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CLINICAL CASE

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# Catheter ablation of frequent premature ventricular complexes: curative strategy for symptom relief

## *Ablación con catéter de complejos ventriculares prematuros frecuentes: una estrategia curativa para el alivio de los síntomas*

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**Keywords:**
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***Palabras clave:***
*complejos ventriculares prematuros, arritmias ventriculares, ablación con catéter, miocardiopatías.*

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### ABSTRACT

**Introduction:** premature ventricular complexes (PVCs) are linked to PVC-induced cardiomyopathy, functional deterioration, and poor quality of life (QOL). **Presentation of case:** a 54-year-old woman presented for evaluation with dyspnea and palpitations as her primary complaints. She was under follow-up for premature ventricular contractions (PVCs) and had been treated with oral antiarrhythmic therapy (bisoprolol). Despite improvement in chest pain, her response to treatment was poor. She experienced an increase in PVC frequency despite medication up-titration over a three-month follow-up period. She also reported a marked decline in functional capacity, characterized by exertional dyspnea, distress, and palpitations during moderate physical activity. Symptoms persisted at rest, with a significant negative impact on quality of life. As a result, she limited her physical activity to walking due to symptom burden and fear of palpitations. **Conclusion:** in selected cases, PVCs ablation can effectively relieve symptoms and enhance QOL.

### *RESUMEN*

***Introducción:*** *los complejos ventriculares prematuros (CVP) se asocian con miocardiopatía inducida por CVP, deterioro funcional y mala calidad de vida (CV).* ***Presentación del caso:*** *mujer de 54 años que acudió a valoración por disnea y palpitaciones como síntomas principales. Se encontraba en seguimiento por complejos ventriculares prematuros (CVP) y en tratamiento con terapia antiarrítmica oral (bisoprolol). A pesar de mejoría del dolor torácico, la respuesta al tratamiento fue pobre. Presentó incremento en la frecuencia de los CVP a pesar de la titulación ascendente del fármaco durante un periodo de seguimiento de tres meses. Asimismo, refirió un deterioro marcado de la capacidad funcional, caracterizado por disnea de esfuerzo, malestar y palpitaciones durante actividad física moderada. Los síntomas persistían en reposo, con un impacto negativo significativo en su calidad de vida. Como consecuencia, limitó su actividad física a caminar debido a la carga sintomática y al temor a las palpitaciones.* ***Conclusión:*** *en casos seleccionados, la ablación de los CVP puede aliviar eficazmente los síntomas y mejorar la calidad de vida.*

**Abbreviations:**
CMR = Cardiac Magnetic Resonance
ICE = IntraCardiac Echocardiography
LCC = Left Coronary Cusp
LVEF = Left Ventricular Ejection Fraction
LVGLS = Left Ventricular Global Longitudinal Strain
PVC = Premature Ventricular Complexes
QOL = Quality Of Life

## INTRODUCTION

A high burden of premature ventricular complexes (PVCs) is associated with deterioration in ventricular function and quality of life (QOL). Current evidence demonstrates that frequent PVCs may lead to PVC-induced cardiomyopathy or aggravate pre-existing cardiomyopathy, particularly in patients with underlying structural heart disease.[1] A PVC

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burden exceeding 10% is generally considered the threshold above which the risk of developing ventricular dysfunction increases.[2] Predictors of PVC-related ventricular dysfunction include a superior PVC axis, non-sustained ventricular tachycardia, a short coupling interval, and male sex.[3] In clinical practice, differentiating PVC-induced cardiomyopathy from PVC-aggravated cardiomyopathy may be challenging, as there are often no definitive clinical or imaging markers to distinguish between the two entities. In many cases, the diagnosis is retrospective and supported by improvement in left ventricular ejection fraction (LVEF) following effective suppression of PVCs. Importantly, even in patients with previously established cardiomyopathy, catheter ablation may result in meaningful improvement in ventricular function.[4] Management strategies for PVCs depend on patient-specific factors and the anatomical location of the arrhythmogenic focus.[5] Catheter ablation may be performed using an endocardial, epicardial, or combined endo-epicardial «sandwich» approach, particularly when the focus is located in regions such as the left ventricular summit. While a PVC burden ≥ 10% supports consideration of catheter ablation, more extensive strategies, including combined approaches, are typically reserved for patients with a high PVC burden (generally ≥ 20-30%) or for those with refractory symptoms or ventricular dysfunction despite conventional endocardial ablation. The following case illustrates the diagnostic and therapeutic challenges associated with PVC-related cardiomyopathy.

