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Case Reports

Myocardial Remission in High Burden Outflow Tract Premature Ventricular Complex-Induced Cardiomyopathy after Radiofrequency Catheter Ablation: Case Report

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Abstract

Background: Premature Ventricular Complex (PVC)-induced Cardiomyopathy (PVC-CMP) is a spectrum of dilated cardiomyopathy.

Case Illustration: A 51-year-old female patient who was diagnosed with high suspicion of PVC-CMP underwent successful 3D mapping radiofrequency catheter ablation with a good result. Post-ablation 24-hour ECG-Holter evaluation showed a significant reduction of PVC burden. Echocardiographic evaluation 5 months post-ablation showed improvement in left ventricular systolic function parameters. The presence of high-burden PVC with a typical outflow tract origin could raise suspicion of a specific PVC-CMP aetiology. PVC burden emerged as a major predictor of the development of CMP. Several criteria can be used to identify PVC-CMP. Our case met those descriptive criteria, increasing the likelihood of PVC-CMP.

Conclusions: PVC-CMP should be considered in patients with dilated cardiomyopathy who are accompanied by frequent outflow tract origin PVC (> 10 % burden). Early recognition of PVC-CMP is essential, as removal of the primary aetiology improves ventricular structure and function.

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Introduction

Premature Ventricular Complex (PVC)-induced Cardiomyopathy (PVC-CMP) is a dilated cardiomyopathy spectrum that has greater reversibility.[1-2] Early recognition of PVC-CMP is essential, since the removal of its primary aetiology will lead to improvement in ventricular structure and function.[1,3] Longitudinal strain echocardiography evaluation is a reliable method to detect initial changes in ventricular systolic function and to track therapeutic response in this patient population.[4-5]

Case Illustration

A 51-year-old female patient with a chronic palpitation history presented to the emergency ward with shortness of breath for 1 week before admission. The symptom worsened over 4 days, accompanied by chest tightness. The patient has no history of routine medication use. The patient denied any history of recent pregnancy, no history of recent fever or flu-like syndrome, and no history of alcohol consumption or other drugs. The patient's previous history was a hospital admission caused by palpitation 17 years ago, without knowing the cause. No familial history of cardiac disease was noted.

On-admission vitals sign: blood pressure 140/90 mmHg, pulse 85 beats/min, regular, and SpO_2 97 % in room air. Electrocardiogram (ECG) showed sinus rhythm with bigeminy and trigeminy PVC with Left Bundle Branch (LBBB) and inferior axis morphology, which suggests Outflow Tract (OT) origin. Echocardiography findings showed Left Ventricle (LV) dilation with Left Ventricular End-Diastolic Volume (LVEDV) of 185 mL, reduced LV systolic function with Left Ventricular Ejection Fraction (LVEF) of 37% (Simpson's Biplane) and reduced LV Global Longitudinal Strain (GLS) of -9.2%, as well as LV global hypokinesia and severe functional Mitral Regurgitation (MR) as shown in Figure 1 and Video 1.

Blood test examination showed normal renal function, serum troponin I, and thyroid function. At hospital admission, the patient was diagnosed with Dilated Cardiomyopathy (DCM) with high

