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Prinzmetal Angina: A Case Report of Recurrent Ventricular Fibrillation

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Abstract

Prinzmetal angina, also known as vasospastic or variant angina, is characterized by episodic coronary artery spasm leading to transient myocardial ischemia, typically presenting with chest pain and transient ST-segment elevation on ECG. Although usually benign and reversible, severe cases may result in life-threatening ventricular arrhythmias, including ventricular fibrillation (VF). We report the case of a 66-year-old male with a history of hypertension and prior aortic root replacement who presented with recurrent VF secondary to coronary vasospasm despite optimal medical

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therapy. Electrocardiography demonstrated transient ST-segment elevation, and coronary angiography revealed moderate atherosclerotic stenosis with superimposed severe coronary vasospasm. Despite aggressive vasodilator therapy, the patient continued to experience recurrent malignant arrhythmias requiring multiple defibrillations. Ultimately, coronary stenting of the affected vessels led to clinical stabilization and cessation of arrhythmic events. This case highlights the diagnostic and therapeutic challenges of refractory vasospastic angina complicated by malignant ventricular arrhythmias and suggests a potential role for percutaneous coronary intervention in selected cases unresponsive to medical therapy.

Keywords: Vasospastic angina; variant angina; coronary artery spasm; ventricular fibrillation; ST-segment elevation; coronary stenting; refractory angina

Introduction

Variant angina (VA), also known as Prinzmetal or vasospastic angina, is a distinct form of angina characterized by episodic coronary artery spasms that result in transient myocardial ischemia (Mayer & Hillis, 1998). Unlike typical exertional angina caused by fixed atherosclerotic obstruction, VA is primarily due to reversible coronary vasospasm, often occurring in arteries with little or no significant plaque burden. These spasms lead to transient chest pain and are frequently associated with ST-segment elevation on electrocardiography, mimicking acute coronary syndromes (Song, 2018).

The pathophysiology of VA is complex and multifactorial. A key mechanism involves endothelial dysfunction, in which reduced nitric oxide bioavailability impairs vasodilation and increases vascular smooth muscle contractility, predisposing coronary arteries to spasm. Atherosclerosis may also contribute by further disrupting endothelial function and nitric oxide production in affected segments, thereby enhancing vasoreactivity (Yoo et al., 2009; Kawashima & Yokoyama, 2004; Lin et al., 2022). Supporting this association, studies have reported that up to 88% of coronary artery spasm episodes occur at sites of underlying atherosclerotic lesions (Lee et al., 2002).

Smoking is a well-established risk factor for vasospastic angina (VA), with multiple studies demonstrating a strong association [8]. This is consistent with our patient, who had a significant smoking history. Although VA is typically considered a manageable condition, severe or prolonged coronary spasms can lead to life-threatening arrhythmias, including ventricular fibrillation (VF). While VF has been reported in association with VA, it remains an uncommon and underrecognized complication, with limited data on its management in refractory cases (Khiatah et al., 2020).

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The gold standard for diagnosing VA is coronary angiography with provocation testing using acetylcholine, ergonovine, or methylergonovine to induce and directly visualize coronary spasms (Picard et al., 2019). Notably, acetylcholine provocation testing has been shown to yield higher positivity rates in male patients, which is consistent with our case of a male patient with VA and a positive test result (Saito et al., 2022).

First-line treatment for VA includes calcium channel blockers and nitrates, which are effective in preventing coronary spasms and relieving symptoms. However, in some cases, VA remains refractory to standard therapy, resulting in recurrent ischemic episodes and arrhythmias despite optimal medical management (Harris et al., 2016; Kusama et al., 2011).

This case report describes a patient with recurrent ventricular fibrillation (VF) caused by vasospastic angina (VA) who did not respond to calcium channel blockers or nitrates and ultimately required coronary stenting. It highlights the difficulty of managing VA-related arrhythmias and underscores the importance of considering alternative treatment strategies in refractory cases.

