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Recurrent syncope with seizure-like features: the role of emergency monitoring in revealing intermittent AV block

Huong Thi Thanh Le[1*], Ninh Xuan Nguyen[1], Ngoc Tien Pham[1], Quoc Viet Tran[1], Hang Ngoc Thuy Tran[1] and Nhat Hong Tran[2]

Abstract

Background Recurrent syncope with seizure-like stiffening may mimic epilepsy or neurological disorders, and when routine tests are repeatedly normal, intermittent arrhythmia can be easily overlooked.

Case presentation We report a case of a 65-year-old man with hypertension, hyperuricaemia, dyslipidaemia, and asthma who experienced recurrent syncopal episodes over one month. He presented several times to emergency departments and tertiary hospitals. Neurological and cardiovascular investigations—including brain MRI, echocardiography, coronary angiography, laboratory tests, and a previous Holter ECG—were consistently unremarkable. The initial diagnosis was hypertensive crisis based on marked post-event blood pressure surges, which was later recognized as a secondary phenomena rather than the primary cause of syncope. On the index admission, continuous emergency monitoring captured a sinus arrest of ~ 17 s with absent arterial pulse waveform, followed by bradyarrhythmia. Post-event blood pressure spiked to 220/110 mmHg. A repeat Holter ECG confirmed intermittent Mobitz II and complete AV block with asystole up to 18.6 s. EEG, performed during this admission to exclude epilepsy, was normal. A dual-chamber permanent pacemaker was implanted with complete resolution of symptoms (Shen et al, Circulation 136(5):e60-e122, 2017; Kusumoto et al, Circulation 140(8): e382-e482, 2019; Brignole et al, Eur Heart J 39(21):1883-1948, 2018).

Conclusion This case demonstrates how intermittent AV block may masquerade as seizure or hypertensive crisis, underlining the critical role of emergency department monitoring and prolonged ECG recording in recurrent unexplained syncope.

Keywords Recurrent syncope, Atrioventricular block, Intermittent complete heart block, Adams–Stokes syndrome, Emergency monitoring, Seizure-like activity, Post-syncopal hypertension

*Correspondence:
Huong Thi Thanh Le
v.huongltt14@vinmec.com; ltthuong115@gmail.com
[1]Department of Emergency Medicine, Vinmec International Hospital, Ho Chi Minh City, Vietnam
[2]Department of Cardiology, Vinmec International Hospital, Ho Chi Minh City, Vietnam

Introduction

Syncope is common in older adults but poses diagnostic challenges when episodes overlap with seizure-like activity or post-event hypertension. Intermittent AV block often escapes detection with standard tests, delaying treatment [1–3]. Our case illustrates how repeated negative work-ups across multiple hospitals culminated in diagnosis only after continuous emergency monitoring and prolonged ECG recording.

Timeline A chronological summary of the patient's symptoms, investigations, and interventions is provided in Table 1.

Case presentation

A 65-year-old man with hypertension, hyperuricemia, dyslipidemia, and asthma presented with recurrent episodes of syncope for more than one month. Each episode was characterized by sudden transient loss of consciousness (5–20 s), urinary incontinence, and brief limb stiffening, unlike the tonic–clonic movements characteristic of a generalized seizure. He regained consciousness immediately without confusion, though often appeared fatigued and anxious. Notably, each event was followed by marked blood pressure elevation (up to 220/110 mmHg).

Prior admissions

- **Feb 1, 2025**: First emergency department (ED) visit. Brain MRI: no acute lesions, only Fazekas 1 white matter changes. CBC, electrolytes, cardiac enzymes within normal limits. A Holter ECG performed at another hospital was unremarkable. Diagnosis: hypertensive crisis → treated with antihypertensives, but syncope recurred.
- **Feb 6, 2025**: Second ED visit. Similar episode, with mild hyponatremia and normal thyroid and troponin levels. Again diagnosed as hypertensive crisis and treated with antihypertensives.
- **6–15 Feb 2025**: Further evaluations at two tertiary hospitals, including echocardiography, coronary angiography, laboratory tests, and Holter ECG monitoring, all unremarkable. The patient continued antihypertensive therapy but remained symptomatic.