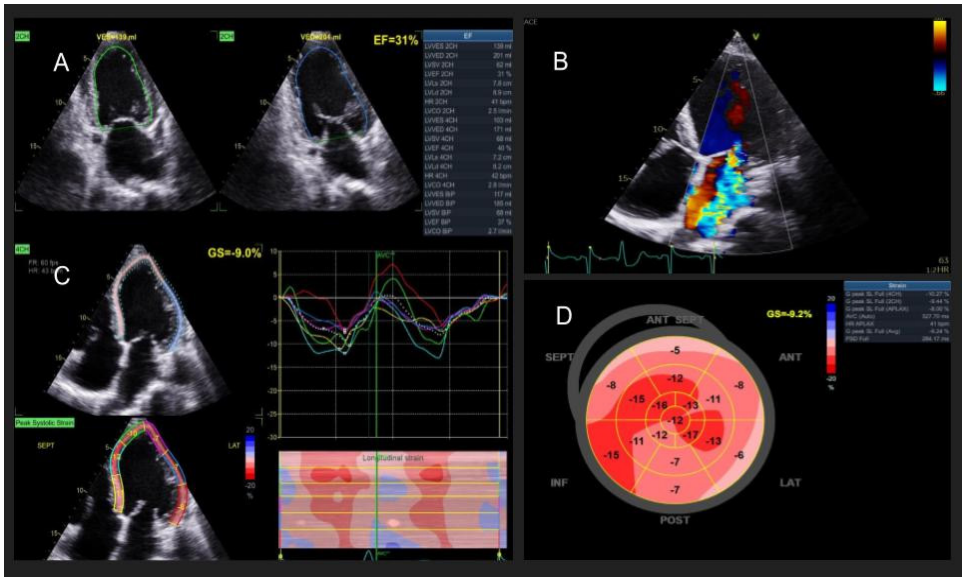

Figure 1. Baseline echocardiography examination: A. LVEF assessment by Simpson biplane method; B. Apical 4-chambers view showed dilated LV with severe MR; C. 4 chamber LV GLS tracing showed reduced 4ch GLS value; D. Average GLS showed reduced value (-9.2 %) (LVEF: Left ventricle ejection fraction; LV: Left ventricle; MR: Mitral regurgitation; GLS: Global longitudinal strain).

suspicion of PVC-CMP. A 24-hour Holter monitor showed very frequent LBBB with inferior-axis morphology, and OT-origin PVC with a 35% PVC burden. (Figure 2). Coronary Computed Tomography Angiography (CCTA) was noted for non-significant stenosis (50 % stenosis at proximal left anterior descending (LAD) artery, 30 % stenosis at mid LAD, mixed plaque with 50 % stenosis at proximal right coronary artery, and left circumflex artery was normal).

Standard guideline-directed medical therapy (GDMT) for Heart Failure with reduced Ejection Fraction (HFrEF) was administered and up-titrated during hospital admission, including a loop diuretic,

bisoprolol 5 mg o.d., spironolactone 25 mg o.d., and sacubitril/valsartan 100 mg b.d. The patient's definitive management for the high clinically suspicious PVC-CMP was made. The patient was referred for further management, including radiofrequency catheter ablation.

The patient underwent successful radiofrequency catheter ablation with a favorable outcome using the 3D EnSite$_{TM}$ electroanatomical mapping system. Multifocal PVCs from the Left Coronary Cusp (LCC) were detected. The mapping revealed the highest LAT of -34 ms, with a QS pattern, in the unipolar lead at the anterior LCC. Multiple radiofrequency ablations were delivered at those areas (30 W, 45°C, 17 ml/sec), and PVCs were terminated.

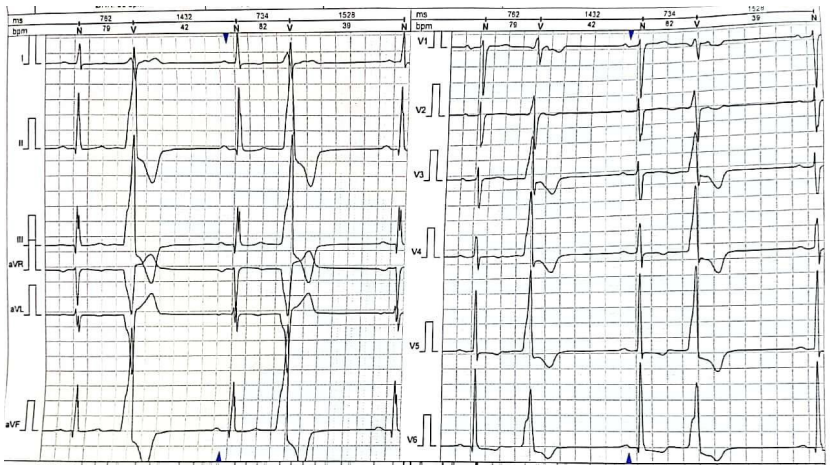

Figure 2. Baseline Holter electrocardiography showed sinus rhythm with bigemini outflow tract premature ventricular complex.

Post-ablation 24-hour ECG-Holter monitoring showed sinus rhythm with a minimal PVC burden of 3%. There was no symptom persistence. Echocardiographic evaluation 5 months post-ablation showed LV volume reduction (LVEDV 163 mL) with LVEF improvement to 43%, an increase in LV GLS value to -14%, and a decrease in mitral regurgitation severity as shown in Figure 4 and Video 2. This echocardiographic finding depicts reverse remodelling following removal of the primary insult in cardiomyopathy.