Case Presentation:

A 66-year-old man presented to the emergency department with chest pain, shortness of breath, generalized weakness, and a brief loss of consciousness. His vital signs were: blood pressure 135/80 mmHg, temperature 36.3°C, heart rate 70 beats per minute, respiratory rate 18 breaths per minute, and oxygen saturation 98% on room air.

The patient had a history of smoking one pack per day and arterial hypertension, with maximum recorded blood pressure values of up to 180/100 mmHg. He had undergone an aortic root replacement in 2015 for an ascending aortic dissection. The patient has been treated for hypertension with losartan 100 mg and lercanidipine 10 mg.

Presentation and Initial Evaluation

On admission, the physical examination revealed clear lungs, equal breath sounds bilaterally, and a soft, non-tender abdomen. Blood samples were collected and laboratory tests were conducted (Table 1). Initial laboratory tests showed a troponin level of 0.4ng/dl (Normal <0.5ng/dl). His ECG indicated sinus rhythm at 73 beats per minute, with ST elevation in leads II, III, and aVF , and ST depression in leads I and aVL. (Figure 1).

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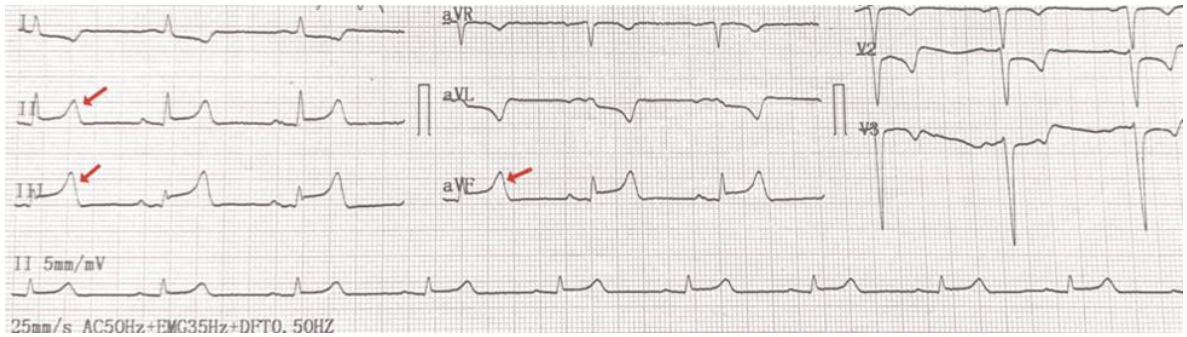
Echocardiography Findings

Figure 1: Initial ECG done on admission



ECG shows ST elevation of 0.3mV in leads II, III and aVF.

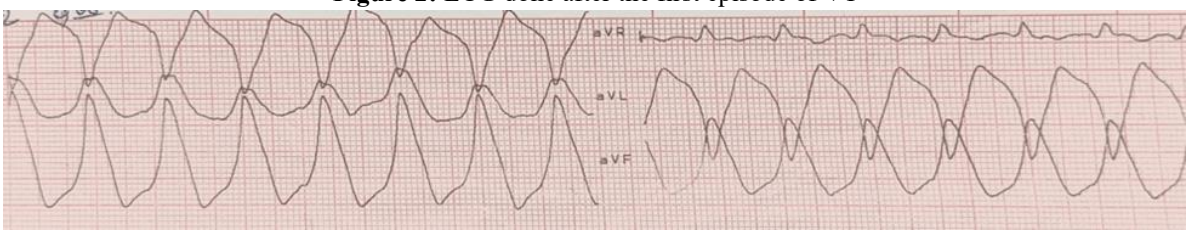
Table 1: Laboratory studies conducted on admission