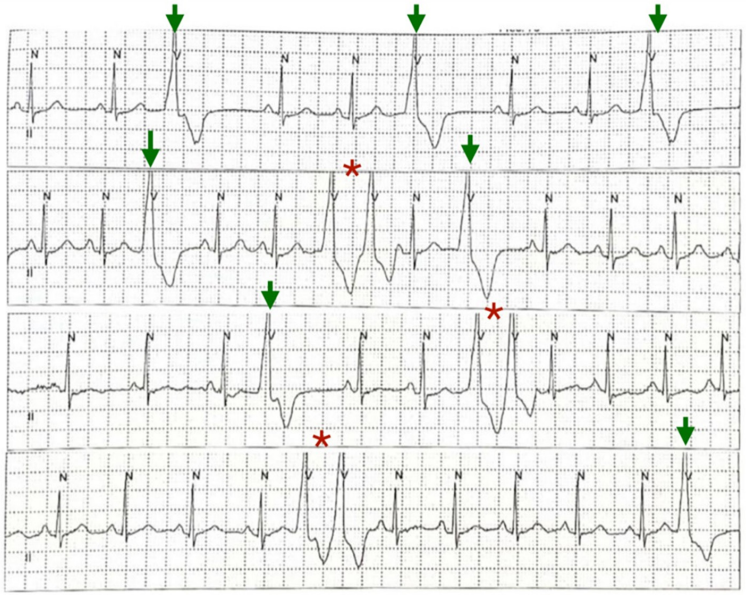

**Figure 1:** Ambulatory rhythm monitoring (Holter) demonstrating frequent monomorphic ventricular ectopic beats, primarily isolated (green arrow), occurring in a bigeminal pattern (red asterisks).

## CASE PRESENTATION

History of presentation: a 54-year-old female presented for evaluation, reporting dyspnea and palpitations as her primary concerns. The patient was under follow-up for ventricular extrasystoles, treated with oral antiarrhythmic medication. Despite improving chest pain, the patient's response to treatment was poor. The patient had an increase in the frequency of PVCs despite the up-titration of her medication in a three-month follow-up. Furthermore, the patient had a notable decrease in functional capacity attributed to functional impairment, distress, and dyspnea during moderate exertion. The symptoms remain at rest, with an essential effect on the patient's quality of life. As a consequence, the patient was compelled to limit her physical activity to walking, considering their symptoms and palpitations fear.

Past medical history: the patient had a past medical history of myocarditis one year prior, presenting with dyspnea, chest pain, and acute heart failure with preserved ejection fraction. Cardiac magnetic resonance (CMR) at that time demonstrated an inferior and inferolateral fibrotic scar, with a left ventricular ejection fraction (LVEF) of 62% *(Figure 1)*. Following the acute event, PVCs persisted, and she remained classified as ACC/AHA stage B heart failure with NYHA functional class II symptoms. Post-myocarditis echocardiography revealed preserved biventricular systolic function, no regional wall motion abnormalities, grade I diastolic dysfunction, mild mitral regurgitation, and an LVEF within normal limits. A treadmill test was negative for ischemia and positive for arrhythmia due to frequent monomorphic PVCs. Holter monitoring demonstrated an 11% burden of monomorphic PVCs, with symptom-

