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CASE REPORT

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Case of idiopathic left ventricular summit premature ventricular contractions successfully treated by catheter ablation

Na Tian[1] | **Like Ma**[1] | **Yinjiao Ma**[1] | **Zhen Yang**[1,2,3]

[1]Institute of Medical Sciences, General Hospital of Ningxia Medical University, Yinchuan, Ningxia, China

[2]Institute of Cardiovascular Medicine, General Hospital of Ningxia Medical University, Ningxia, China

[3]Department of Cardiology, General Hospital of Ningxia Medical University, Ningxia Medical University, Ningxia, China

Correspondence
Zhen Yang, Department of cardiovascular medicine, General Hospital of Ningxia Medical University, Institute of Cardiology, Ningxia Medical University, 804 Shengli Street, Xinqing District, Yinchuan, Ningxia, China.
Email: yangzhen080@163.com

Key Clinical Message

CA of LVS premature beats complexes is difficult due to anatomical limitations. We report a patient with PVCs originating from the LVS region who was successfully ablated by ablation.

Abstract

Catheter ablation (CA) of premature ventricular contractions (PVCs) arising from the left ventricular summit (LVS) presents technical challenges due to the regional anatomy and frequently intramural site of origin. Herein, we demonstrated a case of a successful CA, originating from the LVS region. We further discussed the detailed anatomical background and clinical feasibility of CA as an alternative ablation route for PVCs originating from the LVS.

KEYWORDS

cardiology, cardiovascular disorders, catheter ablation, premature ventricular beats

1 | INTRODUCTION

The left ventricular summit (LVS) is a challenging location for catheter-based percutaneous ablation of cardiac arrhythmias due to its anatomical location. Radiofrequency catheter ablation (RFCA) in this region is often limited by both high impedance and inadvertent damage to surrounding structures. There have been case reports of ablation performed in this region, but the technique may be underutilized when RFCA fails. We present a case of successful ablation of summit PVCs via the coronary vein approach.

2 | CASE PRESENTATION

2.1 | Medical history

A 57-year-old woman was admitted with the chief complaint of intermittent palpitations for 1 year, which aggravated for the latest 10 days. One year ago, the patient developed intermittent palpitations accompanied by chest tightness and shortness of breath after exertion or emotional stress, lasting approximately 10 min. She had no relevant previous interventions ever done. Ten days prior to admission, her symptoms deteriorated. The patient

was admitted to our hospital with diagnosis of frequent PVCs. She had a two-year history of hypertension, which was well controlled. She also had a history of coronary artery disease for 2 years, and had undergone percutaneous coronary intervention 2 years ago. The patient had no history of smoking, alcohol abuse, and drug misuse. And there was no family, and psychosocial history including relevant genetic information.

2.2 | Examination findings

A 24-hour ambulatory electrocardiogram showed a total of 20,568 PVCs, with a burden of 20.35%. The morphology of PVCs showed an R pattern with high amplitude in leads II, III and aVF; an rS pattern in lead I; a QS pattern in aVR and aVL; and an R pattern in leads $V_{1\text{-}6}$, consistent with the electrocardiographic features of PVCs originating from the left ventricular outflow tract(LVOT) (Figure 1). Transthoracic echocardiography revealed mild tricuspid regurgitation, reduced left ventricular diastolic function, a normal left ventricular ejection fraction (LVEF) of 65.81% and a normal left ventricular end-diastolic diameter (47 mm). Biochemical indices did not show any significant abnormality. Due to severe symptoms and high burden of premature ventricular complexes, CA was recommended. The patient and her family members signed the consent form and the CA procedure was performed.

2.3 | Ablation technique

The QRS pattern of PVCs suggested LVOT origin. Therefore, the right femoral artery was punctured, a long sheath was inserted into the descending aorta, and an ST (HERMOCOOL SMARTTOUCH, Biosense Webster) ablation catheter was inserted into the aortic sinus through the sheath. Under the guidance of the Carto system, a three-dimensional model of the aortic sinus and LVOT was reconstructed, and activation mapping was performed. The earliest local electrogram during PVCs was mapped to be beneath the aortic valve of the left coronary sinus, which was 28 ms earlier than the QRS of body surface electrocardiogram (ECG). However, PVCs persisted even after ablation with a power of 35 W and a temperature limit of 43°C. Considering the structural complexity of the outflow tract and the fact that some of the PVCs with even an upright QRS in lead V_1 need to be mapped and ablated in the RVOT, the right femoral vein was punctured, and a long sheath was inserted and delivered into the right atrium. After the RVOT model was reconstructed, activation mapping was performed within the RVOT, and the earliest local electrogram of PVCs was mapped to the anterior septum of the RVOT, 30 ms earlier than the QRS of the body surface ECG. Ablation at this site still failed to eliminate the PVCs. Because the ablation of both the left and right outflow tracts failed, it was considered that the PVCs of this patient might originate from the LVS area. The ablation catheter was then inserted into the summit region via the coronary vein. The local electrogram in the summit region during PVCs was 33 ms earlier than the body surface QRS (Figure 2), and the PVCs disappeared after ablation at this site. After ablation, an aggressive stimulation protocol induced sustained atrial fibrillation, which had not been previously documented in this patient and therefore was not treated. During a 30-min waiting period, no PVCs occurred. Postoperative review via routine 12-lead surface ECG did not reveal any PVCs (Figure 3).