Discussion

We reported a 51-year-old female patient clinically suspected of PVC-CMP in our local community hospital settings. Tachycardia-induced cardiomyopathy is a known reversible cause of LV systolic dysfunction. Hence, the aetiology of HFrEF, in the absence of any apparent reason, with frequent PVCs, must be carefully investigated.[1,6] In our case, the presence of a high PVC burden of typical OT origin from ECG could raise a high suspicion index of specific PVC-CMP etiology.[3]

Among patients with PVC burden > 10%, it was reported that the prevalence of PVC-CMP is about 7%.[7] Furthermore, PVC burden > 10% is considered significant enough to trigger CMP.[1-2] Several studies revealed the correlation between PVC burden and the degree of LV dysfunction. A patient with low LVEF at initial presentation was found to have a higher mean of PVC burden.[7] Therefore, PVC burden was reported to become a major predictor of PVC that can develop into CMP (OR 1.25 for each percent increase in PVC burden).[1] In our case, the patient showed a PVC burden of 35 % from a 24-hour ECG-Holter monitor, which obviously increases the chance for the patient to develop PVC-CMP.

The most common PVC-CMP is idiopathic Ventricular Arrhythmia (VA), which commonly originates from the OT.[2,6] Our patient also showed a typical OT origin of PVC, which increased the possibility of PVC-CMP diagnosis, though 3D electroanatomical mapping showed the PVC location origin was from LCC.

There is no specific test or marker to confirm PVC-CMP. The underlying tachyarrhythmia is not always documented at initial presentation, particularly in cases of paroxysmal AF or flutter.[6] Therefore, a 24-hour Holter monitoring may be helpful as a first-line tool for diagnosing PVC-CMP

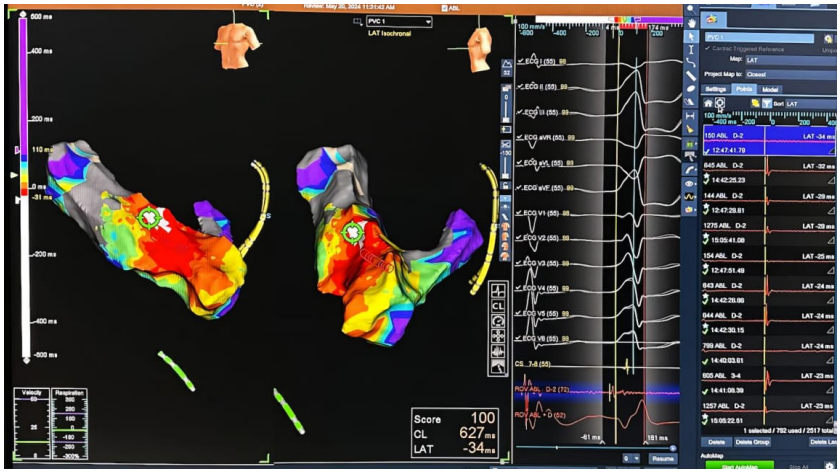

Figure 3. 3D Electroanatomical mapping of premature ventricular complex ablation.

