ESI Preprints **Not Peer-reviewed**

Prinzmetal Angina: A Case of Recurrent Ventricular Fibrillation

Lali Gujejiani, MD, PhD
Associate Professor, European University, Tbilisi, Georgia
Kakhaber Latsuzbaia, MD
Invited Lecturer, East European University, Tbilisi, Georgia
Jaba Tsivilashvili, MD
Invited Lecturer, Caucasus International University, Tbilisi, Georgia
Khatuna Jalabadze, MD
Associate Professor, Tbilisi State Medical Academy, Georgia
Maia Kharebashvili, MD
Assistant Professor, Caucasus International University, Tbilisi, Georgia
Nato Jinjolia, MD, PhD
Georgian-American University (GAU), Tbilisi, Georgia
Vakhtang Tetradze, MD
Invited Lecturer, Tbilisi State Medical University, Georgia
Prof. Iamze Taboridze, PhD
David Aghmashenebeli University of Georgia, Tbilisi, Georgia

Doi: 10.19044/esipreprint.2.2026.p24

Approved: 08 February 2026
Posted: 10 February 2026

Cite As:
Gujejiani, L., Latsuzbaia, K., Tsivilashvili, J., Jalabadze, K., Kharebashvili, M., Jinjolia, N., Tetradze, V. & Taboridze, I. (2026). *Prinzmetal Angina: A Case of Recurrent Ventricular Fibrillation.* ESI Preprints. https://doi.org/10.19044/esipreprint.2.2026.p24

Abstract

Prinzmetal angina (also known as vasospastic or variant angina) is characterized by episodic coronary artery spasms, leading to transient ischemia and angina, often accompanied by ST elevation on ECG. While typically non-myocardial infarction in nature, severe cases can progress to life-threatening complications such as ventricular fibrillation (VF). This report details the case of a 66-year-old male with hypertension and a history of aortic root replacement, presenting with recurrent VF secondary to coronary vasospasm despite optimal medical therapy. ECG showed ST elevation, and coronary angiography revealed moderate stenosis with severe

vasospasm. Despite treatment with vasodilators, the patient experienced persistent arrhythmias, necessitating multiple defibrillations. Coronary stenting of the affected arteries ultimately led to stabilization. This case highlights the challenges of managing refractory arrhythmias in VA and underscores the potential role of interventional therapy when medical management fails.

Keywords: Vasospastic angina, dissection coronary artery, Treatment

Introduction

Variant angina (VA), also known as Prinzmetal or vasospastic angina (VSA), is a distinct type of angina characterized by episodic coronary artery spasms that lead to transient myocardial ischemia (Mayer, S., & Hillis, 1998). Unlike typical angina associated with atherosclerosis, VA results from reversible coronary spasms, often occurring in arteries with little or no atherosclerotic plaque. These spasms cause transient chest pain, frequently accompanied by ST-segment elevation on electrocardiography (ECG), similar to acute coronary syndromes (Song,2018). VA is also linked to endothelial dysfunction, where non-obstructive plaques or heightened vascular smooth muscle sensitivity predispose the arteries to spasm (Rehan, 2022).

The underlying mechanisms of VA remain complex and multifactorial. One widely accepted hypothesis attributes VA to endothelial dysfunction, where reduced nitric oxide bioavailability leads to impaired vasodilation and increased coronary artery contractility, promoting spasms. Another significant contributing factor is atherosclerosis, which may further impair nitric oxide production in affected segments, exacerbating vasospasm (Yoo et al., 2009), Kawashima & Yokoyama, 2004; Lin et al., 2022). Supporting this, studies have shown that up to 88% of coronary artery spasms localize to sites of atherosclerotic lesions (Lee et al., 2002).

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

The gold standard for diagnosing VA is coronary angiography with provocation testing using acetylcholine, ergonovine, or methylergonovine to induce and directly visualize spasms (Picard et al., 2019). Notably, acetylcholine provocation testing has been found to yield more positive

results in male patients, 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 help prevent spasms and alleviate symptoms. However, in some cases, VA remains refractory to standard therapy, leading to recurrent ischemic episodes and arrhythmias despite optimal medical management (Harriset al,. 2016; Kusamaet al., 2011).

