

Case report

Beyond idiopathic ventricular fibrillation: Vasospastic angina uncovered after ICD implantation

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Abstract

A woman in her 40s presented with epigastric pain and subsequently developed ventricular fibrillation (VF) cardiac arrest requiring resuscitation. The initial coronary angiography (CAG) revealed no significant stenosis. Due to limited resources and the unavailability of a provocation test, the patient was diagnosed with idiopathic VF, and an implantable cardioverter-defibrillator (ICD) was placed. Despite experiencing epigastric pain for two years with no significant endoscopic findings, an exercise stress test (EST) later identified ventricular arrhythmia, which suggested an ischemia-related mechanism despite normal coronary anatomy. During follow-up, she developed ST-elevation myocardial infarction, and a subsequent CAG revealed coronary spasm. She was diagnosed with vasospastic angina (VSA) and treated with nitrates and diltiazem. The patient is now free of epigastric pain and arrhythmic episodes. This case highlights the diagnostic challenges of VSA because of its atypical presentation and the limitations of resource-constrained settings, which can result in delayed diagnosis. Clinicians should consider VSA in patients with angina, sudden cardiac death, and abnormal EST results, especially in the absence of significant stenosis. While CAG with provocative testing is the standard diagnostic approach, alternative diagnostic pathways should be applied in resource-limited settings. Treatment options include medications and ICD placement, which should be considered for patients with high-risk VSA and recurrent ventricular arrhythmias.

Keywords: CAG with provocative test, vasospastic angina, ventricular fibrillation.

Vasospastic angina (VSA), also known as Prinzmetal or variant angina, is characterized by transient coronary artery spasm that leads to reversible myocardial ischemia, typically presenting as rest angina with transient ST-segment elevation. ⁽¹⁾ The diagnostic criteria include: (i) nitrate-responsive angina, (ii) transient ischemic electrocardiographic changes, and (iii) angiographic evidence of coronary artery spasm. ⁽²⁾ Although VSA is commonly associated with episodic chest pain at rest, it may also present as acute myocardial infarction, life-threatening

ventricular arrhythmias, or sudden cardiac death (SCD), even in patients with structurally normal hearts and no significant coronary obstruction. ⁽³⁾

Malignant arrhythmias in VSA are thought to originate from acute transmural ischemia during intense coronary spasm, which creates repolarization heterogeneity and electrical instability. Thus, predisposing patients to polymorphic ventricular tachycardia (VT) and ventricular fibrillation (VF). ⁽⁴⁾ Despite these potentially fatal consequences, VSA remains underrecognized, particularly when coronary angiography (CAG) reveals no fixed stenosis, and provocative testing is unavailable.

This case highlights the diagnostic complexity of VSA presenting with VF in a structurally normal heart with atypical symptoms, as well as delayed diagnosis in a resource-limited setting. Furthermore, it

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emphasizes the importance of considering coronary vasomotor disorders before establishing a diagnosis of idiopathic VF.

Case report

A 49-year-old woman developed acute epigastric pain and lost consciousness while returning home after dinner. She was brought to the hospital following a cardiac arrest, and her initial electrocardiogram (ECG) revealed VF. After 7 min of cardiopulmonary resuscitation with three defibrillation shocks, return of spontaneous circulation was achieved. The patient promptly regained full consciousness (GCS 15) without any neurological deficits, and her vital signs stabilized without requiring vasopressor support. Information on epinephrine or amiodarone administration was unavailable.

The postresuscitation ECG demonstrated paroxysmal atrial fibrillation without acute ischemic ST-segment changes (**Figure 1A**). A subsequent ECG showed accelerated idioventricular rhythm (**Figure 1B**) and transient ST-segment elevation lasting for a few minutes. Given the concern for acute coronary syndrome (ACS), she was treated with enoxaparin, aspirin, and clopidogrel for possible non-ST elevation ACS. She had no preceding symptoms and no known underlying diseases.

The patient was transferred to our center for further management. An elective CAG was performed one week after the initial arrest, once the patient was hemodynamically stable and neurologically intact. The CAG confirmed the absence of significant coronary stenosis.

