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Case Reports

From Benign Origins to Final Moments: Right Ventricular Outflow Tract Premature Ventricular Complexes Culminating in Asystole on Holter Monitoring

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Abstract

Background: Premature Ventricular Complexes (PVCs) are a common cardiac arrhythmia, typically of a benign nature. Their origin in the right ventricular outflow tract (RVOT) is often of interest due to its implications for treatment strategies. While the vast majority of PVC RVOT cases remain uneventful, isolated incidents challenge this common perception. Continuous monitoring methods, such as the Holter monitor, have provided invaluable insights into the real-world dynamics of arrhythmias, capturing rare events that can be of paramount clinical significance.

Case Illustration: A 60-year-old female presented to Harapan Kita Hospital Jakarta, in May with palpitations. Over several visits, physical examinations consistently indicated a heart within normal parameters, free of murmurs or gallop. Successive ECGs revealed persistent PVCs of RVOT origin. Despite medical intervention, her arrhythmic pattern persisted. By September, her symptoms had diversified, including occasional chest pain, nausea, and dyspnea. An ECG, yet again, confirmed PVCs with RVOT origin. During a Holter monitoring session in September, a distressing sequence of events was captured. The monitor initially registered a non-sustained Ventricular Tachycardia (VT), which escalated to sustained VT, ventricular fibrillation, and culminated in asystole, marking the patient's final moments.

Conclusions: The pathophysiological journey from benign PVCs of RVOT origin to a fatal arrhythmic event underscores the unpredictability and inherent dangers of cardiac arrhythmias. This case is a reminder of the critical importance of persistent monitoring, timely interventions, and the nuanced understanding of conditions conventionally deemed 'benign'.

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Keywords: Premature Ventricular Complex, Right Ventricular Outflow Tract, Ventricular Tachycardia, Sudden Cardiac Death, Holter Monitoring

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Introduction

Ectopic beats originating from the ventricles are identified as Premature Ventricular Complexes (PVCs). These are prevalent and can manifest in numerous clinical contexts and among a broad spectrum of individuals.[1-3] In the past, PVCs were generally viewed as harmless. While some patients may not exhibit any symptoms, others may present acute symptoms directly related to PVCs or gradual symptoms resulting from the sustained impact of frequent PVCs on the heart's ability to contract.[2,4] PVCs are widely distributed, with most individuals demonstrating their presence during extended ambulatory assessments.[5-6] Factors like advancing age, greater stature, elevated blood pressure, prior heart conditions, decreased physical activity, and smoking habits tend to increase the likelihood of PVC occurrence.[1] While the core origins of PVCs are not definitively understood, potential triggers might encompass spontaneous activity, inherent automaticity, or reentrant pathways. Often, these PVCs might go unnoticed, but they can also manifest through symptoms like heart palpitations, shortness of breath, near fainting, and fatigue.[4-5]

Diagnosing and evaluating PVCs critically relies on a combination of patient history, physical examination, and 12-lead ECG. In instances where there are explicit symptoms or an unusually high frequency of PVCs, an echocardiogram is recommended.[6] Further, cardiac magnetic resonance imaging becomes pivotal when there's an indication of accompanying structural heart anomalies. To gauge the regularity of PVCs, ambulatory monitoring becomes indispensable.[3] The long-term outlook for individuals with PVCs ranges widely. Specific key indicators that might predict unfavourable outcomes remain ambiguous. However, a high frequency of PVCs can potentially lead to heart complications or even mortality.[7]

Case Illustration

A 60-year-old female, weighing 53 kg and standing at a height of 147 cm, initially presented to the National Cardiovascular Center Harapan Kita Jakarta with palpitations. Physical examinations revealed normal heart sounds without any murmur or gallop. Pulmonary auscultation was vesicular with no signs of wheezing or rhonchi. The ECG identified PVCs, as showcased in Figure 1, which illustrated a PVC with QRS morphologies of Left Bundle Branch Block (LBBB) and inferior axis, suggesting an origin from the Right Ventricular Outflow Tract (RVOT) on sinus rhythm.