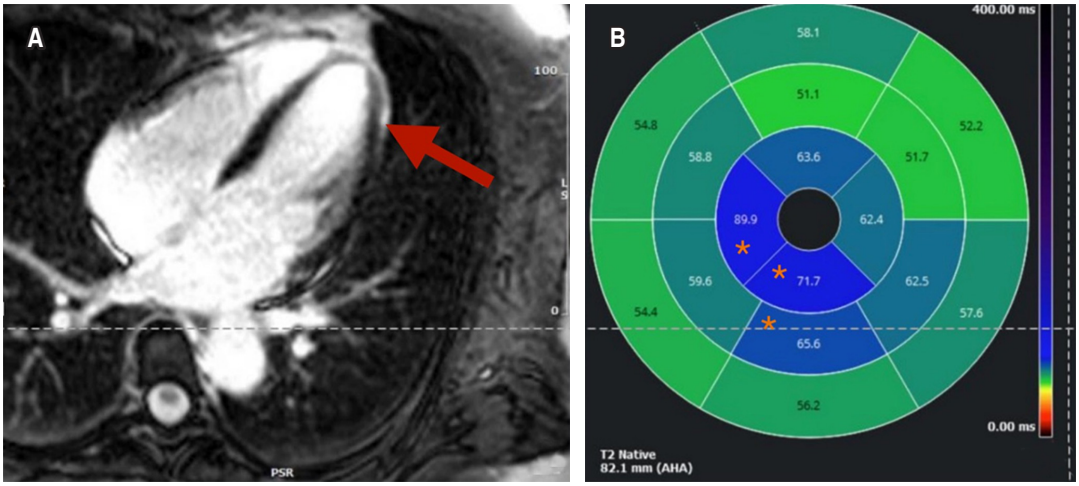


**Figure 2: A)** Subepicardial late gadolinium enhancement (LGE) at the inferior and inferolateral wall (red arrow). **B)** Corresponding T2 map, demonstrating increased signal intensity in the same inferolateral region (orange asterisks), suggestive of myocardial edema.

correlated episodes *(Figure 2)*. Medications prior to admission included bisoprolol; amiodarone had been previously trialed and discontinued due to lack of efficacy.

Differential diagnosis: our differential diagnosis included tachyarrhythmia, an acute electrolyte disorder, hypoxia, ischemic cardiomyopathy, deterioration in functional class, and relapse of myocarditis.

Investigation: the initial electrocardiogram demonstrated ventricular bigeminy, with PVCs exhibiting a right bundle branch block morphology *(Figure 3)*. Chest radiography was unremarkable. Transthoracic echocardiography revealed mild aortic and tricuspid regurgitation, a reduced LVEF, and mildly impaired left ventricular global longitudinal strain (LVGLS) of -13%. Despite optimized medical therapy, repeat Holter monitoring showed a 13% burden of monomorphic PVCs, frequently in bigeminy. Repeated exercise testing remained negative for ischemia but demonstrated a 50% reduction in exercise capacity, from 8:15 minutes (10.2 METs) to 4:10 minutes (7 METs), with a high PVC burden during exertion. Repeat CMR demonstrated LVEF 43%, RVEF 39%, reduced LVGLS, and persistent subepicardial late gadolinium enhancement at the inferior and inferolateral walls, consistent with prior imaging findings. Laboratory testing was unremarkable, except for an elevated BNP level. Given persistent symptoms, declining ventricular function, and a PVC burden exceeding 10%, a recognized threshold for considering catheter ablation—an electrophysiological procedure was proposed.

Management: she was admitted for an electrophysiological study and catheter ablation of PVCs. Three-dimensional electroanatomic mapping identified endocardial arrhythmogenic foci located near the left coronary cusp (LCC) at the left ventricular summit (LVS) *(Figure 3)*. The local electrograms demonstrated a complex, fractionated signal with a far-field component, and the earliest local ventricular activation was recorded 35 ms before QRS onset. Mapping and ablation were performed via a retrograde aortic approach using femoral arterial access, with femoral venous access for intracardiac echocardiography (ICE). An irrigated-tip catheter was used for radiofrequency ablation. Ablation around the LCC (120 seconds of RF delivery) resulted in a marked reduction in ectopic activity, prompting subsequent ablation at the summit, which ultimately led to complete PVC suppression

Outcome and follow-up: subsequent follow-up demonstrated enhanced functional capacity, improvement of LVEF and LVGLS, and the complete resolution of symptoms.

The subsequent HM showed a residual PVCs burden of 0%. Medical therapy was discontinued «timeline» *(Figure 4)*.

## DISCUSSION

European Society of Cardiology (ESC) guidelines propose a cut-off of > 10% to define a high PVC burden, above which an association with left ventricular dysfunction and quality-of-life (QOL) impairment becomes clinically relevant.[2,6] This threshold supports consideration of catheter ablation, particularly when symptoms or ventricular dysfunction are present. However, we believe that an individualized approach is essential, integrating symptom burden, QOL impairment, and potential confounders commonly encountered in this patient population. Higher PVC burdens confer progressively greater risk. In the ABC-VT score, a PVC burden > 20% is considered an independent predictor of PVC-induced cardiomyopathy, alongside other factors such as QRS duration, epicardial origin, underlying structural heart disease, and response or intolerance to medical therapy.[7] In clinical practice, PVC burdens ≥ 20-30% are often used to justify a more aggressive rhythm-control strategy, including early referral for catheter ablation and consideration of advanced or combined endo-epicardial approaches, particularly in patients with ventricular dysfunction or refractory symptoms. Importantly, the absence of a very high PVC burden does not exclude clinically significant disease. Our patient presented with an 11% PVC burden and evidence of cardiomyopathy, with subsequent improvement in ventricular function and symptoms following ablation. This finding underscores that the > 10% threshold remains clinically meaningful and highlights the importance of a thorough diagnostic evaluation to exclude alternative causes of cardiomyopathy and to identify patients who