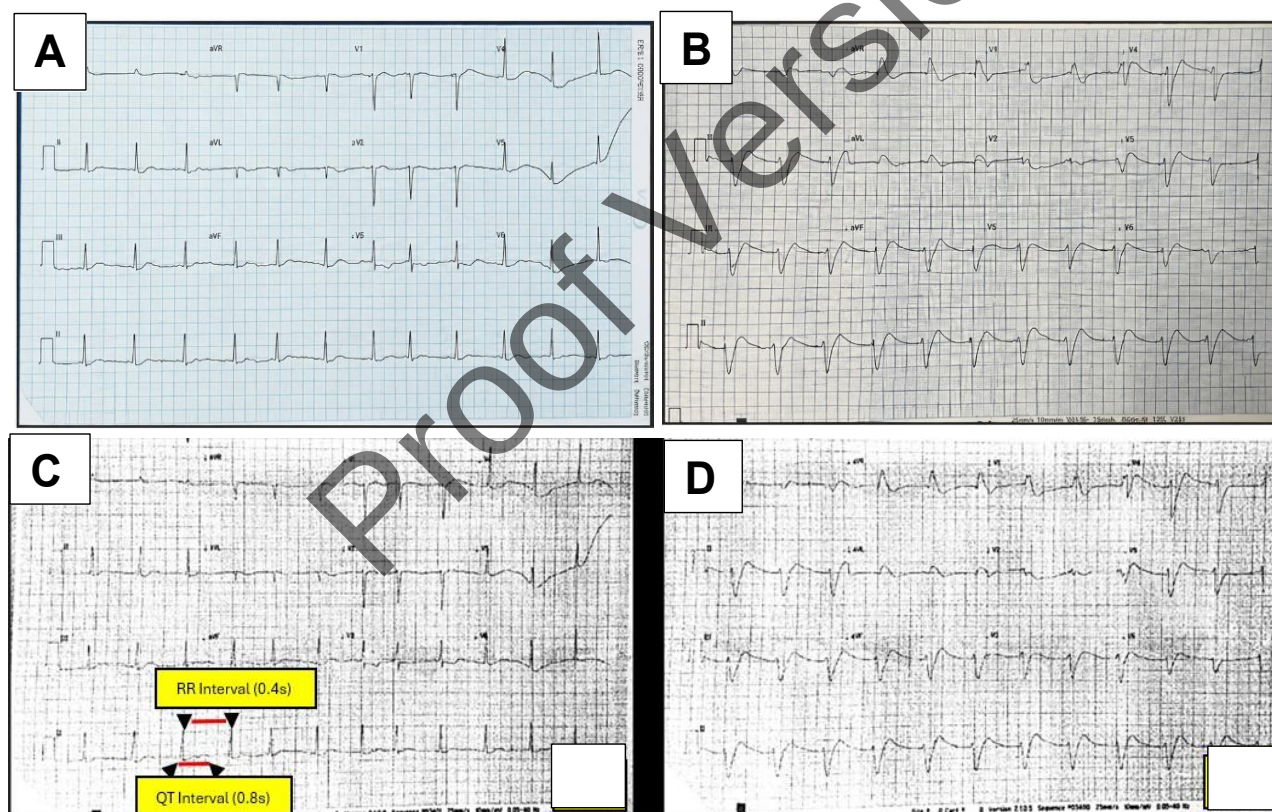


Figure 1. Electrocardiography after resuscitation from cardiac arrest. (A) Initial ECG showing paroxysmal atrial fibrillation without acute ischemic ST-segment changes, (B) follow-up ECG showing accelerated idioventricular rhythm (AIVR) with transient ST-segment elevation. (Image clarity was enhanced using AI-assisted processing for visualization only; ECG findings were not modified), (C) original ECG after the return of spontaneous circulation (ROSC). The rhythm shows a defined morphology with a QRS duration of 80 ms and a QTc interval of 440 ms; and (D) original ECG demonstrating wide QRS complexes. The rhythm is suggestive of AIVR with transient ST-segment elevation.