Parameters	Laboratory values	Reference range
Lactate	<0.9 mmol/L	0.5-2.1 mmol/L
C-Reactive Protein	70.3 mg/L	0-5 mg/L
Magnesium	0.86 mmol/L	0.85-1.10 mmol/L
Calcium	2.16 mmol/L	2.1–2.6 mmol/L
Glucose	81 mg/dL	70-99 mg/dL
INR	2.15	0.8-1.1
Prothrombin Time	21.8 seconds	11-15 seconds
Activated thromboplastin time (PTT)	43.0 seconds	25-40 seconds
Thrombin Time	14.0 seconds	11-19 seconds
Creatine Kinase	43 U/L	25–150 U/L
Troponin I	0.4 ng/dL	<0.5 ng/dL

Echocardiography showed an aortic bulbar aneurysm with dissection, descending aortic dilation, abdominal aortic dissection and mild aortic and mitral valve regurgitation. The left atrium was mildly dilated. Left ventricular ejection fraction (LVEF) of 60%.

Approximately 10 minutes after admission, the patient experienced ventricular tachycardia (VT) (Figure 2), which required defibrillation. He was promptly transferred to the cardiac catheterization laboratory.

Figure 2: ECG done after the first episode of VT



ECG shows VT.

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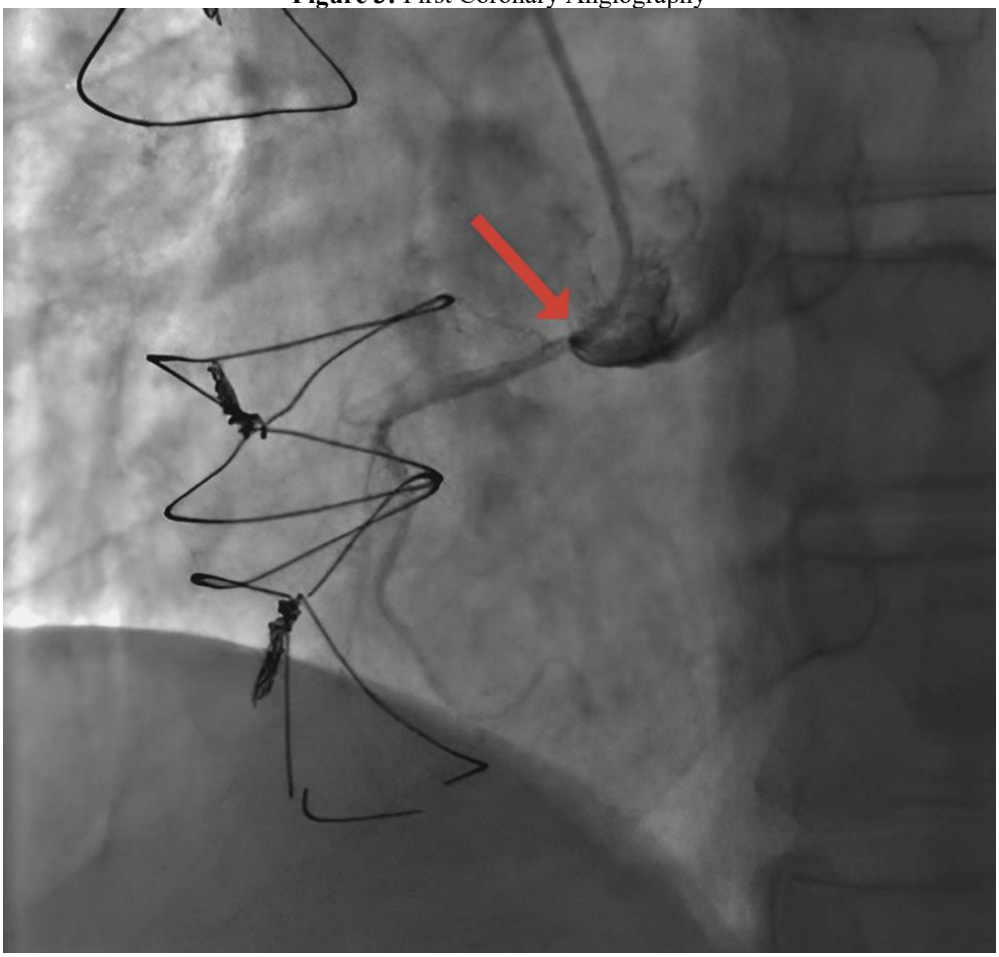
The coronary angiography performed revealed a 30-40% stenosis in the left anterior descending (LAD) artery, a 90% stenosis in the proximal segment of a small-diameter diagonal artery, and a 30-40% stenosis in the distal circumflex artery (LCX). The right coronary artery appeared hypoplastic and was only partially visualized due to dissection (Figure 3). Despite the diagonal artery having 90% stenosis, a decision was made that Drug-eluting stent (DES) placement was not necessary due to its small diameter.