This case report describes a patient with recurrent VF due to VA who failed to respond to calcium channel blockers and nitrates and ultimately required coronary stenting. It highlights the challenges in managing VA-related arrhythmias and emphasizes the need for alternative therapeutic 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 of 18 breaths per minute and oxygen saturation 98% on room air.

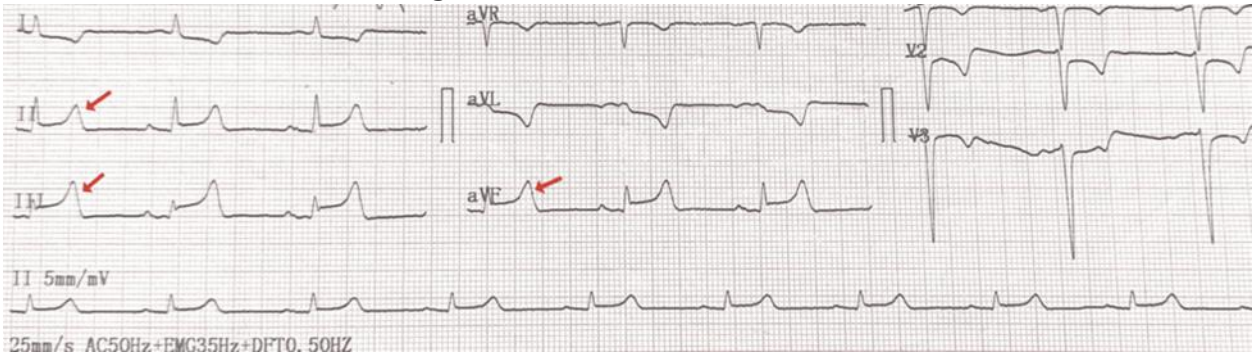
The patient had a history of smoking one pack a day and arterial hypertension, with maximum readings up to 180/100 mmHg, and had undergone an aortic root replacement in 2015 for ascending aortic dissection. The patient has been treating hypertension with Losartan 100mg and Lercanidipine 10mg.

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, aVL.(Figure 1).

Echocardiography Findings

Figure 1: Initial ECG done on admission



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

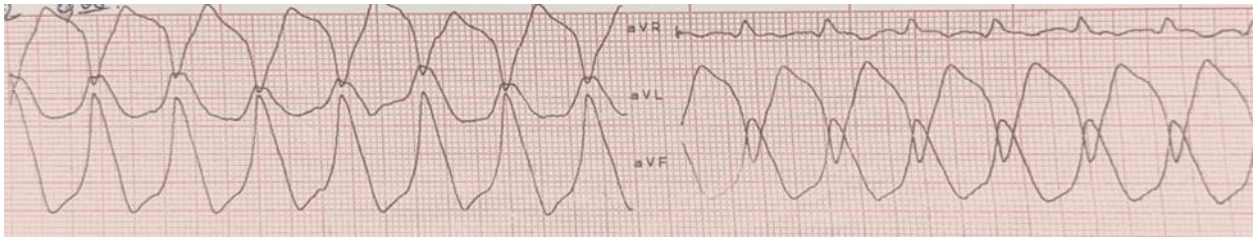
Table 1: Laboratory studies conducted on admission

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.