On her follow-up visits, the patient presented

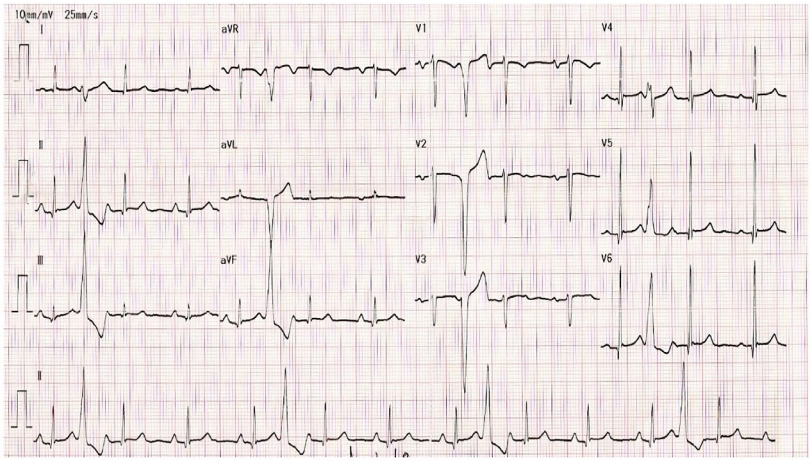

Figure 1. Electrocardiogram displaying PVC originating from RVOT on sinus rhythm.

with a stable condition. The RVOT-origin PVCs remained consistent on her ECG. The treatment strategy at this time was optimized medical treatment. However, during one visit, the patient reported occasional epigastric pain, nausea, and shortness of breath, but no palpitations. Physical examinations remained consistent with previous results. The ECG still displayed PVCs of RVOT origin. Given these findings, a Holter monitoring was planned along with the scheduling of ablation. During the Holter monitoring, a catastrophic event occurred. The patient experienced an episode of nonsustained VT, as shown in Figure 2, transitioning from sinus rhythm to nonsustained monomorphic VT and then to sustained monomorphic VT. The situation continued, transitioning from monomorphic VT to Ventricular Fibrillation (VF) and finally to asystole, as shown in Figure 3 and Figure 4.

It is worth noting that a year before the first reported episode, the patient was referred with a history of monomorphic VT. Her complaints were intermittent palpitations without any associated syncope. Past medical history and risk factors were non-contributory. Physical examinations revealed normal heart and lung findings. The ECG at that time demonstrated frequent PVCs originating in the RVOT.

She was planned for VT ablation; however, at the beginning of the ablation procedure, she remained asymptomatic. Notably, baseline ECG showed no PVC or VT. Provocative agents Isuprel up to 2 mcg/kg/min and epinephrine up to 0.15 mcg/kg/min failed to induce any PVC or VT during the session. After 20 minutes of observation, no PVCs reappeared, and the planned ablation was cancelled. The discharge medications included Concor 1.25 mg, Candesartan 8 mg, Simvastatin 20 mg, and Miniaspi 80 mg. Then, the patient was advised to schedule a follow-up and continue her medications as prescribed.

