exchange/Sandoglobulin Guillain-Barré Syndrome Trial Group. Randomised trial of plasma exchange, intravenous immunoglobulin, and combined treatments in Guillain-Barré syndrome.

Lancet. 1997;349(9047):225–30.

Figures

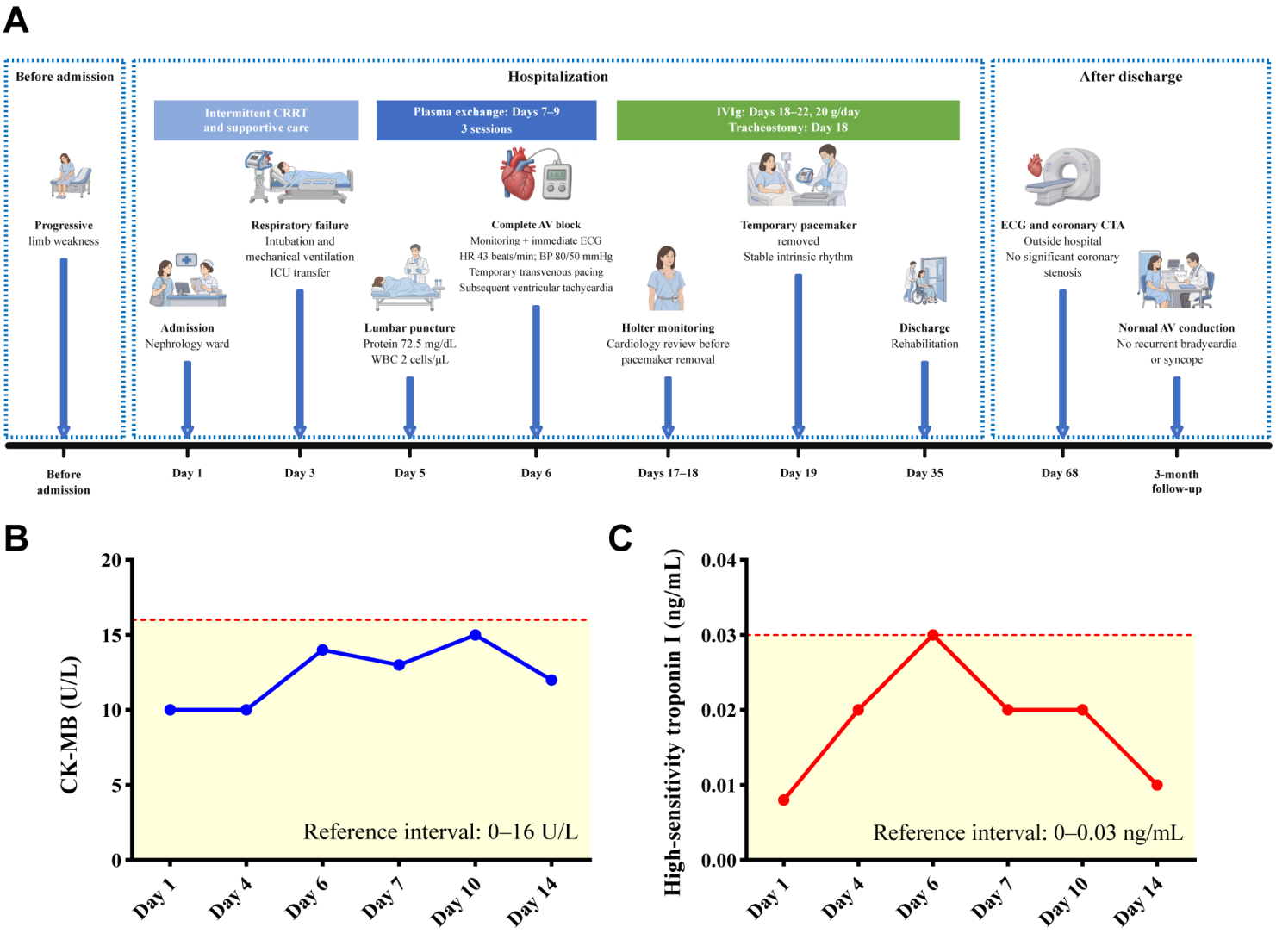


Figure 1

Clinical course and serial cardiac biomarkers. (A) Clinical course and major interventions. (B) CK-MB activity. (C) High-sensitivity cardiac troponin I. Dashed lines in B and C indicate upper reference limits of 16 U/L and 0.03 ng/mL. Day 1 denotes admission on 15 July 2023. Time intervals are not drawn to scale. CRRT, continuous renal replacement therapy; IVIg, intravenous immunoglobulin; AV, atrioventricular; ECG, electrocardiogram; CTA, computed tomography angiography.