Index admission (Feb 15, 2025)

- On arrival, the patient was alert and oriented. BP 172/76 mmHg (falling to 159/94 after rest), HR 70 bpm, SpO_2 98%. No focal neurological deficits. Normal ECG (Fig. 1).
- During observation, he developed syncope with limb stiffening but no tonic–clonic jerks. He recovered immediately, BP rose markedly.
- At 14:26, another episode occurred. Continuous monitoring captured sinus arrest (~17 s) with absent arterial waveform, followed by bradyarrhythmia (Fig. 2). Post-event BP spiked to 220/110 mmHg.
- Overall, on the day of index admission, the patient experienced at least six syncopal episodes occurring at home, in the ED and during the first night of hospitalization. No specific triggering factors were identified. Following initiation of temporary pacing, no further syncopal episodes were observed.

Table 1 Clinical timeline of events, investigations, diagnoses, and interventions

Date	Event / Setting	Findings	Diagnosis and Treatment / Intervention
01 Feb 2025	First ED admission	CBC, electrolytes, cardiac enzymes normal; Brain MRI: Fazekas 1 small vessel changes	**Hypertensive crisis** → Antihypertensive medication; no effect on recurrent syncope
06 Feb 2025	Second ED admission	Mild hyponatremia; normal thyroid; troponin negative	**Hypertensive crisis** → Antihypertensive medication; no control of post-event BP spikes
06–15 Feb 2025	Outpatient visits at 2 tertiary hospitals	Multiple evaluations: Cardiology consult, echocardiography, Holter ECG, blood tests — all unremarkable	**Hypertensive crisis** → Continued antihypertensives; no arrhythmia detected
15 Feb 2025	Index ED admission	Syncope with transient limb stiffening; ED monitoring: sinus arrest ~ 17s → BP spike 220/110	**Arrhythmic syncope suspected** → Hospital admission for monitoring
16 Feb 2025	Inpatient cardiology (Holter ECG)	Longest asystole 18.6s; multiple 6–10 s pauses; intermittent Mobitz II AV block; temporary pacing activated	**High-grade AV block** → Temporary pacing initiated
19 Feb 2025	Intervention	Pacemaker implantation (dual-chamber, transvenous)	**Definitive treatment**
24 Feb 2025	Discharge and follow-up	No recurrence of syncope or hypertensive crises; normal daily activities resumed	**Stable outcome**

a. Brain MRI showed chronic small vessel disease (Fazekas 1), no acute lesion

b. Labs excluded metabolic and endocrine causes

c. Despite extensive tests (echo, Holter ECG, labs), no abnormality found before index admission