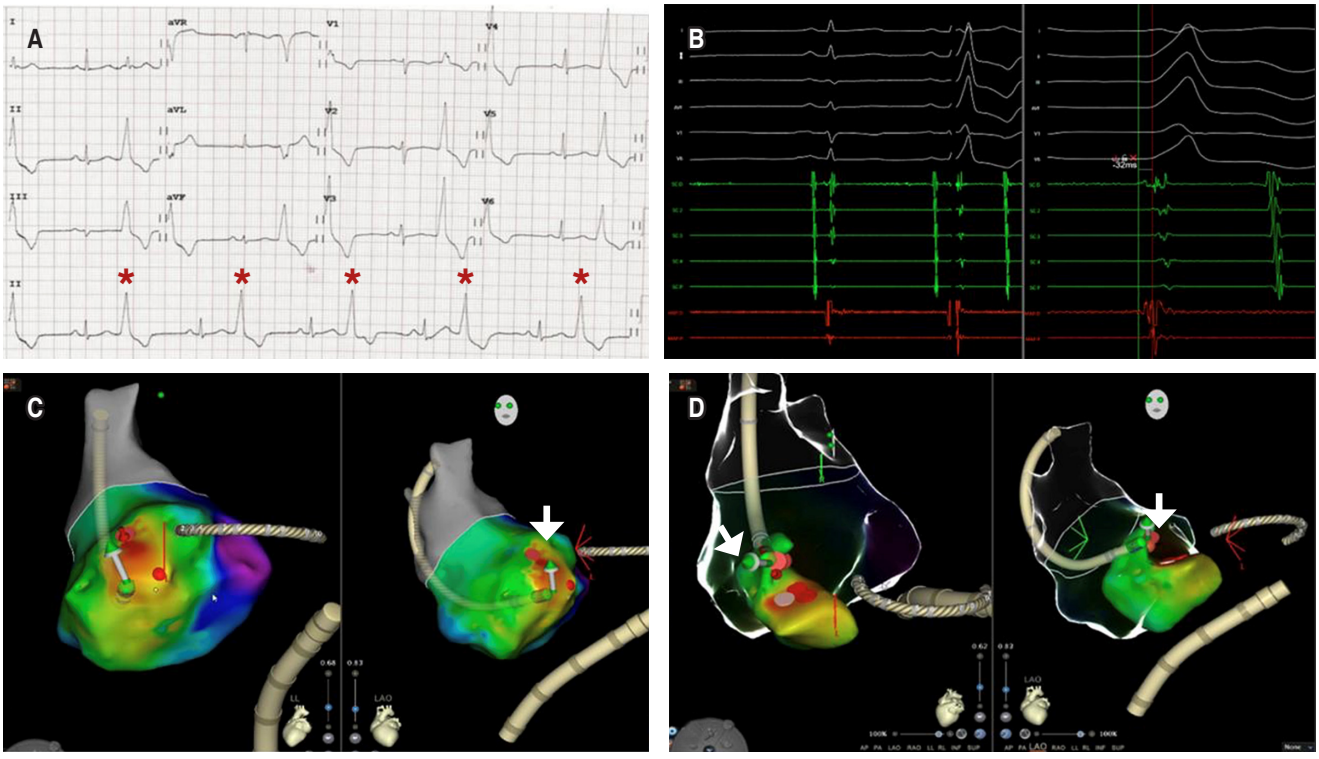


**Figure 3: A)** Initial electrocardiogram with ventricular bigeminy (red asterisks). **B)** Polygraph screen with PVC with a preexcitation of 35 ms (blue brackets). **C)** Initial activation map. **D)** Endomyocardial ablation for PVC around the left coronary cusp and left ventricular summit (white arrows). PVC = premature ventricular complexes.