Figure 3: First Coronary Angiography



The arrow indicates the dissection of the hypoplastic right coronary artery.

Further Interventions and Course

The patient underwent open-heart cardiac surgery for the resection of a false aneurysm of the aortic bulb using a vascular prosthesis. Following surgery, he was placed under observation and remained hemodynamically stable, with a heart rate of 85 bpm, blood pressure of 130/65 mmHg, and oxygen saturation of 99%.

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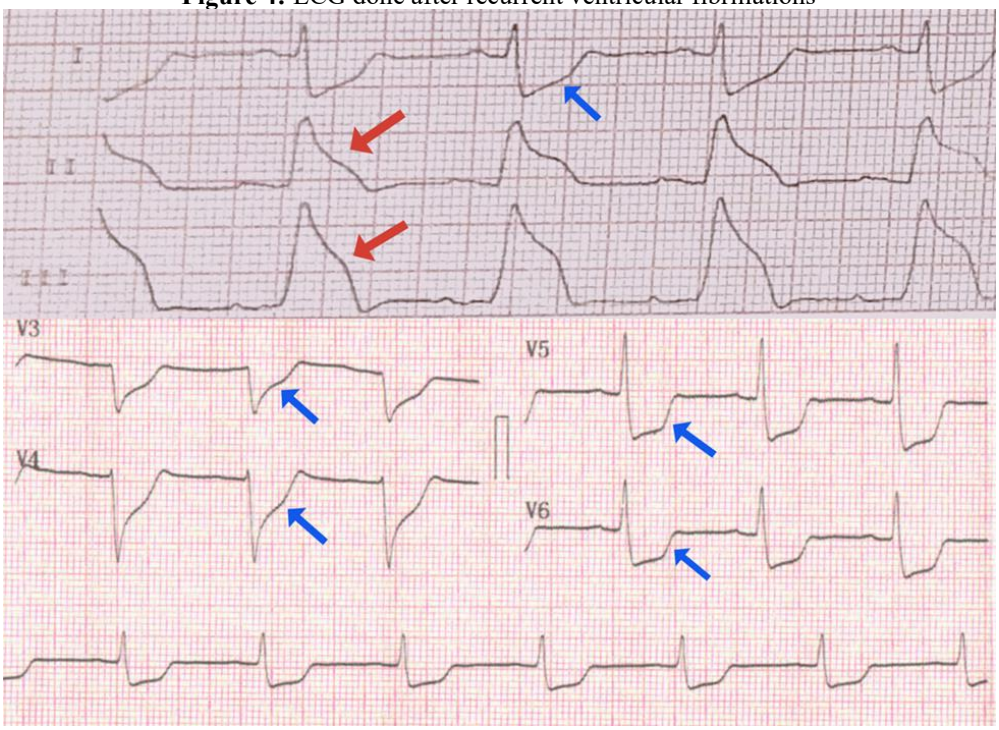
However, on the following day, he developed a complete atrioventricular (AV) block and became hemodynamically unstable, with a blood pressure of 80/40 mmHg and a heart rate of 35 bpm, necessitating temporary cardiac pacing and an infusion of dopamine at 200mg/50mL (5mL/hr) and adjusted based on the patient's hemodynamic readings. The patient was stabilized within an hour, and the infusion was discontinued. By the next day, the AV block had resolved, but he was maintained on temporary cardiac pacing in demand mode.