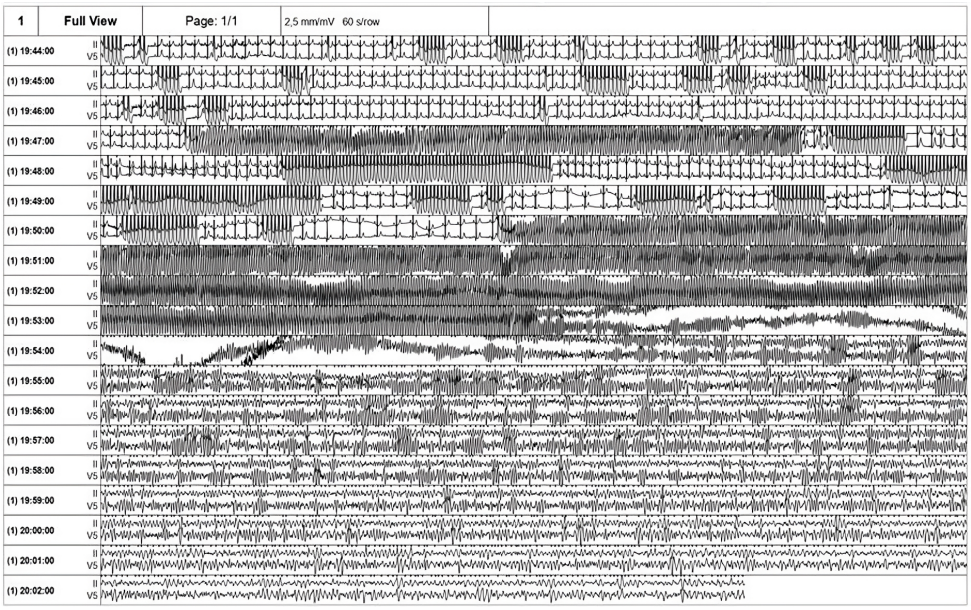

Figure 2. Electrocardiographic progression from sinus rhythm to non-sustained monomorphic VT and subsequently into sustained monomorphic VT.

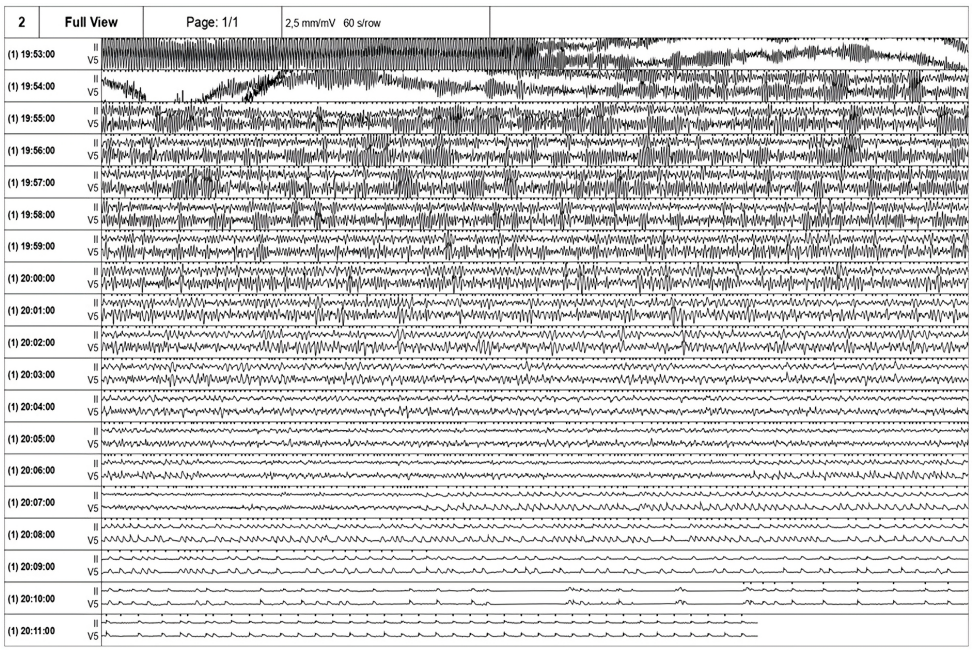

Figure 3. Transition from monomorphic VT to polymorphic VT, ventricular fibrillation, and bradycardia.

