An aborted sudden cardiac death in a case of prinzmetal angina.

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Abstract

Prinzmetal Angina (PzA) is characterized by recurrent episodes of chest pain owing to coronary artery vasospasms. These episodes are associated with electrocardiographic evidence of transient ST segment elevations. Although rare, patients with PzA can have ventricular arrhythmias and Sudden Cardiac Death (SCD), even in the absence of any risk factors for SCD. We herein report a case of a 47-year-old female, who is a known case of PzA, on adequate medical therapy and still having recurrent episodes of chest pain, which were complicated by sudden cardiac arrest, requiring an ICD in view of preventing future risk of SCD.

Keywords: Sudden cardiac arrest, Prizemetal angina, Vasospastic angina, Cardiac arrest, Implantable cardioverter defibrillator.

Introduction

Prinzmetal angina (PzA), also known as vasospastic angina, is characterized by localized spasm of one or more coronary arteries that results in transient myocardial ischemia and acute coronary syndrome symptoms without evidence of obstructive coronary artery disease. Recurrent episodes afflict around 5 to 30 percent of individuals [1]. Vasoconstrictor medications, smoking, catecholamine release, redistribution of blood flow, high blood pH, recreational drugs such as cocaine, stress, cold weather, and exercise can all be responsible for these episodes [2]. PzA often develops at rest, either in the early hours of the morning or later at night. Patients have intense crushing substernal chest pain during these episodes, and an EKG shows peaked and symmetrical T waves present before ST-segment elevations according to the degree of ischemia and vasospasm. The electrocardiograph returns spontaneously to baseline after a brief period of these fluctuations, which lasts around 15 minutes [3]. According to Coronary Vasomotion Disorders International Study Group (COVADIS) recommendations, PzA may be diagnosed by 24-48 hours of continuous EKG monitoring, nitrate-responsive chest pain, or by inducing vasospasm during angiography with acetylcholine or ergonovine.

While calcium channel blockers and nitrates are often used to treat angina, some patients have spasms that are severe enough to lead to STEMI and put them at risk for ventricular arrhythmia and sudden cardiac death (SCD). A previous history of aborted sudden cardiac arrest, preexisting hyperlipidemia, hypertension, multivessel spasm or left anterior descending artery spasm, and a family history of SCD are the most frequent indicators of higher mortality in these individuals. Despite receiving the best medical care, any episode of PzA may result in ventricular ischemia severe enough to produce a fatal ventricular arrhythmia, making an implanted cardiac defibrillator an essential life-saving intervention [4].

Case Presentation

A 47-year-old female patient with a medical history of Prinzmetal angina, on isosorbide mononitrate and nifedipine, presented as an unconscious individual at her home. Emergency responders initiated cardiopulmonary resucitation CPR and detected ventricular fibrillation (v-fib) during rhythm assessment. After three cycles of defibrillator shocks and administration of amiodarone, the patient's rhythm was successfully restored. She was subsequently intubated and admitted to the Intensive Care Unit (ICU) for further evaluation.

The patient's 12-lead Electrocardiogram (EKG) revealed ST-segment Elevation Myocardial Infarction (STEMI), and her troponin I level was elevated (807 ng/L). Initial Arterial Blood Gas (ABG) analysis showed no remarkable findings, and an emergency echocardiogram demonstrated normal left ventricular ejection fraction. Post-CPR chest X-ray appeared normal, and urine toxicology screening yielded negative results. Computed Tomography Pulmonary Angiography (CTPA) was performed to rule out pulmonary embolism, which came back negative. Additionally, a non-contrast head computed tomography scan showed no abnormalities to rule out neurological causes of unconsciousness.

The patient was proceeded to be further investigated for cardiac causes with a coronary angiography revealing no presence of atherosclerotic disease in any of the coronary arteries. During this series of investigations and time, the patient became agitated and decided against medical advice to leave. Subsequently, on the same evening, she presented to the Emergency Room (ER) with symptoms of chest pain, dyspnea, palpitations, and lightheadedness. A 12-lead EKG was performed, demonstrating normal sinus rhythm, while the cardiac troponin level was measured at 18 ng/L. Considering

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the patient's recent history of CPR, we suspected the chest pain and elevated troponin level could be attributed to this event. A chest CT was decided to be conducted to rule out any missed fractures by the previous chest x-ray, but it was non-yielding.