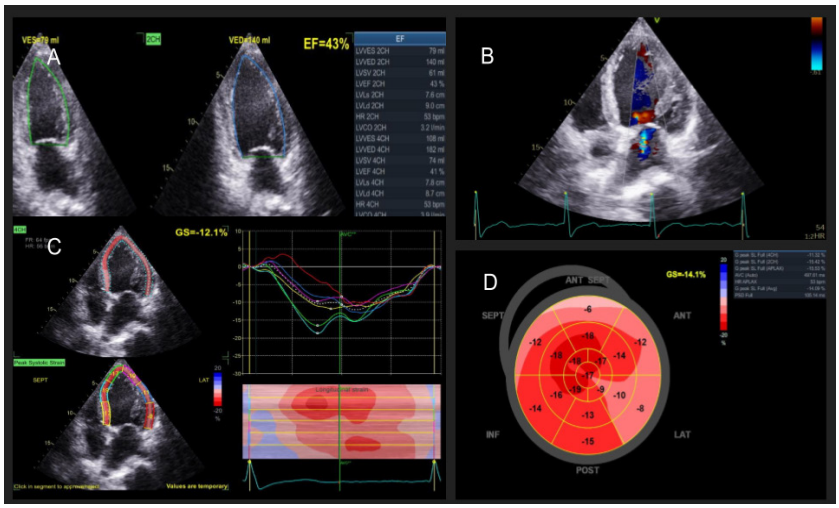

Figure 4. Post-catheter ablation echocardiography examination: A. LVEF assessment by Simpson biplane method showed LVEF improvement; B. Apical 4-chambers view showed LV volume reduction with MR severity reduction; C. 4-chamber LV GLS tracing showed reduced 4-ch GLS value; D. Average GLS showed improved value (-14.1 %) (LVEF: Left ventricle ejection fraction; LV: Left ventricle; MR: Mitral regurgitation; GLS: Global longitudinal strain).

in patients with frequent PVC and ventricular systolic dysfunction. In our case, we meticulously obtained the patient's clinical history of exclude other DCM aetiologies. Routine blood tests, including renal function, thyroid function, serum troponin I, and an infection marker, exclude potential aetiologies of LV dysfunction, such as myocarditis, thyrotoxicosis, diabetic cardiomyopathy, and end-stage kidney disease.

The role of non-invasive diagnostics for the evaluation of systolic function, for instance, the longitudinal strain imaging echocardiography, is of paramount importance to recognize even the subclinical LV systolic dysfunction in PVC-CMP cases,[4-5] especially in limited hospital settings, to exclude any underlying coronary artery disease that underlies systolic dysfunction. Fortunately, the patient's CCTA result was typical, thus we can exclude significant epicardial coronary stenosis as an aetiology of the patient's DCM. However, the final diagnosis of PVC-CMP can only be made if there is an improvement of myocardial systolic function after precipitating PVC has been eliminated, as observed in our case.[1-3,6]

There are challenges in daily clinical practice to differentiate among PVCs that induce cardiomyopathy or VA manifestation in primary cardiomyopathy. In this concern, echocardiography and PVC characteristics could help to define the primary cause. Bozkurt et al describe several criteria for PVC-CMP. Those criteria are global hypokinesis with 'not too low' LVEF of 37 ± 10%,

monomorphic PVC pattern, PVC burden > 10%, and outflow tract or epicardial QRS morphology on ECG. Unfortunately, CMR criteria cannot be evaluated because CMR is of available in our setting.[9] However, Ailoaei et al showed that higher PVC burden and LBBB with inferior axis morphology are predictors of LGE presence by CMR in PVC with a structurally normal heart.[10]

On the other hand, different yet similar criteria for PVC-CMP were recently proposed by Bhushan and Asirvatham. These criteria apply to otherwise young, healthy individuals with no evidence of abnormal cardiovascular substrate who have more than 20,000 PVCs per day, with no more than two PVC morphologies, OT-origin PVCs, or fascicular origin, and preserved myocardial wall thickness, and are the best candidates for a PVC-CMP.[2,11] Our case met all the criteria mentioned above.

Hence, echocardiographic and PVC characteristics could help determine whether PVC is the primary cause of CMP. These criteria could be helpful in clinical practice for defining PVC-CMP for referral to radiofrequency catheter ablation, given its reversibility. PVC-CMP should always be considered in patients with idiopathic DCM who are accompanied by frequent OT origin PVC (more than 10% burden).[6,12]

A catheter ablation management strategy of high-burden OT PVC is superior to pharmacological anti-arrhythmic therapy in suppressing arrhythmic events without differences in complications. It is recommended as first-line therapy for OT PVC. Catheter ablation is also recommended as a first-line management strategy for PVC-CMP, with a reported success rate of approximately 90%.[12] In our case, we performed a 3D electroanatomical mapping system that revealed LCC as the origin of the patient's PVCs. After successful catheter ablation, as confirmed by 24-hour ECG-Holter monitoring, we observed improvement in LV systolic function and echocardiographic parameters. Despite improvements of LV GLS parameters, they did not fully recover to the standard GLS value (-14%), and the follow-up period was limited to a short-term evaluation. The short-term follow-up in our case reflects a limitation in this report. However, this finding indicated a glimpse of myocardial recovery that may be complete with extended follow-up.