Later that day, he experienced VT and VF, requiring multiple defibrillations and aggressive treatment. A continuous infusion of IV amiodarone 600mg/50mL at 2.2 mL/hr was initiated. Despite these interventions, VF recurred, prompting the addition of verapamil (80 mg twice daily) and IV nitroglycerin. These arrhythmic episodes were frequently preceded by angina and ST elevation on ECG, suggesting an ischemic etiology (Figure 4).

The patient remained under observation, with VF episodes becoming less frequent. However, given the concern for an ischemic cause, a coronary angiography was scheduled.

Figure 4: ECG done after recurrent ventricular fibrillations



ECG shows ST elevation of 0.8 mV, especially visible in leads II, III and ST depression of 0.8mV in I and V3-V6.

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A repeat coronary angiography demonstrated persistent stenoses, including a 30-40% stenosis in the LAD artery, a 90% stenosis in the diagonal artery, a 30-40% stenosis in the LCX, and a 90% proximal stenosis in the right coronary artery (RCA), which was previously only partially visualized. There were no signs of aortic valve dissection. A DES was placed in the proximal right coronary artery (Figure 5).

Figure 5: Coronary Angiography done a week later

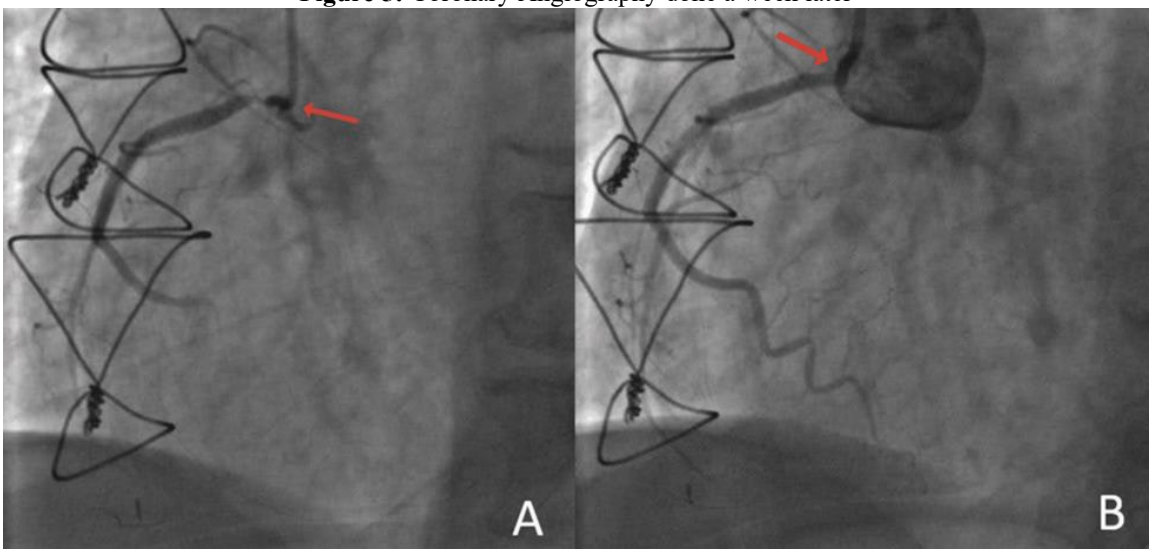

Figure A shows 90% stenosis in the right coronary artery.
Figure B: RCA angiography after DES placement.

Despite these interventions and aggressive treatment with antiarrhythmic and vasodilators, the patient continued to experience recurrent VF in the Intensive Care Unit, frequently preceded by angina and ST elevation.

CT angiography was performed to rule out postoperative mechanical compression of the coronary arteries. The imaging revealed a calcified atherosclerotic plaque in the mid-segment of the LAD, causing 50-60% luminal narrowing. Notably, the degree of narrowing appeared more severe compared to the previous coronary angiography, despite CT angiography generally tending to underestimate stenosis severity. Given this discrepancy, vasospasm was suspected because a higher degree of stenosis was detected on CT angiography compared to coronary angiography; a suspicion of coronary spasm was therefore raised. For this underlying cause, coronary angiography with provocation testing was scheduled.