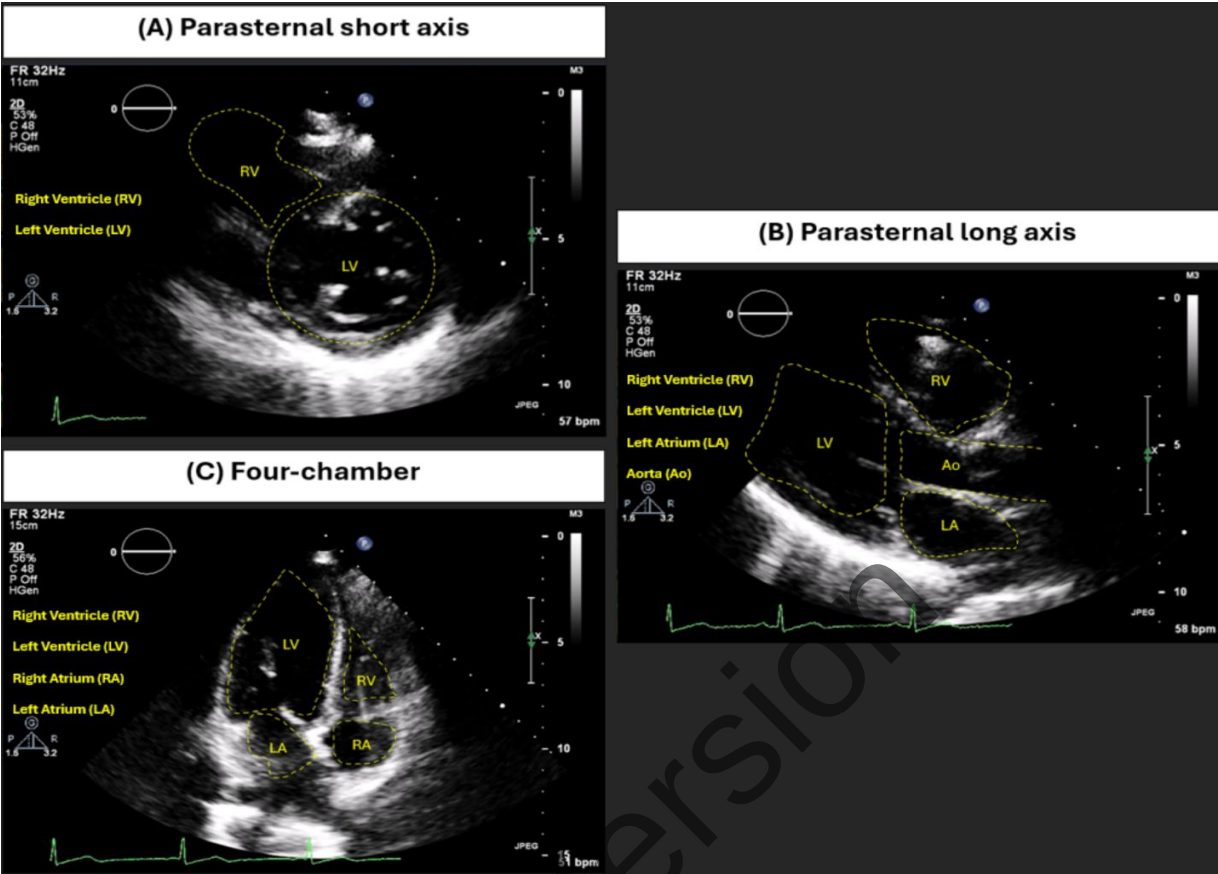


Figure 2. Transthoracic echocardiography findings. (A) Parasternal short-axis view demonstrating normal left ventricular (LV) and right ventricular (RV) morphology, (B) parasternal long-axis view showing the LV, RV, left atrium (LA), and aorta (Ao); and (C) apical four-chamber view demonstrating all four cardiac chambers. Dashed lines indicate chamber boundaries.

Table 1. ICD interrogation showed that Event V-4 was a 14-second-long spontaneous VT that spontaneously resolved and was not recognized by the patient. Event V-5 corresponds with EST.

Event	Data/Time	Type	Therapy	Duration(hh:mm:ss)
V-5	19 Apr 2019 10:56	VT	ATPx3	00:00:41
V-4	13 Sep 2018 19:10	VT	No Therapy	00:00:14
V-3	21 Mar 2017 17:44	VF	Induced: 23J	00:00:37
V-2	21 Mar 2017 17:43	VF	Induced: No Therapy	00:00:16
V-1	21 Mar 2017 17:42	VF	Induced: No Therapy	00:00:19

EST, exercise stress test; ICD, implantable cardioverter-defibrillator; VF, ventricular fibrillation; VT, ventricular tachycardia.