d. ED monitoring captured sinus arrest and BP surge

e. Holter ECG confirmed prolonged asystole and Mobitz II AV block

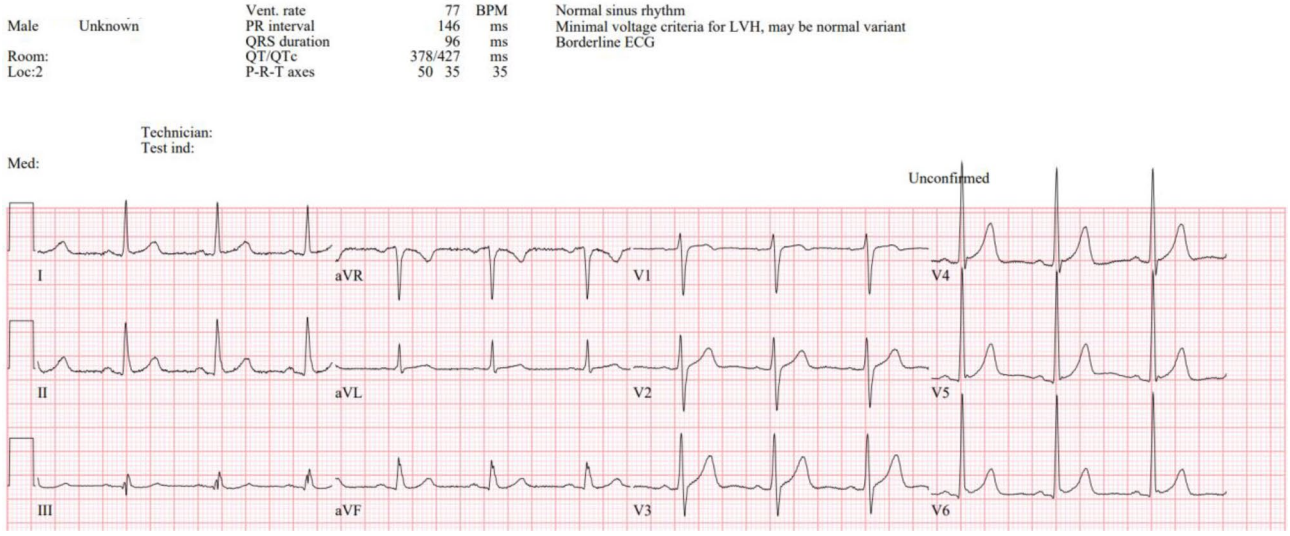

Fig. 1 Electrocardiogram at hospital admission showing normal sinus rhythm

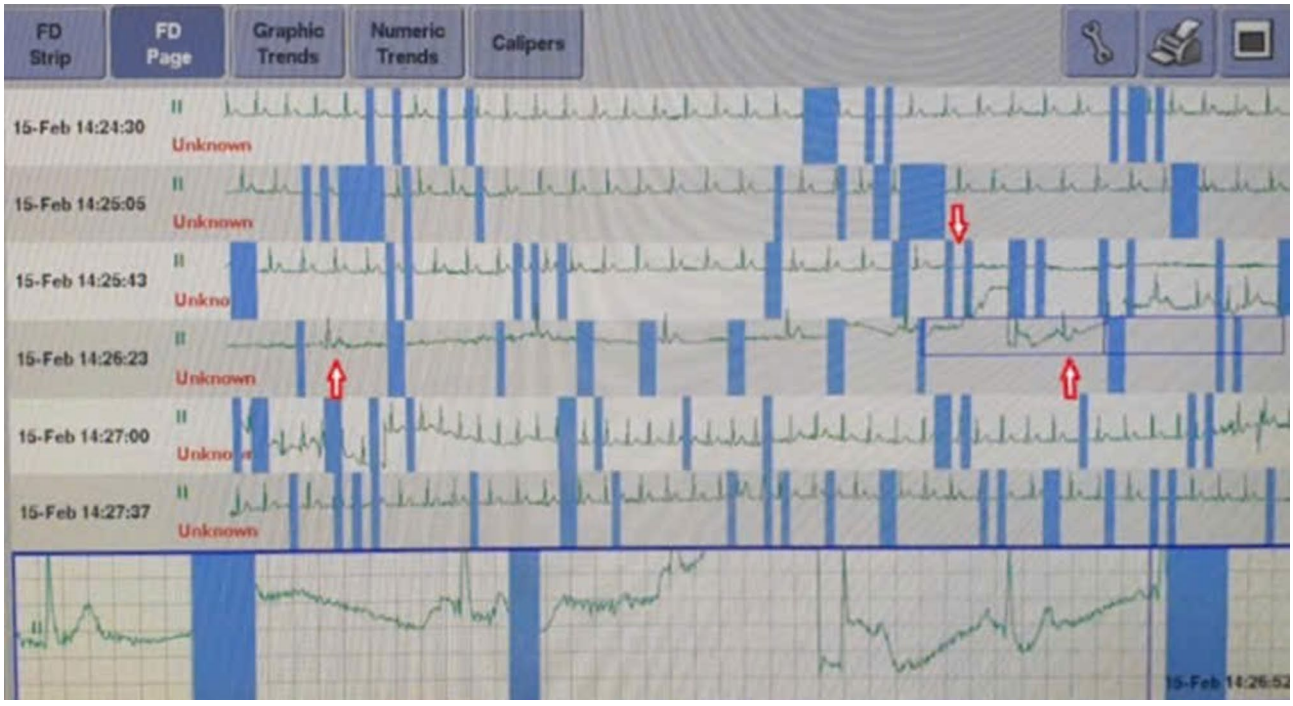

Fig. 2 Emergency department ECG monitoring during a syncopal episode. Continuous ECG monitoring captured a prolonged asystolic event. Red arrows indicate the onset of asystole, the end of the asystolic pause with reappearance of slow, irregular junctional escape beats, and subsequent restoration of normal sinus rhythm

Investigations

- Repeat Holter ECG (with temporary pacing) (Fig. 3): Predominant sinus rhythm, mean HR 80 bpm, SVT at 120 bpm. Longest asystole 18.6 s, multiple pauses 6–10 s, Mobitz II and intermittent complete AV block. Temporary pacing active 03:54–04:01.
- EEG: Normal during wakefulness and stage I–II sleep.
- Brain MRI: No acute infarction or hemorrhage.