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Subsequent Imaging and Findings

To confirm the initial suspicion, coronary angiography was performed. It revealed multiple coronary artery lesions, including a 30–40% stenosis in the LAD, which was significantly less severe than that shown on CT angiography; a 90% stenosis in the diagonal artery; and a 30–40% stenosis in the LCX.

To further evaluate the underlying cause, an acetylcholine provocation test was performed. This confirmed the presence of coronary vasospasm, with 50–60% stenosis in the LAD and 90% stenosis in the LCX, supporting the diagnosis of vasospastic angina (VA). Notably, LCX spasm triggered ventricular fibrillation (VF) on ECG, further supporting the role of vasospasm in the arrhythmic events.

Given the association between LCX spasm and VF, drug-eluting stents (DES) were implanted in both the LCX and LAD arteries to stabilize the condition (Figure 6). Although the plaques were not hemodynamically significant, they likely contributed to vasospasm susceptibility. The initial episode of VF in the emergency department was also likely caused by coronary spasm.

Ultimately, the diagnosis of VA was confirmed based on the patient's clinical presentation, ST-segment elevation on ECG, and documentation of coronary artery spasm during angiography.

Figure 6: Last coronary angiography

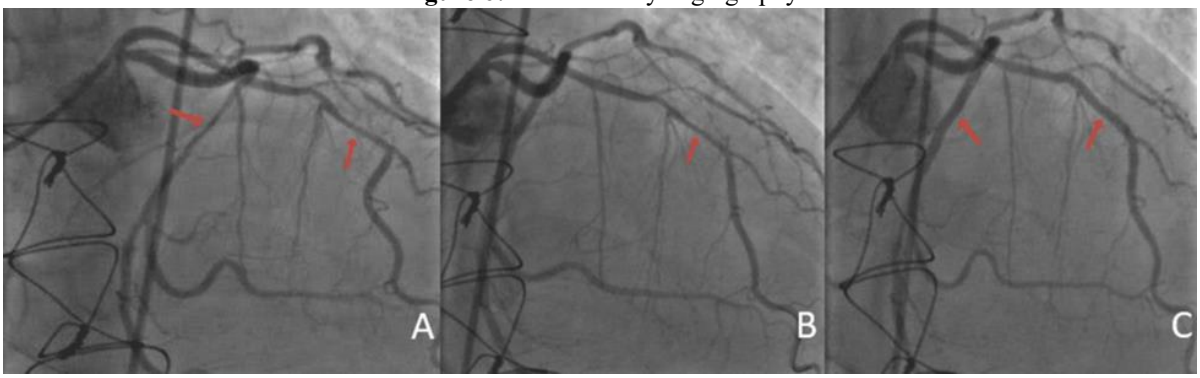

Figure A: Shows spasm of the LAD and LCX arteries; Figure B: Shows spasm of only the LAD artery after the LCX artery was stented; Figure C: Both LAD and LCX arteries have been stented.

The patient continued treatment in the Intensive Care Unit, and within a few hours became active, with marked clinical improvement. No further episodes of arrhythmia were observed, and his condition remained stable post-intervention.

He was discharged in satisfactory condition and prescribed aspirin (100 mg once daily), clopidogrel (75 mg daily), rosuvastatin (40 mg daily),

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verapamil (80 mg twice daily), perindopril (10 mg once daily), and pantoprazole (40 mg once daily).

This case highlights the importance of recognizing VA as a potential cause of ventricular arrhythmias and underscores the role of early coronary intervention and medical therapy in achieving a favorable patient outcome.

Discussion

This study describes a rare and severe complication of vasospastic angina (VA), presenting as ventricular fibrillation (VF) that was refractory to standard pharmacologic therapy, including calcium channel blockers and nitrates. Although the patient initially demonstrated partial responsiveness, recurrent episodes ultimately necessitated definitive management with coronary stenting.