Coronary vasospasm provocation testing was not performed because the pharmacologic agents required for the test, specifically intracoronary acetylcholine or ergonovine, were not available at our center at that time. Given the presentation of unexplained VF with a normal CAG, inherited primary arrhythmic syndromes were systematically considered. Serial ECGs demonstrated normal corrected QT intervals, and no spontaneous type 1 Brugada pattern was observed, including assessment of right precordial leads. Moreover, early repolarization patterns were not identified. Transthoracic echocardiography showed normal cardiac structure and preserved ventricular

function (**Figure 2**). Serum electrolytes, thyroid function, and toxicology screening were within normal limits. Cardiac magnetic resonance imaging and sodium channel blocker challenge testing were not performed due to limited availability at the initial institution. The patient was therefore diagnosed with idiopathic VF and underwent implantation of an ICD. Over the next two years, the patient experienced recurrent epigastric pain despite treatment for dyspepsia. During a follow-up exercise stress test (EST), she developed symptoms, and the ICD recorded a 41 s polymorphic VT, which was successfully terminated by ATPx3 (V-5 in **Table 1**).

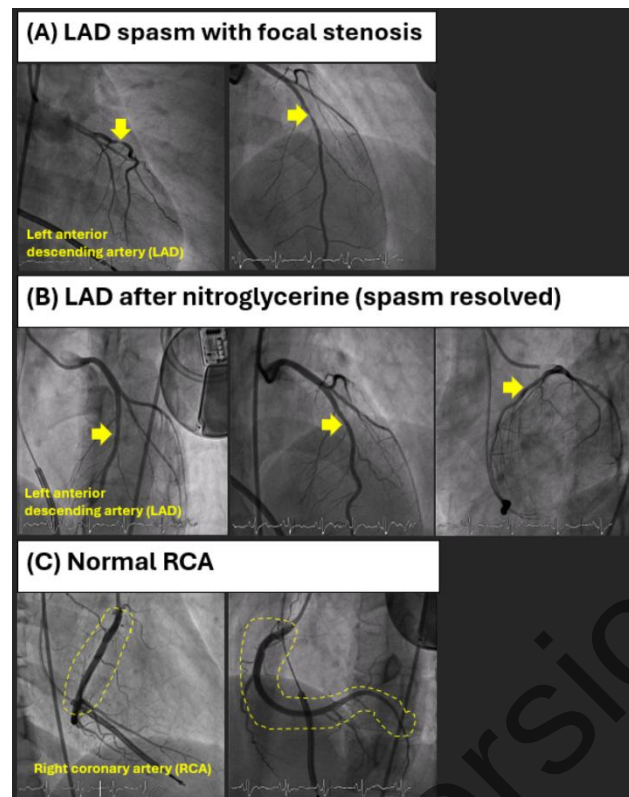


Figure 3. (A) CAG during the latest STE-ACS showed coronary spasm with a focal significant LAD stenosis; (B) The LAD vasospasm resolved after nitroglycerine (NTG) administration; and (C) CAG revealed a normal RCA. CAG, coronary angiography; LAD, left anterior descending artery; RCA, right coronary artery.

In total, two spontaneous ventricular arrhythmic events were documented: the VT during the EST and a 14 s self-terminating VT (V-4). This excludes three iatrogenic episodes (V-1 to V-3) induced during defibrillation threshold (DFT) testing to verify device efficacy. During DFT testing, the first two induced self-terminated episodes (V-1 and V-2), while a 23-joule shock terminated the third (V-3).

During the visit, when the diagnosis was finally established, she developed chest pain during an outpatient follow-up. Her ECG showed transient ST elevation in leads V2–V3, and troponin T was positive; therefore, she was diagnosed with ST-elevation myocardial infarction. She received dual antiplatelet therapy of enoxaparin and a statin. CAG revealed left anterior descending artery (LAD) spasm without obstructive disease, which normalized after nitroglycerin administration (**Figure 3**). She was subsequently diagnosed with VSA and started on diltiazem 180 mg and isosorbide mononitrate 30 mg daily. She has remained asymptomatic with regular 6-month follow-ups.

Discussion

A 49-year-old healthy woman presented with epigastric discomfort and SCD secondary to VF. She had initially been diagnosed with functional dyspepsia and idiopathic VF but was finally diagnosed with VSA following a protracted diagnostic workup.

The prevalence of VSA is not well-documented; however, it appears to be higher among the Japanese population compared to the Caucasian population.^(1,4) Determining the exact incidence is challenging due to the variability in clinical presentations (such as stable angina, SCD, ACS, arrhythmia, or syncope), diagnostic methods, and population characteristics, with substantial differences between Eastern and Western countries. VSA has a high prevalence of coronary spasm in Asian populations.⁽⁴⁾

Currently, VSA may be underdiagnosed because fixed obstruction is a considerably more common cause of angina. Moreover, VSA may exhibit various manifestations and is rarely encountered by physicians⁽¹⁾, as can be noted from our patient, who was initially misdiagnosed as “functional dyspepsia” with “idiopathic VF”.