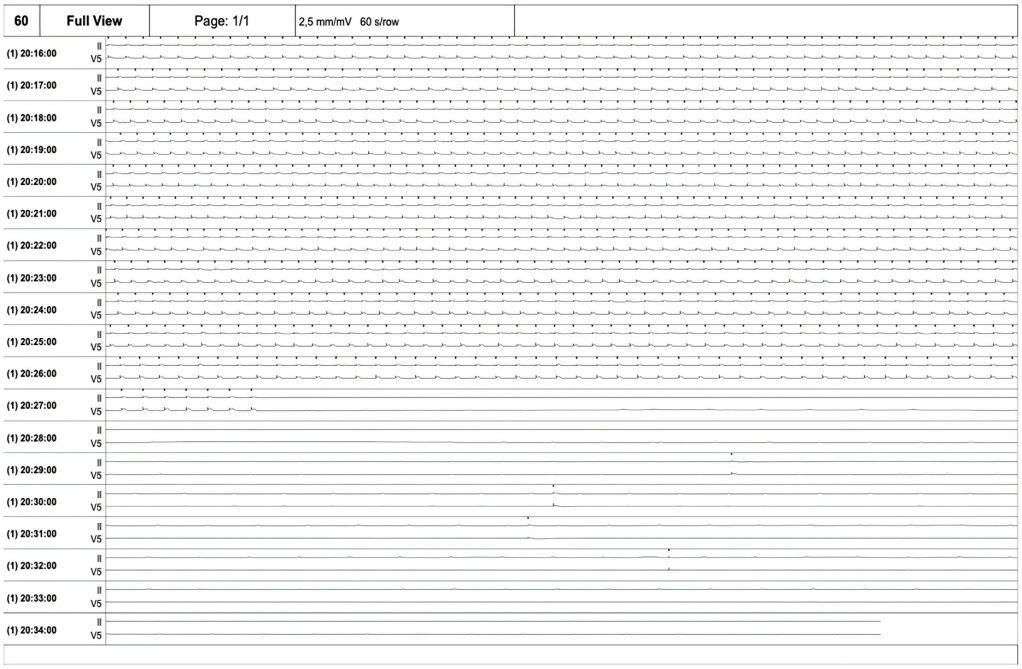

Figure 4. Transition from bradycardia to asystole.

Following the cancelled ablation, the patient was discharged on bisoprolol 1.25 mg, candesartan 8 mg, simvastatin 20 mg, and aspirin 80 mg, and was advised regular follow-up. During this period, she continued to experience intermittent palpitations without syncope. Several months later, her symptoms evolved to include epigastric pain, nausea, and dyspnea, while her ECG consistently showed PVCs of RVOT origin. During a Holter monitoring session, these arrhythmias progressed from non-sustained VT to sustained VT, then VF, and ultimately asystole, resulting in her death.

This case exemplifies the deceptive nature of RVOT-origin PVCs, which are often regarded as benign. The patient's trajectory from presenting with simple palpitations to a critical asystole episode captured on Holter, alongside the detailed figures, emphasizes the importance of vigilant monitoring and aggressive intervention when indicated. The progression displayed in the statistics is rare on continuous Holter monitoring, underscoring the crucial and invaluable insights it can offer, even when the patient remains asymptomatic.

Discussion

Holter monitoring is a valuable tool of assessing PVCs, providing insights into their frequency, burden, and potential risks.[8] This case highlights the significance of Holter monitoring findings, particularly when PVCs originate from the RVOT, and underscores the importance of continuous surveillance in patients with seemingly benign PVCs.

Benign PVCs are typically considered harmless, but in rare cases, they can lead to life-threatening arrhythmias.[8] Literature reports have documented extreme outcomes associated with PVCs of RVOT origin, including ventricular fibrillation and sudden cardiac death.[9-10] Although such events are infrequent, they remind us of the need for vigilance when managing patients with PVCs, even when these arrhythmias appear benign.

In this case, although the initial ventricular arrhythmia was monomorphic VT consistent with RVOT origin, the Holter strip demonstrated a transition to polymorphic VT, possibly torsades-like, likely precipitated by an R-on-T phenomenon. This highlights the potential for PVCs to trigger polymorphic VT through afterdepolarization mechanisms. The recording concluded with electrical silence consistent with asystole. This evolution underscores the complexity and unpredictability of PVC-related arrhythmogenesis.