Although the patient received analgesics in the ER, the chest pain persisted. Consequently, she was admitted to the hospital for further evaluation. Upon reviewing her medical records and current complaints, we concluded that the patient experienced episodes of vasospastic angina leading to sudden cardiac arrest. In light of this finding, we recommended implantation of an Implantable Cardioverter-Defibrillator (ICD) for the patient. In the interim, a wearable cardioverter defibrillator was ordered.

During her hospital stay, the patient reported experiencing chest pressure lasting approximately 10-15 minutes, accompanied by a sensation of a strong and rapid heartbeat. An EKG performed during this episode revealed ST elevation in leads II and III, along with ST depression in leads I and aVL. The patient resumed taking her medications, including nifedipine and metoprolol. After stabilizing her chest pain, a repeat EKG conducted after 20 minutes showed no ST elevation. Subsequently, The patient successfully underwent subcutaneous ICD implantation and defibrillation threshold testing for secondary prevention of sudden cardiac death. Regular follow-up visits were scheduled every three months, during which no additional episodes of shockable rhythm were detected during the device check.

Discussion

Prinzmetal Angina (PzA), is characterized by recurrent chest pain resulting from vasospasm of the coronary arteries. The spasm can be caused by two main mechanisms: decreased blood flow due to endothelial dysfunction and/or abnormal contraction of vascular smooth muscle cells [5]. The pain can occur during rest or exertion and is not triggered by exercise. It is associated with ST-segment elevation on ECG and is responsive to sublingual nitroglycerin.

The coronary artery spasm underlying the condition can lead to stable angina, unstable angina, myocardial infarction, arrhythmias, or sudden cardiac death. ECG monitoring typically shows fixed spasms in specific vessels, although variations can occur. Arrhythmias are dependent on the affected area, such as ventricular tachycardia resulting from spasms of the left anterior descending artery. Fatal ventricular arrhythmia incidence is < 10% but clinically significant as it leads to SCD. Risk factors for sudden cardiac arrest in vasospastic angina include age, family history of sudden cardiac death, multivessel spasm, chronic hypertension, and dyslipidemia . While a positive family history of coronary artery disease is related to the frequency of vasospastic chest pain, some patients, including those without a family history, may experience multiple episodes of chest pain [6]. The significance of coronary spasms in this disease is greater than those associated with coronary plaque, as it results in a higher degree of stenosis and an increased risk of sudden cardiac death [7]. Genetic etiologies such as Brugada syndrome and prolonged QT intervals should also be ruled out in patients with a family history of sudden cardiac death.

PzA is differentiated from stable angina as it is not commonly triggered by an increase in myocardial oxygen demand. The underlying pathogenesis involves vascular endothelial dysfunction and decreased endothelium-dependent vasodilation due to deficient release of nitric oxide from endothelial NO synthase. Another proposed theory involves vascular smooth muscle hyper reactivity due to changes in the Rho/Rho kinase pathway, resulting in increased activity of myosin light chain kinase and contraction of vascular smooth muscle cells. Causative factors for vasospasm include smoking, increased oxidative stress, magnesium deficiency, dyslipidemia, abnormal glucose metabolism, sympathomimetic agents, hyperventilation, and certain genetic factors [8].

The diagnosis of PzA can be made based on the COVADIS criteria, which include nitrate-responsive chest pain, continuous ECG monitoring showing transient changes during a spontaneous episode (ST-segment elevation $\geq$ 0.1 mV, ST segment depression $\geq$ 0.1 mV, or new negative U waves), and coronary artery spasm provoked (by hyperventilation, acetylcholine [shorter half-life], or ergonovine) or spontaneous on coronary angiography. Additional markers, such as C-reactive protein and Rho-kinase activity in circulating leukocytes, can further support the diagnosis.