A comprehensive literature review was performed using Google Scholar and PubMed to identify similar cases and evaluate evidence-based treatment strategies associated with optimal clinical outcomes. This case underscores the potential for refractory vasospastic angina and highlights the need for further research into management strategies for patients who do not respond to conventional medical therapy.

A study exploring treatment options for patients unresponsive to calcium channel blockers and nitrates highlighted the potential benefits of alternative pharmacological approaches, such as anti-adrenergic agents (e.g., prazosin and clonidine), as well as interventional procedures like stent implantation (Lanza & Shimokawa, 2023), which was successfully applied in the present case.

Our review identified multiple reports of refractory VA successfully treated with stent implantation. For example, a similar case of VA complicated by ventricular tachycardia (VT) was managed with coronary stenting, leading to the resolution of recurrent arrhythmias (Ono et al., 2024). This supports the role of percutaneous intervention in refractory cases where medical therapy alone is insufficient. Additionally, a case series on stent implantation for recurrent VA reported that four out of five patients experienced recurrent spasm, with three requiring additional stenting and one responding to pharmacologic therapy. While some patients remained asymptomatic during follow-up, restenosis and new lesions were observed in others (Martí et al., 2006). Another report described two cases of VA associated with coronary artery disease, both successfully treated with percutaneous coronary intervention and stent placement, further reinforcing the viability of this approach in managing VA (Kleyman et al., 2019).

To assess potential complications associated with stenting in vasospastic angina (VA), we reviewed relevant studies evaluating procedural risks. A study on coronary stenting for severe, pharmacologically refractory

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coronary artery spasm reported no cases of recurrent unstable ischemia requiring hospitalization after stent placement; however, restenosis occurred in three patients, necessitating repeat revascularization (Khatri et al., 2002). These findings suggest that while medical therapy remains the first-line treatment, stenting may serve as an adjunctive option in carefully selected cases. Nevertheless, stent implantation in VA patients is not without risk. Another study reported that stent-edge spasm occurred in 19.2% of cases during follow-up, although only one patient developed acute stent thrombosis, and most patients experienced no immediate post-procedural complications (Kaku et al., 2005).

Given these limitations, alternative therapeutic strategies for refractory VA have also been explored. One study indicated that patients with severe or refractory VA may fail to respond to conventional therapy and can experience life-threatening arrhythmias or sudden cardiac death during ischemic episodes, which have been linked to immune-inflammatory mechanisms. Notably, the study reported successful remission following treatment with glucocorticoids and immunoglobulin therapy, suggesting a potential alternative approach in selected cases (He, 2023).

Conclusions

This case highlights the challenges in managing ventricular arrhythmias (VA) complicated by recurrent ventricular fibrillation (VF) that was refractory to standard vasodilator therapy, including calcium channel blockers and nitrates. While medical therapy remains the first-line approach, this case suggests that alternative interventional strategies may be considered in selected patients who do not respond to conventional treatment.

The resolution of VF episodes following drug-eluting stent (DES) implantation raises the possibility that coronary revascularization may play a role in preventing recurrent malignant arrhythmias, particularly when coronary vasospasm or ischemia is identified as a potential underlying mechanism. However, a definitive causal relationship between stent implantation and arrhythmia suppression cannot be established based on a single case.

Given the limited available evidence, the use of DES for refractory ventricular arrhythmias should currently be considered on a case-by-case basis. Further studies are warranted to clarify appropriate patient selection, evaluate long-term outcomes, and assess potential risks associated with this approach.

This case adds to the growing body of literature suggesting that coronary stenting may contribute to arrhythmia control in selected patients with vasospasm-related or ischemia-driven ventricular arrhythmias, although its role remains to be defined.

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Declaration for Human Participants: This study has been approved by the LTD Open Heart –University Hospital local ethics committee, and the principles of the Helsinki Declaration were followed.

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