2.4 | Patient outcomes

PVCs originating from the LVS region were successfully ablated, and the patient's symptoms significantly improved. At 6 months follow-up, She has no adverse and

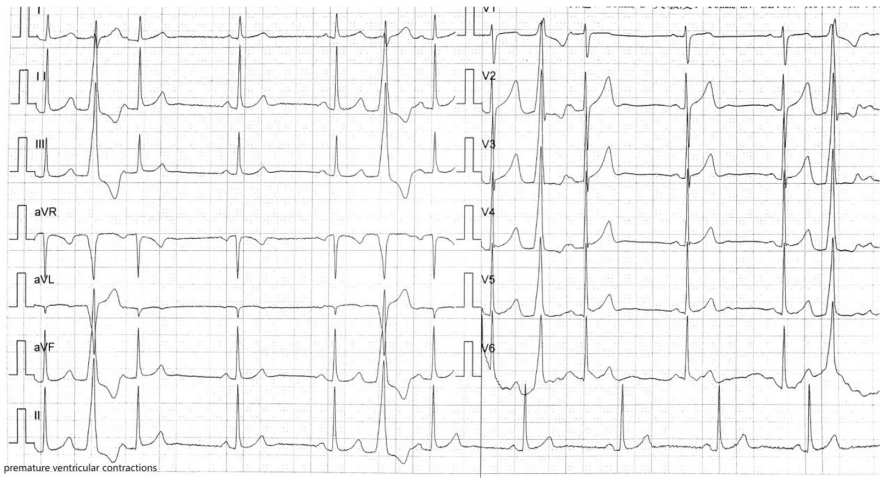

FIGURE 1 Preoperative 12-lead surface ECG indicated PVCs: An R pattern with high amplitude in leads II, III and aVF; an rS pattern in lead I; a QS pattern in aVR and aVL; and an R pattern in leads V1-6, consistent with the electrocardiographic features of PVCs originating from the LVOT.

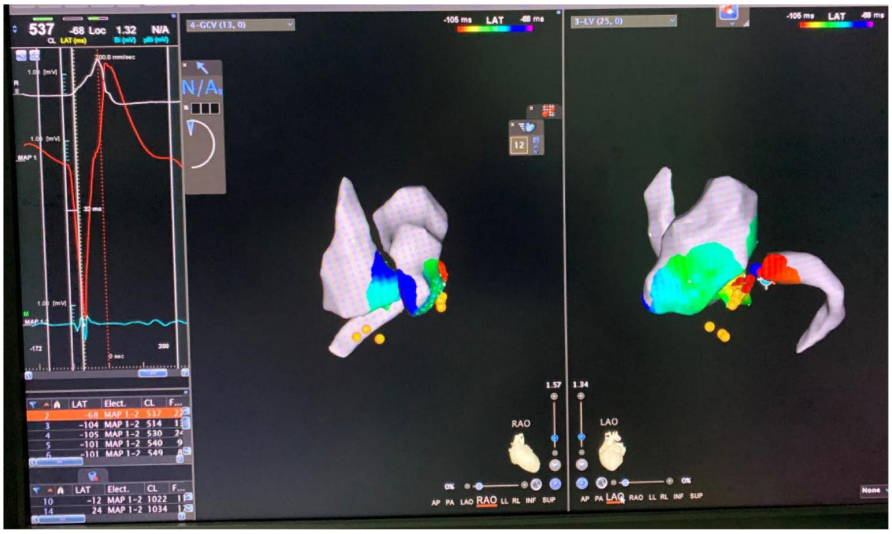

FIGURE 2 Intraoperative three-dimensional electroanatomic mapping of PVCs: The local electrogram of the summit region during PVCs occurred 33 ms earlier than that of the body surface QRS. Ablation at this site successfully eliminated the PVCs; 14.64 × 8.68 cm (1537 × 911 PX).