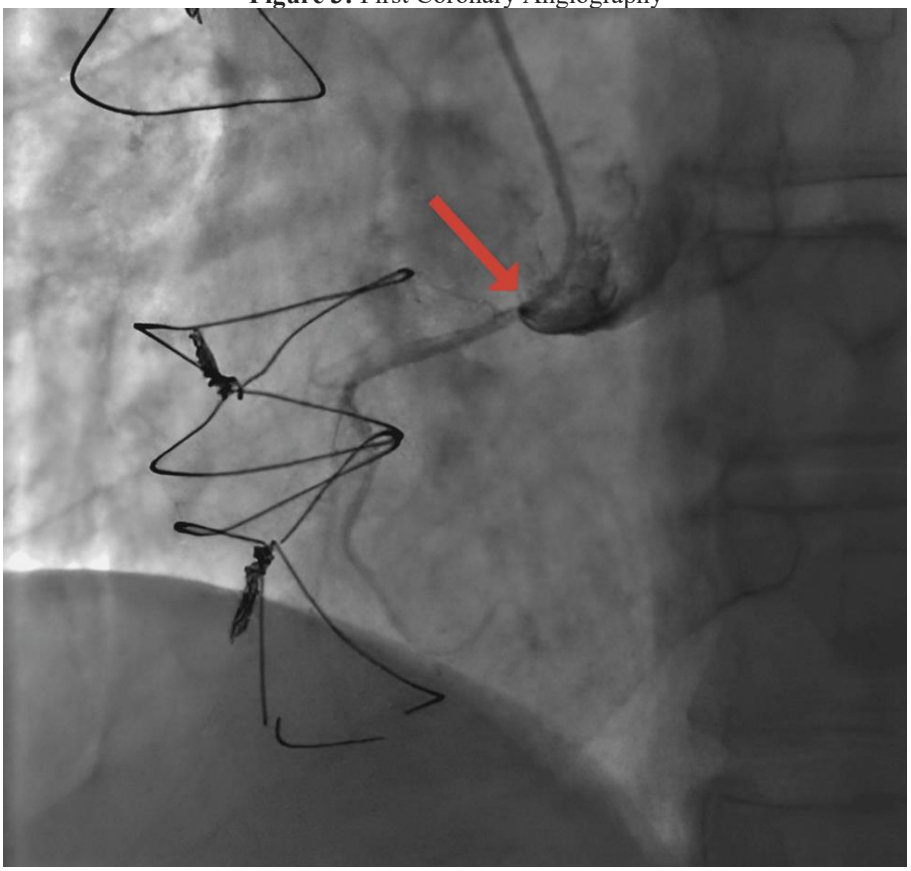
Figure 2: ECG done after the first episode of VT



ECG shows VT.

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-elucidating stent (DES) placement was not necessary due to its small diameter.

Figure 3: First Coronary Angiography



Arrow indicates dissection of hypoplastic right coronary artery

Further Interventions and Course

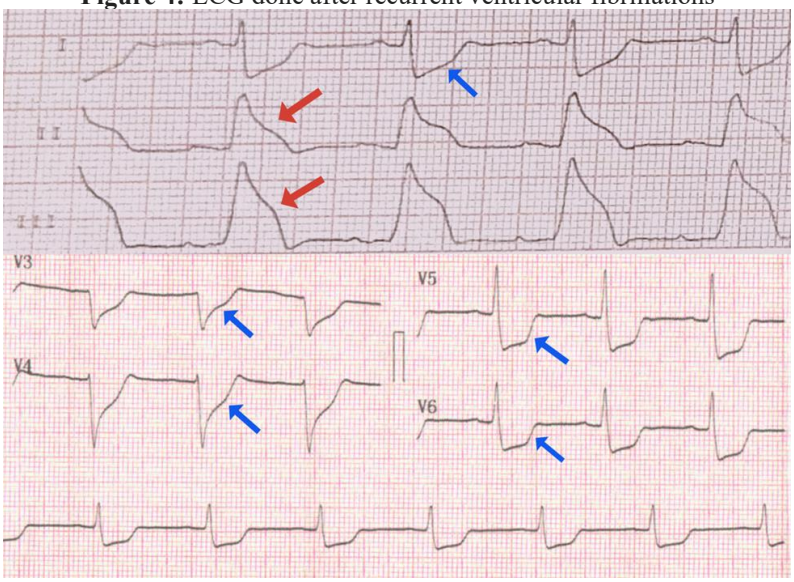
The patient underwent open-heart cardiac surgery for the resection of a false aneurysm of the aortic bulb using a vascular prosthesis. Following surgery, he was placed under observation and remained hemodynamically stable, with a heart rate of 85 bpm, blood pressure of 130/65 mmHg, and oxygen saturation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