Specifically addressing the subset of patients with PzA complicated by a ventricular arrhythmia, Calcium Channel Blockers (CCBs) demonstrate insufficient efficacy in preventing episodes of angina and subsequent arrhythmias. Moreover, predicting future occurrences of ventricular arrhythmias in this population is challenging. As exemplified in our patient, who initially experienced Ventricular Fibrillation (VF) leading to sudden cardiac death (SCD) and subsequently had another anginal episode within a short period, we hypothesize that an augmented sympathetic response may contribute to post-extubation vasospastic chest pain. MacAlpin demonstrated that angina accompanied by syncope or arrhythmias triples the risk of fatal episodes and carries a poor prognosis in PzA patients. For these patients, Implantable Cardioverter-Defibrillator (ICD) placement has been established as an effective intervention for safeguarding against life-threatening arrhythmias. General guidelines for ICD implantation recommend considering it for patients who survive cardiac arrest due to ventricular fibrillation or those with hemodynamically unstable ventricular tachycardia after excluding any reversible causes. In PzA patients, two approaches are proposed: Meisel et al. suggest considering ICD placement as secondary prevention for patients who continue to experience symptoms despite optimal medical therapy, while Matsue et al. suggest considering ICD placement for asymptomatic PzA patients who have experienced a ventricular arrhythmia as secondary prevention. This recommendation is based on their study, where patients reported no preceding chest pain or discomfort prior to the occurrence of fatal ventricular arrhythmias that were terminated by the ICD. However, it should be noted that ICDs implanted in PzA patients with multivessel involvement do not prevent episodes of pulseless electrical activity and necessitate further interventions. Sole reliance on ICD therapy is insufficient if

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Table 1. *Summary of cases reported in the literature.*

Sr. No	Age/gender	Presentation at diagnosis	Comorbidities	Medication given	ICD Placed	Follow up	Reference
1	48/F	SCD	Renal agenesis	N/A	Yes	N/A	[1]
2	60/Transgender	Chest pain	NHL, HL,	N/A	N/A	N/A	[2]
3	60/M	Unresponsive	HTN	After: Verapamil, Nitroglycerin, DAPT	Yes	Asymptomatic at 3 months follow up	[8]
4	51/F	Chest pain + severe dizziness	Wolff-Parkinson-White syndrome	Before: Nicorandil and Diltiazem After: Diltiazem, Benidipine HCL, Nicorandil	No	No adverse cardiac events at 6 month follow up	[10]
5	43/F	Chest pain + dyspnea leading to SCD	Acid reflux, Postnatal depression	After: CCB and nitrates + drug-eluting stent	Yes	Asymptomatic at 3 month follow up	[11]
6	46/F	SCD	HTN, CAD	Before: Aspirin, Isosorbide mononitrate and Diltiazem After: Isosorbide mononitrate and Diltiazem	Yes	At 6 months SCD due to multiple ventricular tachycardia episodes and unrevivable ICD shocks	[12]
7	66/M	Chest pain	HTN, Type II Diabetes, CKD (Stage 3), OSA	Before: Aspirin, Isosorbide mononitrate and nitroglycerin After: Atorvastatin, isosorbide mononitrate, nifedipine, and furosemide	Yes	N/A	[13]
8	27/M	Chest pain	N/A	After: BMS in RCA, calcium channel blockers, nitrates, DAPT→CABG in RCA →DES in LAD	Yes	N/A	[14]
9	53/F	Collapse and VF arrest	HTN, COPD, Asthma, TIA, smoking, obesity	Before: Atorvastatin and aspirin. After: Diltiazem and long acting nitrates	Yes	No chest pain, ECG changes or arrhythmia at follow up	[15]
10	64/M	Syncope, sinus tachycardia with AVB and nonsustained PVT	N/A	After: Diltiazem and long acting nitrates	Yes	Asymptomatic at 4 months follow up	[16]
11	38/F	Chest pain, palpitations and lightheadedness	Smoking, HLD, asthma, psoriasis	After: calcium channel blockers, nitrates	Yes	Recurrent PVT terminated with ICD discharges. Asymptomatic after CCB and nitrates dose was maximised	[17]

*NHL- Non Hodgkin Lymphoma, HL- Hodgkin Lymphoma, SCD- Sudden Cardiac Death, DAPT - Dual antiplatelet therapy, CKD- Chronic Kidney Disease, HTN - Hypertension, CAD- Coronary Artery Disease, OSA- Obstructive Sleep Apnea, CABG- Coronary Artery Bypass Graft, DES- Drug Eluting Stent, VF- ventricular fibrillation, COPD- chronic obstructive pulmonary disease, TIA - Transient ischemic attack, AVB- atrioventricular block, PVT-polymorphic ventricular tachycardia, HLD- hyperlipidemia.

left ventricular function declines due to ischemia; therefore, it is crucial to employ maximal medical therapy for vasospasm control in conjunction with ICD placement. Discontinuation or dose reduction of medications has been associated with an increased risk of recurrent ventricular arrhythmias [9]. We have done a literature review of similar cases and compiled them in Table 1.