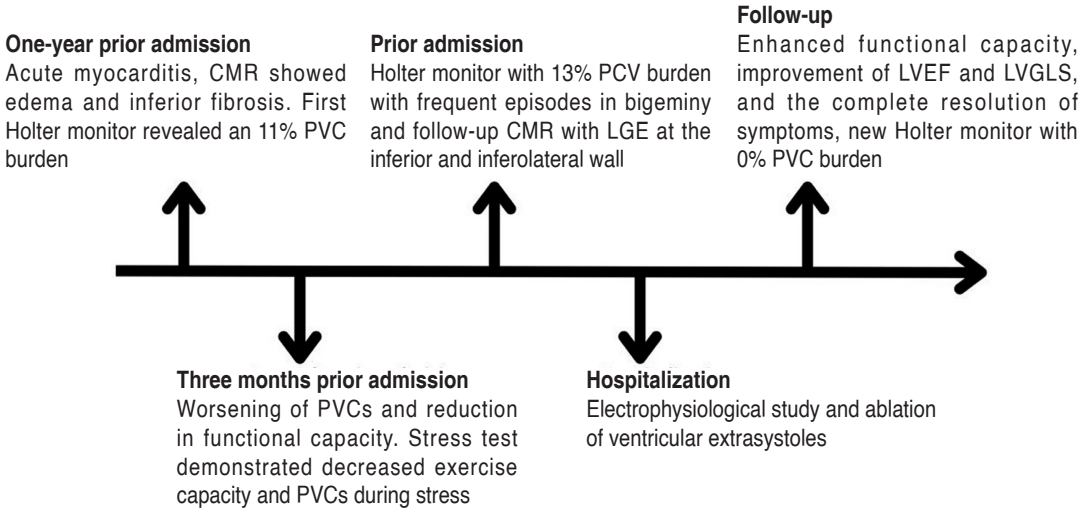


**Figure 4:** Timeline of events of the patient.
CMR = Cardiac magnetic resonance. LVEF = left ventricular ejection fraction. LVGLS = left ventricular global longitudinal strain. PVC = premature ventricular complexes.

may benefit from ablation even at lower burdens.[2] Although epidemiological data are limited, PVC ablation remains relatively uncommon in Latin America.[8] This low reported prevalence contrasts sharply with the high burden of cardiovascular and coronary artery disease in the region,[9] suggesting potential underdiagnosis and underutilization of catheter ablation in eligible patients.

Most idiopathic PVCs (70-80%) originate from the right or left ventricular outflow tracts. In the present case, the arrhythmogenic focus was localized to the left ventricular outflow tract at the level of the LCC, adjacent to the left ventricular summit, rather than the nonspecific term «left ventricular ostium». This anatomical region poses specific technical challenges. Pace mapping accuracy may be limited due to preferential conduction through adjacent structures, resulting in variable QRS morphologies. Additionally, some foci in this area may have an intramural or epicardial component, with close proximity to the coronary venous system or coronary arteries, mandating careful coronary assessment, particularly when coronary ostia are not clearly visualized using intracardiac echocardiography. A recent meta-analysis comparing endocardial ablation alone with combined endo-epicardial «sandwich» approaches demonstrated a significant reduction in PVC recurrence with the combined strategy, without an increase in procedural complications.[10] In our patient, an initial endomyocardial approach was selected, with a predefined plan to proceed to an endo-epicardial strategy if mapping suggested technical feasibility and incomplete suppression. This stepwise approach aimed to maximize long-term efficacy while minimizing recurrence, given the impact on left ventricular ejection fraction, QOL, and the patient's preference to avoid chronic antiarrhythmic therapy.

## CONCLUSIONS

This case highlights the challenges of managing persistent PVCs in patients who, despite initial treatment efforts, exhibit significantly compromised functional capacity and quality of life. The use of advanced electrophysiological techniques, including three-dimensional electroanatomical mapping, intracardiac echocardiography, voltage mapping, epicardial approaches, and radiofrequency ablation, resulted in a substantial reduction in PVC burden and an overall improvement in the patient's quality of life. These findings underscore the effectiveness of targeted electrophysiological interventions in the management of complex ventricular arrhythmias.

## Take-home messages:

1. A PVC burden exceeding 10% is associated with an increased risk of PVC-induced cardiomyopathy or worsening of pre-existing cardiomyopathy.
2. In selected patients, catheter ablation of PVCs can effectively alleviate symptoms and significantly improve quality of life.

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