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

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

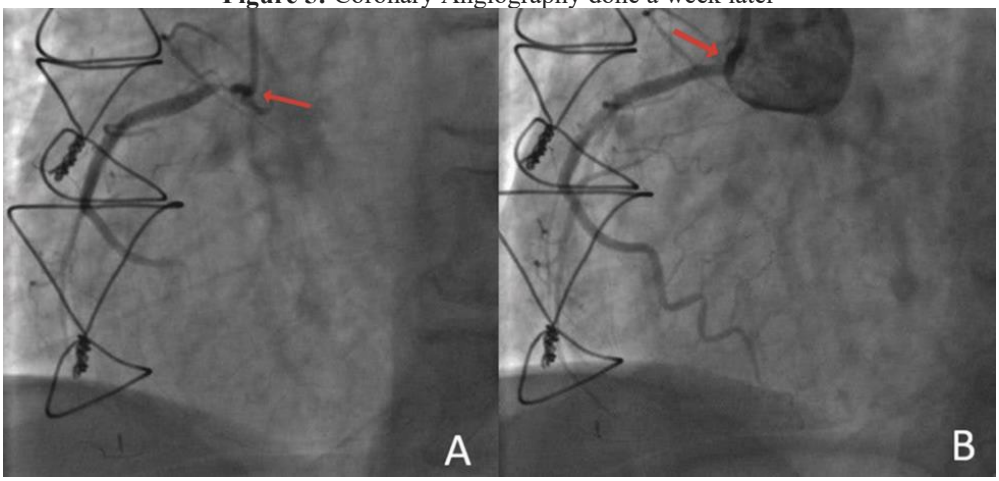


Figure A: shows 90% stenosis in the right coronary artery.
Figure B: RCA angiography after DES placement.

Despite these interventions and aggressive treatment with antiarrhythmics 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 as the underlying cause, and coronary angiography with provocation testing was scheduled.

Subsequent Imaging and Findings

Angiography revealed multiple coronary artery lesions, including a 30-40% stenosis in the LAD, significantly less than what was shown on CT angiography, 90% stenosis in the diagonal artery, and 30-40% stenosis in the LCX. To further assess the cause, acetylcholine provocation testing was performed. This confirmed the presence of vasospasm, with 50-60% stenosis in the LAD and 90% in the LCX, reinforcing the diagnosis of VA. Notably,

LCX spasm triggered ventricular fibrillation (VF) on ECG, supporting the role of vasospasm in the arrhythmic episodes.

Given the association between LCX spasm and VF, DES implantation was performed in both the LCX and LAD arteries to stabilize the condition (Figure 6). While the plaques were not clinically significant, they likely contributed to the vasospasm. The initial episode of VF in the emergency department was also likely caused by vascular spasm.