Differential diagnosis

- Epileptic seizure.
- Transient ischaemic attack.
- Vasovagal syncope / orthostatic hypotension.
- Sick sinus syndrome.
- Intermittent high-grade AV block (confirmed).

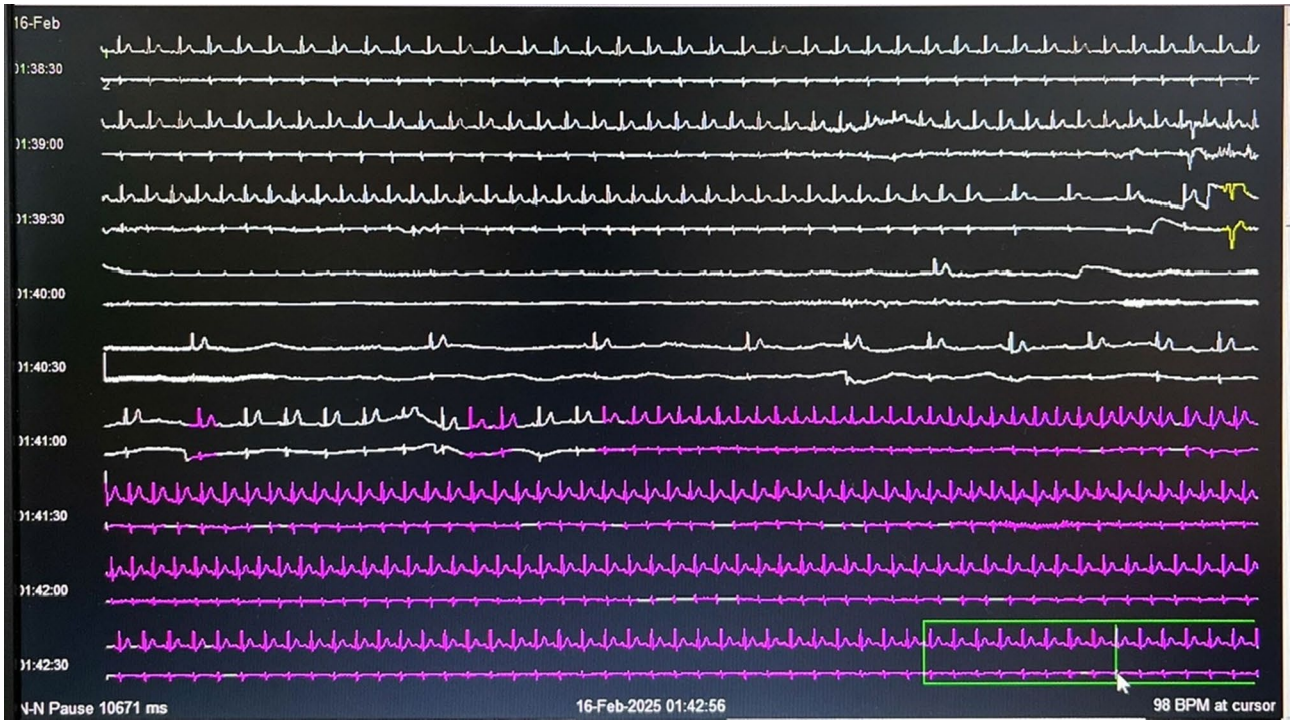

Fig. 3 Holter ECG recorded during hospitalization (16 February 2025). Holter ECG demonstrates intermittent high-grade atrioventricular block with prolonged asystole. Red arrows indicate the onset and termination of the arrhythmic episode with return to sinus rhythm. Blue arrows denote the beginning and end of the asystolic pause, followed by slow, irregular junctional escape beats

- Hypertensive crisis was initially considered due to post-event surges, but later recognized as a misinterpretation of post-syncopal hypertension rather than the true cause of syncope.

Treatment and outcome

- Initially supported with a temporary transvenous pacemaker.
- On Feb 19, 2025, a permanent dual-chamber pacemaker was implanted.
- Discharged on Feb 24, 2025 in stable condition, symptom-free at follow-up.

Outcome and follow-up

After pacemaker implantation, the patient remained symptom-free, with no further syncope or hypertensive crises. At follow-up, he returned to normal daily activities.