The transition from benign PVCs to fatal arrhythmias remains a subject of investigation. In some patients, underlying structural heart disease or genetic factors might play a role.[11-12] PVCs occurring in patients with a history of Myocardial Infarction (MI) have long been associated with poor prognosis, due to increased risk of VT, VF, and sudden cardiac death in this group. Moreover, growing studies observed beneficial outcomes of cardiac ablation in patients with a high PVC burden and decreased left

ventricular function with structural heart diseases.[13] Additionally, the excessive burden of PVCs, as observed on Holter monitoring, can lead to electrical and mechanical remodelling of the myocardium, potentially precipitating arrhythmogenic events.[14] Furthermore, frequent occurrences of PVCs also increase the likelihood of developing PVC-induced cardiomyopathy, whether or not there is pre-existing structural heart disease.[13,15] According to a study by Keles et al., patients with frequent PVCs showed lower early diastolic strain rate than healthy individuals, increasing the risk of left ventricular diastolic dysfunction.[16] The origin of the PVC has also been postulated to affect the risk of developing cardiomyopathy.[14] In a study with a median follow-up time of 5.2 years, the mortality rate in the PVC group was 5.7 per 1000 person-years, significantly lower than the 11.9 per 1000 person-years observed in the control group of the general population without PVC in the absence of structural heart disease.[17]

High-risk PVC morphologies are characterized by short coupling intervals, multifocal origin, or the ability to trigger R-on-T phenomena, all of which predispose to malignant arrhythmias. In our patient, the progression from monomorphic RVOT PVCs to polymorphic VT was most likely facilitated by an R-on-T event, placing this case within the high-risk spectrum despite the initially benign morphology. This highlights the importance of recognizing PVC characteristics that may signal increased arrhythmic risk. Short coupling intervals of 350 ms are considered a high-risk feature.[18] However, in our case, we found that longer coupling intervals, as shown in Figure 5, caused torsades due to the R-on-T phenomenon, whereas shorter coupling intervals, as shown in Figure 6, did not. This underlines the need to re-evaluate the threshold for defining malignant PVC coupling intervals.

Managing patients with benign-seeming PVCs involves a multidimensional approach. Reassurance is often sufficient, but it is crucial to assess the impact of symptoms on a patient's quality of life.[5] In cases where symptoms persist or worsen, medical therapy or catheter ablation may be considered.[2,9] Beta-blockers and calcium channel blockers are commonly used as first-line medications, with catheter ablation demonstrating superior effectiveness.[10] However, the choice between medical therapy and ablation should be guided by patient preference and individual characteristics.

This case offers several lessons for clinicians. First, it underscores the importance of continuous monitoring and thorough examinations in patients with PVCs, even when these arrhythmias initially appear benign. Second, it highlights the need for risk stratification to identify those at higher risk of adverse outcomes.[11] Finally, it emphasizes the evolving nature of PVC management, with ongoing research providing insights into better strategies and interventions.

While this case study provides valuable insights, it has its limitations. The rarity of extreme outcomes in patients with benign PVCs makes it challenging to draw definitive conclusions. Additionally, the case's retrospective nature limits our ability to establish

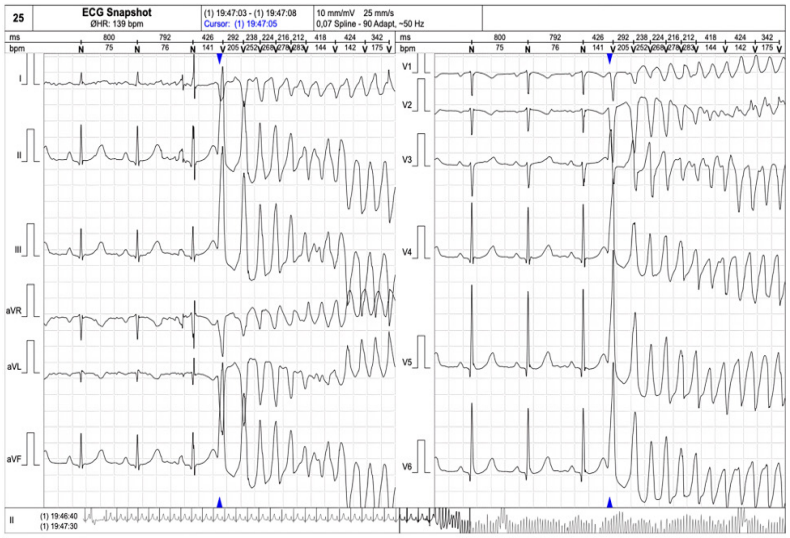

Figure 5. PVC with coupling interval of 426 ms induces Torsades de pointes.