El Kadri et al reported a study in 30 patients with PVC-CMP who underwent catheter ablation. The study population had a mean age of 59.1 ± 12.1 years, a mean baseline LVEF of 38 ± 15%, and a mean PVC burden of 22.7 ± 11.6%. Improvement of LVEF was observed in the successfully ablated patient (33.9% ± 14% to 45.7% ± 17%; $p < 0.0001$), with a corresponding significant reduction in PVC burden (23.1% ± 8.8% to 1% ± 0.9%; $p < 0.0001$). This study found that PVC-CMP cardiomyopathy is a reversible pathological entity after PVC ablation, even though the improvement is not fully recovered.[13] The other previous individual case reports of PVC-CMP with their baseline clinical characteristics and follow-up are summarized in Table 1.

Although the exact mechanism of PVC-induced cardiomyopathy remains unclear, the best-described cellular mechanism, extrapolated from animal studies, involves beat-to-beat variation in action potential duration that reduces the inward and outward movement of L-type calcium current in cardiac myocytes and ultimately results in repolarization heterogeneity. Other cellular pathophysiology of PVC-CMP is related to impaired cardiomyocyte calcium handling from increased levels of phosphorylated ryanodine

Table 1. Summary table of the previous case report of premature ventricular complex-cardiomyopathy with its baseline and follow-up LVEF and PVC burden.

Author (year)	Sex/Age	Baseline LVEF (%)	Baseline PVC burden (%)	Follow-up LVEF (%)	Follow-up PVC burden (%)	Management
Chung *et al* (2021)[14]	M/65	43	32	53	<1	RFCA (no recurrency)
Senapati *et al* (2020)[15]	M/74	43	40	57	2	RFCA (no recurrency)
Sun *et al* (2022)[16]	M/53	34	80	46	1	RFCA (no recurrency)
Bekke *et al* (2025)[17]	M/28	45	30	60	Absent	RFCA (no recurrency)

Abbreviation note: LVEF: Left Ventricular Ejection Fraction; PVC: Premature Ventricular Complex; M: Male; RFCA: Radiofrequency Catheter Ablation.

receptor 2, Na/Ca exchanger 1, Ca^{2+}/calmodulin-dependent protein kinase II-alpha, with the result of downregulation of Sarcoplasmic Reticulum (SR) and decrease of SR $[Ca]^{2+}$ store.[18-19]

In addition, LV dyssynchrony is described as a necessary feature of that may be associated with PVC-CMP.[19] The long-term and frequent LV dyssynchrony can contribute to the development of LV dysfunction because of asymmetrically increased wall thickness in the late-activated LV segment and myocardial perfusion alteration.[18] Myocardial remission and recovery will be developed because of the reversal of the aforementioned process. The PVC-CMP pathological process is a reversible condition after primary PVCs are terminated.[18-19]

Conclusion

We reported a PVC-CMP patient who showed signs of a significant recovery process after successful catheter ablation. The non-significant coronary lesion which was shown by CCTA describes the high burden of PVC in this case, which was established as a primary insult for cardiomyopathy development. Causal factor management in PVC-CMP led to myocardial remission.

List of Abbreviations

CCTA	Coronary Computed Tomography Angiography
CMP	Cardiomyopathy
DCM	Dilated Cardiomyopathy
ECG	Electrocardiogram
GDMT	Guideline-Directed Medical Therapy
GLS	Global Longitudinal Strain
HFrEF	Heart Failure with reduced Ejection Fraction
LBBB	Left Bundle Branch Block
LCC	Left Coronary Cusp
LGE	Late-Gadolinium Enhancement
LV	Left Ventricle
LVEDV	Left Ventricular End-Diastolic Volume
LVEF	Left Ventricular Ejection Fraction
MR	Mitral Regurgitation
OT	Outflow Tract
PVC	Premature Ventricular Complex
PVC-CMP	Premature Ventricular Complex-induced Cardiomyopathy
SR	Sarcoplasmic Reticulum
VA	Ventricular Arrhythmia

Ethical Clearance

Not Applicable.

Publication Approval

All authors consent to the publication of this manuscript.

Authors Contributions

All authors have made a significant intellectual contribution to the manuscript according to the criteria formulated by the International Committee of Medical Journal Editors.

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Conflict of Interest

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