Discussion

Bradyarrhythmia and Adams–Stokes syndrome

Analysis of the Holter ECG demonstrated stable PP intervals preceding the onset of atrioventricular block, followed by variability in QRS timing and subsequent sinus arrest. This electrophysiological pattern supports an intrinsic atrioventricular conduction abnormality rather than a vagally mediated mechanism, consistent with the patient's abrupt clinical presentation and favorable response to permanent pacemaker implantation [4, 5].

Intermittent high-grade atrioventricular block with prolonged pauses leads to transient cerebral hypoperfusion, the underlying mechanism of Adams–Stokes syndrome. Classical features include abrupt syncope, brief tonic or seizure-like movements, urinary incontinence, and rapid recovery once cardiac rhythm is restored. The patient's documented 18.6-second asystolic pause with limb stiffening and urinary incontinence is characteristic of this syndrome [6, 7].

Diagnostic challenges and role of emergency monitoring

Holter ECG is considered the gold standard for detecting intermittent arrhythmias, but its diagnostic yield depends on the occurrence of arrhythmic events during the recording period [8–10]. In this patient, a previous 24-hour Holter ECG was nondiagnostic, as no syncopal episodes occurred during monitoring.

In contrast, continuous ECG monitoring during the index emergency department admission captured a prolonged sinus arrest with absent arterial pulsations, providing the first definitive diagnostic clue. Subsequent Holter ECG confirmed intermittent Mobitz II and complete atrioventricular block.