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

Figure 6: Last coronary angiography

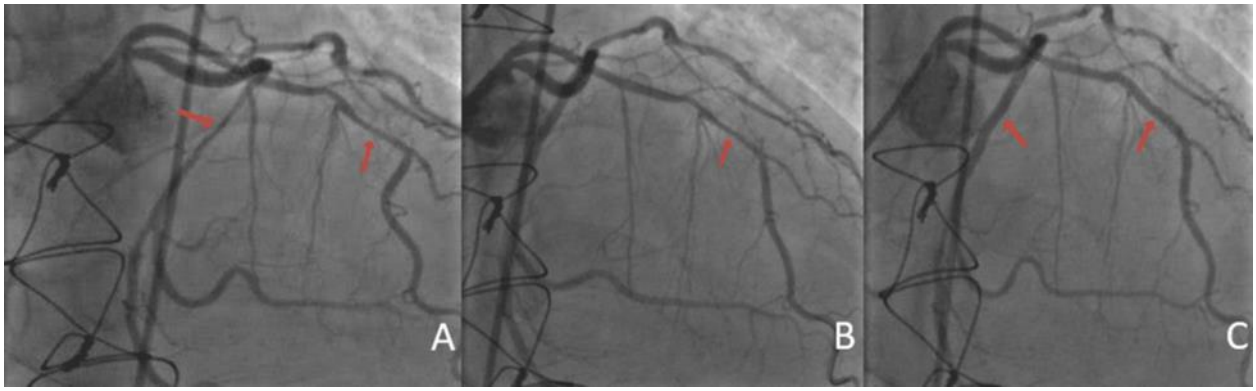


Figure A: Shows spasm of the LAD and LCX arteries; Figure B: Shows spasm of only the LAD artery after the LCX artery was stented; Figure C: Both LAD and LCX arteries have been stented.

The patient continued treatment in the Intensive Care Unit, and within a few hours, he became active, with noticeable 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 per day), rosuvastatin (40 mg per day), verapamil (80 mg twice per day), perindopril (10 mg once per day), and pantoprazole (40 mg once per day).

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 successful patient outcomes.

Discussion

This study presents a rare and severe complication of VA: VF that did not respond to standard pharmacologic treatment with calcium channel blockers and nitrates. While the patient initially showed some

responsiveness, recurrent episodes required definitive intervention with coronary stenting.

A thorough literature review was conducted using Google Scholar and PubMed to identify similar cases and evaluate evidence-based treatment strategies associated with the best patient outcomes. This case highlights the need for further research on patients with VA who fail to respond to conventional therapy.

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

Our review identified multiple reports of refractory VA successfully treated with stent implantation. For example, a similar case of VA complicated by VT was managed with coronary stenting, leading to 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 found 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, V 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 of stenting in VA patients, we reviewed studies evaluating its risks. One study on coronary stenting for severe, pharmacologically refractory coronary artery spasm found no cases of recurrent unstable ischemia requiring hospitalization post-stenting, although restenosis occurred in three patients requiring repeat revascularization (Khatriet al, 2002). These findings suggest that while medical therapy remains the primary treatment, stenting can serve as an adjunct in select cases. However, stent implantation in VA patients carries risks. One study examining stent complications found that stent-edge spasm occurred in 19.2% of cases during follow-up. Although one patient developed acute thrombosis post-stenting, most experienced no immediate complications (Kaku, B et al., 2005).

Given these risks, we also reviewed alternative treatment strategies for refractory VA. One study found that patients with severe or refractory VA often fail to respond to conventional therapy and may develop life-

threatening arrhythmias or sudden cardiac death during ischemic episodes, which are linked to immune-inflammatory responses. The study reported successful remission following treatment with glucocorticoids and immunoglobulin therapy, suggesting a potential alternative approach in select cases (He, Z., 2023).

Conclusions

This case highlights the challenges of managing VA complicated by recurrent ventricular fibrillation (VF) that proved refractory to standard therapy with calcium channel blockers and nitrates. While medical management remains the first-line approach, this case underscores the need for alternative treatment strategies in patients who fail to respond to conventional therapy. The successful resolution of VF episodes following coronary stent placement suggests that DES implantation may serve as a viable option for certain patients with refractory VA, particularly those experiencing life-threatening arrhythmias.

Given the limited data on the long-term efficacy and risks of stenting in VA patients, further research is warranted to better understand its role in preventing recurrent ischemic events and arrhythmias. Additionally, more studies are needed to explore the most suitable clinical scenarios for DES implantation, potential complications, and long-term outcomes in refractory VA. This case adds to the growing body of evidence suggesting that coronary stenting may not only relieve vasospasm but also serve as a definitive treatment for preventing recurrent malignant arrhythmias in select cases.

Conflict of Interest: The authors reported no conflict of interest.

Data Availability: All data are included in the content of the paper.

Funding Statement: The authors did not obtain any funding for this research.