Previous studies have reported malignant ventricular arrhythmias and SCD as uncommon but recognized manifestations of VSA.^(5,6) Chevalier *et al.* described seven VF/VT cardiac arrest survivors who had normal coronary arteries and positive ergonovine provocation testing.⁽⁷⁾ However, unlike our case, none exhibited prolonged atypical symptoms, such as recurrent epigastric pain, and exercise-induced ventricular arrhythmia. Moreover, in those cases, diagnosis was promptly established with provocative testing. In contrast, the absence of such testing in a resource-limited setting contributed to a 2-year delay before spontaneous ST-elevation with angiographically confirmed vasospasm established the diagnosis for our patient.

Although the patient's CAG demonstrated no significant stenosis, the clinical presentation raised suspicion for VSA. Furthermore, the EST-induced polymorphic VT broadened the differential diagnosis to include transient ischemia from dynamic coronary vasospasm as well as primary electrical disorders such as long QT syndrome type 1 and catecholaminergic polymorphic ventricular tachycardia.^(4,8)

In addition to plaque rupture, coronary vasospasm may precipitate life-threatening ventricular arrhythmias such as VT and VF, which can rapidly result in cardiac arrest.⁽⁵⁾ Patients with multivessel coronary spasm are at particularly high risk for fatal arrhythmias.⁽⁹⁾ VF in VSA is thought to result from acute transmural ischemia during intense coronary spasm. An abrupt reduction in coronary flow creates marked repolarization heterogeneity between ischemic and non-ischemic myocardium, thereby increasing QT dispersion and electrical instability. This facilitates phase 2 reentry, polymorphic VT, and VF degeneration. Moreover, ischemia-related alterations in ion channels may further promote arrhythmogenesis, which suggests that VF in VSA arises from dynamic ischemia-mediated electrical instability rather than from structural heart disease.⁽⁴⁾

The Coronary Vasomotion Disorders International Study Group (COVADIS) diagnostic criteria for VSA include: 1) nitrate-responsive angina, 2) transient ischemic ECG changes during spontaneous episodes without increased oxygen demand, and 3) angiographic evidence of coronary spasm.⁽²⁾ VSA is classified as definitive when all three criteria are met and as suspected when two of the criteria are present.⁽⁴⁾ In addition, diagnosing episodes in non-hospital settings is challenging, which makes CAG with

provocative testing a practical diagnostic approach. Agents such as ergonovine, acetylcholine, hyperventilation, or cold-air inhalation may be used to induce chest pain, ECG changes, and >90% coronary constriction. However, provocative testing may not always induce ventricular arrhythmias, with a specificity of 99.0% and a sensitivity of 90%–94%.⁽¹⁰⁾ Furthermore, the limited availability of provocation agents and advanced intracoronary imaging modalities, such as intravascular ultrasound or optical coherence tomography, may restrict the comprehensive evaluation in VSA.⁽¹¹⁾

In resource-rich settings, provocation testing and sodium channel blocker testing are often employed to exclude VSA and Brugada syndrome before establishing a diagnosis of idiopathic VF. Our case underscores the importance of considering VSA and performing pharmacologic provocation testing before diagnosing idiopathic VF and proceeding with ICD implantation. Moreover, in resource-limited settings where provocative testing is unavailable, patients with suspected VSA remain at risk for recurrent VF/VT before definitive diagnosis. Therefore, careful interim surveillance with ambulatory rhythm monitoring may help detect recurrent arrhythmias and guide management. In addition, an empirical trial of calcium channel blockers and nitrates may serve diagnostic and therapeutic purposes. In retrospect, earlier vasodilator therapy in our patient may have reduced recurrent vasospastic episodes and ventricular arrhythmias during the prolonged diagnostic period.