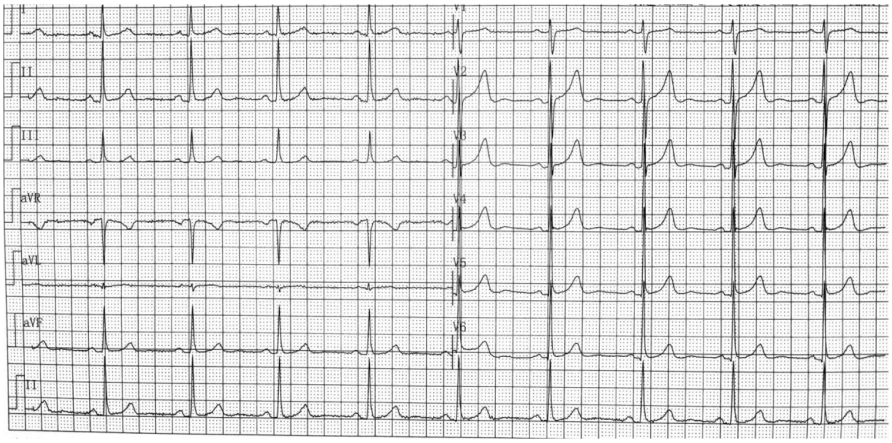

FIGURE 3 Postoperative 12-lead electrocardiogram of the body surface. No PVCs were documented; 14.65 × 7.18 cm (3872 × 1898 PX).

unanticipated event, and was arrhythmia free without antiarrhythmic drug therapy. She indicated that she had significant benefits from CA.

3 | DISCUSSION

LVS arrhythmias are difficult to ablate due to their proximity to critical structures and potential for severe complications.[1] In a significant number of cases, it is difficult to advance the ablation catheter into the coronary venous system and get to the great cardiac vein (GCV)/anterior interventricular vein (AIV) and their branches. In addition, the high impedance and limited cooling from blood flow possibly contribute to the relatively low success rate of ablation within the distal GCV/AIV.[2–4] The potential complications of CA of PVCs in this region include damage to external vessels/nerves, disruption of coronary flow that may present with ECG changes or may be electrically silent, cardiac perforation, and other traditional complications of RFCA.[1] The alternative trans-epicardial route is hampered by the passage of important coronary arteries, as well as the presence of a fat layer distributed over the epicardium, which impedes the delivery of appropriate RF energy to the target region.[5] Therefore, CA of PVCs in the summit region is clinically challenging.

In this case, based on the ECG manifestations, the PVCs were initially considered to be of LVOT origin. After the ablation of both the left and right outflow tracts failed, the LVS focus of the PVCs was considered. After the ablation catheter reached the summit area through the coronary vein, the local electrogram during PVCs was the earliest, which was 33 ms earlier than the QRS of the body surface ECG, and after delivery of radiofrequency energy, the PVCs disappeared.

The findings in this case suggested the ablation catheter can reach the LVS region via the coronary vein, exactly map and identify the ablation target in the summit region, and successfully eliminate the PVCs. It is mandatory to precisely identify the LVS area, which may have an unusual location. In order to ensure the safety of the ablation, a contact force-monitoring ablation catheter was recommended to help control the force of the apposition and the vector of the force, and the coronary vein was clearly defined by three-dimensional mapping system to assist in the smooth entry of the catheter into the coronary sinus

and the GCV/AIV. It is suggested that although the ablation of the PVCs originating from the LVS region remains a big challenge, catheter-based RFCA is still effective in the treatment of PVCs of LVS origin when performed carefully with the help of a contact force monitoring catheter and a three-dimensional electroanatomic system.

4 | CONCLUSION

The LVS region is anatomically complex. With the help of the contact force-monitoring ablation catheters and three-dimensional electroanatomic mapping system, CA is safe and effective for the treatment of PVCs of LVS origin.

AUTHOR CONTRIBUTIONS

Na Tian: Conceptualization; writing – original draft. **Like Ma:** Supervision; writing – original draft. **Yinjiao Ma:** Supervision. **Zhen Yang:** Resources; writing – review and editing.

FUNDING INFORMATION

None.

CONFLICT OF INTEREST STATEMENT

The research was conducted in the absence of any commercial or financial relationships that could be construed as potential conflicts of interest.

DATA AVAILABILITY STATEMENT

All data generated or analyzed during this study are included in this published article.

ETHICAL APPROVAL

The article describes a case report. Therefore, no additional permission from our Ethics Committee was required.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

PERMISSION TO REPRODUCE MATERIAL FROM OTHER SOURCES

Our researchers are agreed to make our data available to others, subject to legal, ethical, and commercial limitations. There is no clinical trial registration.

ORCID

Na Tian https://orcid.org/0009-0000-8197-3370

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