References:

1. Harris, J. R., Hale, G. M., Dasari, T. W., & Schwier, N. C. (2016). Pharmacotherapy of vasospastic angina. *Journal of cardiovascular pharmacology and therapeutics*, *21*(5), 439-451.
2. He, Z., Xu, X., Zhao, Q., Ding, H., & Wang, D. W. (2023). Vasospastic angina: past, present, and future. *Pharmacology & Therapeutics*, *249*, 108500.
3. Kaku, B., Honin, K., Horita, Y., Uno, Y., Yamazaki, T., Funada, A., & Ohka, T. (2005). The incidence of stent-edge spasm after stent

implantation in patients with or without vasospastic angina pectoris. *International Heart Journal*, *46*(1), 23-33.
4. Kawashima, S., & Yokoyama, M. (2004). Dysfunction of endothelial nitric oxide synthase and atherosclerosis. *Arteriosclerosis, thrombosis, and vascular biology*, *24*(6), 998-1005.
5. Khatri, S., Webb, J. G., Carere, R. G., & Dodek, A. (2002). Stenting for coronary artery spasm. *Catheterization and cardiovascular interventions*, *56*(1), 16-20.
6. Khiatah, B., Philips, D., Dukes, J., & Frugoli, A. (2020). Severe prinzmetal's angina inducing ventricular fibrillation cardiac arrest. *Case Reports in Cardiology*, *2020*(1), 3030878.
7. Kleyman, R., Goyal, R., Patel, N., Joseph, J., & Akel, R. (2019). Vasospastic angina and role of cardiac catheterization. *Cureus*, *11*(9).
8. Kusama, Y., Kodani, E., Nakagomi, A., Otsuka, T., Atarashi, H., Kishida, H., & Mizuno, K. (2011). Variant angina and coronary artery spasm: the clinical spectrum, pathophysiology, and management. *Journal of Nippon Medical School*, *78*(1), 4-12.
9. Lanza, G. A., & Shimokawa, H. (2023). Management of coronary artery spasm. *European Cardiology Review*, *18*, e38.
10. Lee, H. C., Hong, S. R., Kim, H. S., Kim, K. C., Choi, W. H., Kang, M. K., ... & Lee, W. R. (2002). The risk factors of vasospastic angina. *Korean Circulation Journal*, *32*(3), 224-232.
11. Lin, Z., Lin, X., Zhao, X., Xu, C., Yu, B., Shen, Y., & Li, L. (2022). Coronary artery spasm: risk factors, pathophysiological mechanisms and novel diagnostic approaches. *Reviews in cardiovascular medicine*, *23*(5), 175.
12. Martí, V., Ligero, C., García, J., Kastanis, P., Guindo, J., & de Rozas, J. M. D. (2006). Stent implantation in variant angina refractory to medical treatment. *Clinical Cardiology: An International Indexed and Peer-Reviewed Journal for Advances in the Treatment of Cardiovascular Disease*, *29*(12), 530-533.
13. Mayer, S., & Hillis, L. D. (1998). Prinzmetal's variant angina. *Clinical cardiology*, *21*(4), 243-246.
14. Ono, M., Okabe, T., Isomura, N., & Ochiai, M. (2024). Successful treatment of vasospastic angina refractory to various therapies using stenting: A case report. *Journal of Cardiology Cases*, *30*(3), 97-100.
15. Picard, F., Sayah, N., Spagnoli, V., Adjedj, J., & Varenne, O. (2019). Vasospastic angina: a literature review of current evidence. *Archives of cardiovascular diseases*, *112*(1), 44-55.
16. Rehan, R., Weaver, J., & Yong, A. (2022). Coronary vasospastic angina: a review of the pathogenesis, diagnosis, and management. *Life*, *12*(8), 1124.

17. Saito, Y., Saito, Y., Kato, K., & Kobayashi, Y. (2022). Gender differences in factors associated with vasospastic angina. *International Journal of Cardiology*, *349*, 7-11.
18. Song, J. K. (2018). Coronary artery vasospasm. *Korean circulation journal*, *48*(9), 767.
19. Vanhoutte, P. M. (2003). Endothelial control of vasomotor function from health to coronary disease. *Circulation journal*, *67*(7), 572-575.
20. Yoo, S. Y., & Kim, J. Y. (2009). Recent insights into the mechanisms of vasospastic angina. *Korean circulation journal*, *39*(12), 505-511.