In our case, repeat CAG with provocative testing was unavailable at any academic center in the country. However, she developed recurrent epigastric pain on the day of presentation to our hospital, which allowed for the real-time documentation of the episode. ECG and CAG demonstrated transient ST-segment elevation and LAD vasospasm that resolved after nitroglycerin administration. She therefore fulfilled all three COVADIS criteria for definitive VSA diagnosis.⁽²⁾

In summary, this patient developed unprovoked sudden cardiac arrest caused by VF secondary to coronary vasospasm. Because spontaneous vasospastic attacks are associated with poor survival even in the absence of myocardial infarction⁽⁹⁾, and ventricular arrhythmia related to coronary vasospasm has a high risk of recurrence⁽⁴⁾, appropriate medical therapy was required. Calcium channel blockers and nitrates were prescribed to suppress the vasospastic

episodes.⁽⁴⁾ In addition, risk factor modification is essential. Smoking cessation is recommended to reduce the risk of recurrence⁽⁴⁾, and nonselective beta-blockers should generally be avoided because they may cause unopposed alpha-adrenergic vasoconstriction.⁽⁴⁾ Moreover, oral sumatriptan should be avoided because it may precipitate coronary vasospasm.⁽⁴⁾ Percutaneous coronary intervention is not routinely indicated in patients without fixed obstructive disease but may be considered in medically refractory vasospasm or coexisting fixed coronary obstruction.⁽⁴⁾

Although our patient received appropriate medical therapy, patients with VSA complicated by aborted SCD have a worse prognosis than those with VSA alone. Unfortunately, intracardiac electrogram recordings of the arrhythmic episode were not stored by the device and were therefore unavailable for review. We acknowledge that ATP-mediated termination of polymorphic VT is atypical and limits the definitive characterization of the rhythm. Nevertheless, the temporal association with vasospastic symptoms and subsequent response to vasodilator therapy supported coronary vasospasm as the most likely underlying mechanism. ICD implantation is therefore recommended as secondary prevention in high-risk patients.⁽⁴⁾ Our patient experienced recurrent ventricular arrhythmias and survived VF arrest, which supports the continuation of ICD therapy despite symptom control through medical treatment. Importantly, even if VSA had been confirmed at the index presentation, ICD implantation would still have been reasonable because recurrent malignant arrhythmias may occur despite vasodilator therapy. Therefore, the definitive diagnosis of VSA should not delay ICD implantation in high-risk patients. However, evidence supporting ICD therapy in patients with vasospastic disease and prior fatal arrhythmias remains limited, and its mortality benefit remains uncertain. In this context, although subcutaneous ICDs are generally favored in younger patients without pacing requirements, the apparent effectiveness of ATP in our patient may suggest the potential therapeutic advantage of a transvenous ICD capable of painless arrhythmia termination.

This case presents several clinical clues that may facilitate the earlier recognition of VSA. Recurrent rest symptoms, even when atypical, such as epigastric pain, in the setting of unexplained ventricular arrhythmias and angiographically normal coronary

arteries, should raise suspicion for a vasomotor etiology. Exercise-induced polymorphic VT in the absence of structural heart disease should prompt evaluation for dynamic coronary spasm, while primary electrical disorders such as catecholaminergic polymorphic ventricular tachycardia, congenital long QT syndrome (particularly type 1), short QT syndrome, and early repolarization syndrome should also be considered. In our patient, the normal baseline corrected QT interval and absence of structural heart disease made a dynamic ischemic mechanism due to coronary vasospasm more likely. In resource-limited settings where provocative testing is unavailable, the careful assessment of symptom patterns, transient ECG changes, and response to vasodilator therapy may support an earlier diagnosis.

Conclusion

This case underscores that VSA can present as VF and sudden cardiac arrest despite a normal CAG and structurally normal heart. Atypical symptoms and limited access to provocative testing may delay diagnosis. However, clinicians should consider coronary vasomotor disorders in patients with unexplained ventricular arrhythmias before diagnosing idiopathic VF, as timely treatment with calcium channel blockers and nitrates can prevent recurrent vasospasms and life-threatening arrhythmias.

Author contributions

NK, ST, and AW contributed substantially to the study concept and design, data acquisition, literature review, and data analysis and interpretation. NK, ST, and KR contributed substantially to data acquisition. NK, ST, KR, TS, CP, and TS contributed to drafting the manuscript. NK, TY, and NW edited the manuscript critically for important intellectual content.

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Conflict of interest statement

All authors have completed and submitted the International Committee of Medical Journal Editors Uniform Disclosure Form for Potential Conflicts of Interest. All authors declare that they have no conflicts of interest.

Data sharing statement

Data generated or analyzed for the present report are included in this published article. Further details are available from the corresponding author on reasonable request after deidentification of the patient whose data are included in the report.

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