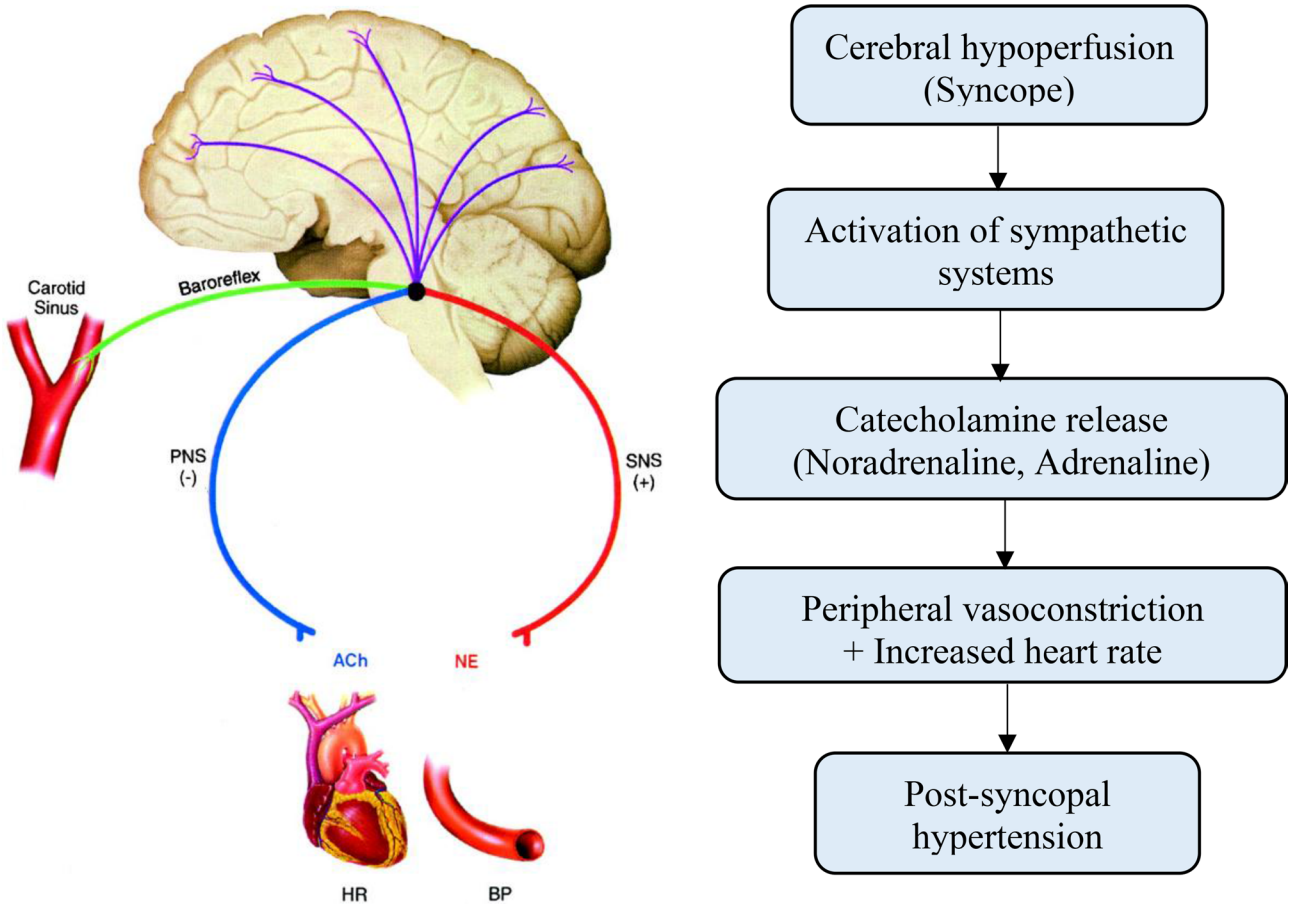

Fig. 4 Mechanism of post-syncopal hypertension. Cerebral hypoperfusion during syncope triggers sympathetic activation and catecholamine release, leading to peripheral vasoconstriction, tachycardia, and transient post-syncopal hypertension

In patients with recurrent unexplained syncope in whom standard investigations and short-term Holter monitoring are nondiagnostic, implantable loop recorders (ILRs) are recommended to improve diagnostic yield [11, 12]. ILRs enable long-term rhythm surveillance and significantly increase the detection of intermittent bradyarrhythmias and atrioventricular block, particularly when syncopal events are infrequent or unpredictable.

However, access to ILR implantation may be limited in some healthcare settings due to availability and cost. In such circumstances, continuous ECG monitoring during emergency department observation represents an immediate, low-cost, and widely accessible diagnostic opportunity, especially when patients present during periods of frequent recurrent syncope, as illustrated in the present case.

Mechanism of post-syncopal hypertension: (Fig. 4)

Each syncopal episode was followed by marked hypertension, initially misinterpreted as hypertensive crisis. These transient surges most likely reflected sympathetic activation and catecholamine release triggered by cerebral hypoperfusion, rather than a primary hypertensive emergency [13–17]. Distinguishing these entities is essential to avoid inappropriate management.

Management and guideline alignment

ESC (2018) and ACC/AHA/HRS (2017, 2023) guidelines recommend permanent pacing as Class I indication for symptomatic high-grade or complete AV block. Our patient's findings warranted pacemaker implantation, which resolved symptoms [1–3].

Novelty and clinical implication

This case underscores the importance of sequential diagnostic reasoning: recognizing arrhythmia consistent with Adams–Stokes syndrome, avoiding misdiagnosis as epilepsy [18, 19] or hypertensive crisis, and emphasizing the unique diagnostic opportunity provided by ED monitoring.

Learning points

- Recurrent syncope with seizure-like features and normal neurological investigations should prompt strong suspicion of an arrhythmic cause.
- Holter ECG is the gold standard for detecting intermittent AV block, but a negative result does not exclude disease if no event occurs during recording.
- Emergency department monitoring provides a critical diagnostic window, as patients are at higher risk of recurrent syncope during acute presentations.
- Continuous monitoring of ECG, blood pressure, and consciousness is essential in patients presenting with unexplained syncope or seizure-like events.
- Post-syncopal hypertension may mimic hypertensive crisis but should alert clinicians to possible arrhythmic syncope.
- Permanent pacemaker implantation is a guideline-based, curative treatment for symptomatic high-grade or complete AV block.

Author contributions

Le HTT (Huong Thi Thanh Le) – conceived the report, drafted the manuscript, coordinated revisions. Nguyen XN (Ninh Xuan Nguyen) – collected clinical data, contributed to case analysis. Pham NT (Ngoc Tien Pham) – reviewed patient records, prepared the clinical timeline. Tran QV (Quoc Viet Tran) – assisted with diagnostic evaluation and interpretation of ECG/Holter findings. Tran HNT (Hang Ngoc Thuy Tran) – contributed to literature review and references. Tran NH (Nhat Hong Tran) – supervised cardiology management and reviewed the final manuscript.All authors read and approved the final manuscript.

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Data availability

All data supporting the findings are included in this article.

Declarations

Ethics approval

Not required for this case report in accordance with institutional and national guidelines.

Consent

Written informed consent for publication was obtained from the patient.

Competing interests

The authors declare no competing interests.

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