Conclusions

In conclusion, this case report highlights the challenges in managing Prinzmetal Angina (PzA) complicated by ventricular arrhythmias. The patient presented with sudden cardiac arrest due to ventricular fibrillation, likely triggered by coronary artery spasm. Despite initial successful resuscitation, the patient experienced recurrent episodes of vasospastic angina and elevated troponin levels. Implantable Cardioverter-Defibrillator (ICD) placement was recommended as secondary prevention for life-threatening arrhythmias. Regular follow-up visits showed no additional episodes of shockable rhythm during device checks. The case underscores the importance of a comprehensive treatment approach involving maximal medical therapy for vasospasm control and ICD placement in high-risk PzA patients to prevent sudden cardiac death.

References

1. Khiatah B, Philips D, Dukes J, et al. Severe prinzmetal's angina inducing ventricular fibrillation cardiac arrest. Case Rep Cardiol. 2020;2020.
2. Lindsay PJ, Frank RC, Bittner EA, et al. ST elevations and ventricular tachycardia secondary to coronary vasospasm upon extubation. Case Rep Anesthesiol. 2020;2020.
3. de Luna AB, Cygankiewicz I, Baranchuk A, et al. Prinzmetal angina: ECG changes and clinical considerations: a consensus paper. Ann Noninvasive Electrocardiol. 2014;19(5):442-53.
4. Kundu A, Vaze A, Sardar P, et al. Variant angina and aborted sudden cardiac death. Curr Cardiol Rep. 2018;20:1-6.

Citation: *Yadav V, Ashraf H, Hussain AT, et al. An aborted sudden cardiac death in a case of prinzmetal angina. Arch Gen Intern Med. 2023;7(5):191*

5. Sanjeeva NC, Shetty RK, Agarwal S. An atypical case of vasospastic angina: demonstrating the usefulness of Holter monitoring. Case Rep. 2015;2015:bcr2015210939.
6. Teragawa H, Oshita C, Orita Y. Clinical significance of prolonged chest pain in vasospastic angina. World J Cardiol. 2020;12(9):450.
7. Meisel SR, Mazur A, Chetboun I, et al. Usefulness of implantable cardioverter-defibrillators in refractory variant angina pectoris complicated by ventricular fibrillation in patients with angiographically normal coronary arteries. Am J Cardiol. 2002;89(9):1114-6.
8. Jiang M, Kaplan RM, Peigh G, et al. Vasospastic arrest: a heart-stopping case of prinzmetal angina. Case Rep. 2020;2(4):611-4.
9. Matsue Y, Suzuki M, Nishizaki M, et al. Clinical implications of an implantable cardioverter-defibrillator in patients with vasospastic angina and lethal ventricular arrhythmia. J Am Coll Cardiol. 2012;60(10):908-13.
10. Okabe T, Kitakata H, Iwahori H, et al. Vasospastic angina resulting in sudden cardiac arrest, initially misdiagnosed as a psychiatric disorder. J Cardiol Cases. 2015;11(5): 150-4.
11. Naqvi SY, Hanley A, Crowley J. Ventricular fibrillation due to coronary vasospasm. Case Rep. 2014;2014:bcr2013203253.
12. Riaz S, Raj V, Shah S. Refractory Vasospastic Angina and Sudden Cardiac Arrest: Is Implantable Cardioverter Defibrillator Indicated and Is It Always Protective?. Cureus. 2020;12(8).
13. Ozdemir D, Kishor J, Hall JM, et al. A Rare Case of Vasospastic Angina Presenting with Inferior Lead ST-segment Elevation and Ventricular Fibrillation in the Absence of Coronary Obstruction: A Case Report. Cureus. 2019;11(12).
14. Nishtar SS, Ali T, Azeem T, et al. Refractory vasospastic angina causing myocardial infarction and VF arrest requiring combined medical, electrophysiological, percutaneous and surgical intervention—A case report. Int J Cardiol. 2007;119(1):e4-7.
15. Adepoju D, Smith JA, Leslie SJ. Life-Threatening Coronary Vasospasm Treated by Implantable Cardioverter Defibrillator: The Warning Signs. Case Rep Cardiol. 2022;2022.
16. Guzzo-Merello G, Romero-Tejero C, García-Cosío F, Martínez-Elbal L. Indication for defibrillator implantation in a patient with Prinzmetal's angina and syncopes. Rev Esp Cardiol. 2009;62(7):820-1.
17. Bhat PK, Quan KJ. Implantable cardioverter defibrillator (ICD) for polymorphic ventricular tachycardia (VT) due to coronary vasospasm. J Hosp Med. 2009;4(6):E23-5.

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