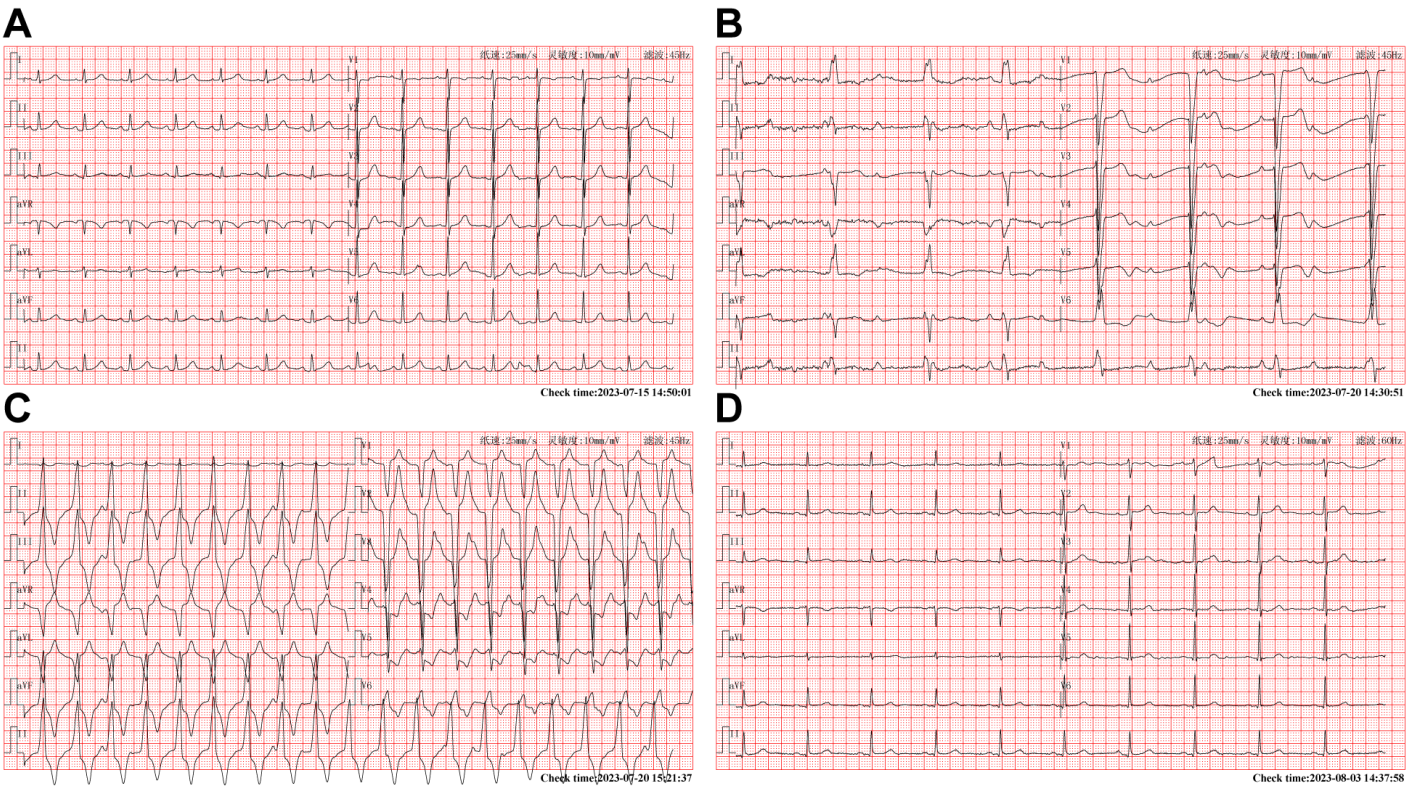


Figure 2

Serial 12-lead electrocardiograms. (A) Admission, 15 July 2023. (B) Complete atrioventricular block, 20 July 2023. (C) Ventricular tachycardia on the same day. (D) Repeat ECG on 3 August 2023 (Day 20), one day after temporary pacemaker removal.

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