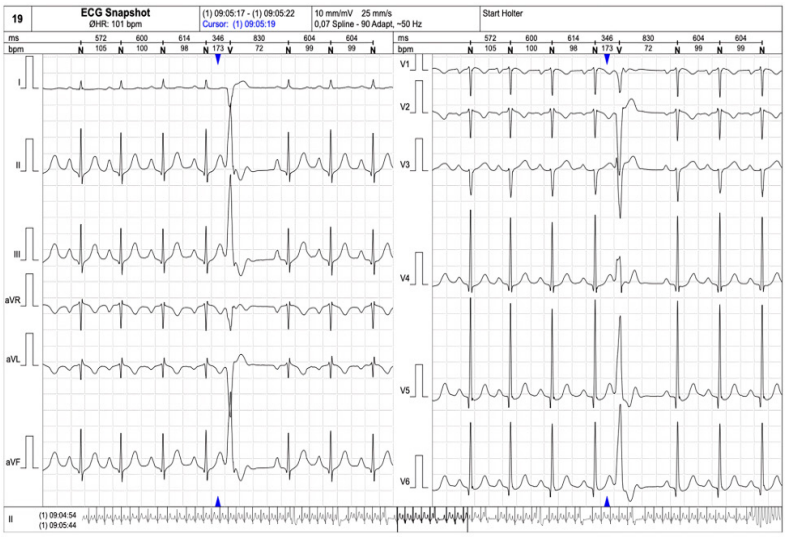

Figure 6. PVC with coupling interval of 346 ms.

causal relationships and generalize findings. There was a lack of evidence to confirm the absence of preexisting structural heart disease in the patient. A significant limitation of this report is the absence of echocardiographic data and a treadmill test, which would have been essential to exclude structural or ischemic heart disease and to assess the potential indication for ICD implantation. This absence restricts the ability to characterize the patient's substrate fully and highlights the need for comprehensive evaluation in similar cases.

Conclusion

In conclusion, this case of benign PVCs ultimately leading to asystole on Holter monitoring serves as a stark reminder of the potential risks associated with these seemingly harmless arrhythmias. Although PVCs are often regarded as benign, especially those originating from the RVOT, they can evolve into life-threatening arrhythmias, as demonstrated in this patient. This case exemplifies the complexity of PVC management and underscores the importance of ongoing advances in diagnostic tools and therapeutic strategies.

List of Abbreviations

ECG	Electrocardiogram
ICD	Implantable Cardioverter Defibrillator
LBBB	Left Bundle Branch Block
MI	Myocardial Infarction
PVC	Premature Ventricular Complex
R-on-T	R-on-T Phenomenon
RVOT	Right Ventricular Outflow Tract
Torsades	Torsades de Pointes
VF	Ventricular Fibrillation
VT	Ventricular Tachycardia

Ethical Clearance

Not Applicable.

Publication Approval

All authors approve the manuscript for publication.

Authors Contributions

SBR was the main author who conceptualized the case, provided clinical supervision, and made critical contributions to the manuscript's writing and final revision. SVR and AP assisted with literature searching and manuscript writing. DAH, DYH, and YY provided valuable clinical input, reviewed the manuscript, and offered additional insights on the case details and clinical implications discussed in the manuscript.

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Conflict of Interest

The authors declare no conflicts of interest.

Availability of Data and Materials

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Generative AI and AI-Assisted Technologies in the Writing Process

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