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Successful ablation for premature ventricular contraction originating from moderator band of morphologic right ventricle in congenitally corrected transposition of great arteries

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ABSTRACT

A 54-year-old man with congenitally corrected transposition of great arteries (CCTGA) was referred to our hospital for palpitation. 24-hour Holter ECG showed frequent premature ventricular contraction (PVC) and we performed catheter ablation for this PVC. Pace-mapping was performed in morphologic right ventricle (RV) by transaortic approach. Perfect pace-map was achieved in morphologic RV midpart lateral and ablation at this site could eliminate the clinical PVC. After the ablation, by integrating ablation site and 3D mapping, we diagnosed that the clinical PVC was originated from the moderator band (MB) of morphologic RV.

Introduction

Congenitally corrected transposition of great arteries (CCTGA) is a rare complex cardiac anomaly, which has atrioventricular and ventriculoarterial discordance [1]. The morphologic left ventricle (LV) functions as the pulmonary ventricle, and in reverse, the morphologic right ventricle (RV) functions as the systemic ventricle. Although catheter ablation has been established as the treatment of premature ventricular contraction (PVC) arising from RV [2], there are few reports about catheter ablation of PVC for morphologic RV of CCTGA. We report rare case of successful ablation for PVC originated from moderator band of morphologic RV in CCTGA.

Case report

A 54-year-old man with CCTGA had a history of surgical ventricular septal defect (VSD) closure and operation for pulmonary stenosis in 1981, and systemic atrioventricular tricuspid valve replacement (#29 MJ-501, Sent Jude Medical, St. Paul, MN), pulmonary atrioventricular mitral valve repair (#32 Cosgrove ring, Edwards Lifesciences Irvine, California, United States), and epicardial pacemaker lead placement in 2012 owing to heart failure. He was referred to our hospital for palpitation. The 12-lead electrocardiogram (ECG) showed frequent PVC with right bundle branch block morphology, superior axis and negative polarity in I lead (Fig. 1). Twenty four-hour Holter ECG monitoring revealed a PVC burden of 9645/day (9.462%) without nonsustained ventricular tachycardia episodes. Chest radiograph showed mildly enlarged cardio-thoracic ratio and epicardial pacemaker lead. Transthoracic echocardiography revealed decreased systemic ventricular ejection fraction (38%, modified Sympson's method), with no valve dysfunction including prosthetic valve. Because his symptom was intolerable palpitation, we performed catheter ablation for the clinical PVC.

Electrophysiological study and catheter ablation was performed under 3-dimentional (3D) mapping system (CARTO3; Biosense Webster, Diamond Bar, CA, USA). Because there were insufficient PVC load to map the PVC origin during the procedure, we mainly use the pace map to detect the PVC origin. First, we positioned an intracardiac echocardiography (ICE) probe with a CARTO navigation sensor (SOUNDSTAR®, Biosense Webster) in the right atrium or the pulmonary ventricle (morphologic LV), and geometry of the systemic ventricle (morphologic RV) and moderator band (MB) was constructed. Next, we performed pace-mapping in the morphologic RV using an ablation catheter (ThermoCool; Biosens Webster) by transaortic approach. A perfect pace-map (PaSo score 0.987) was

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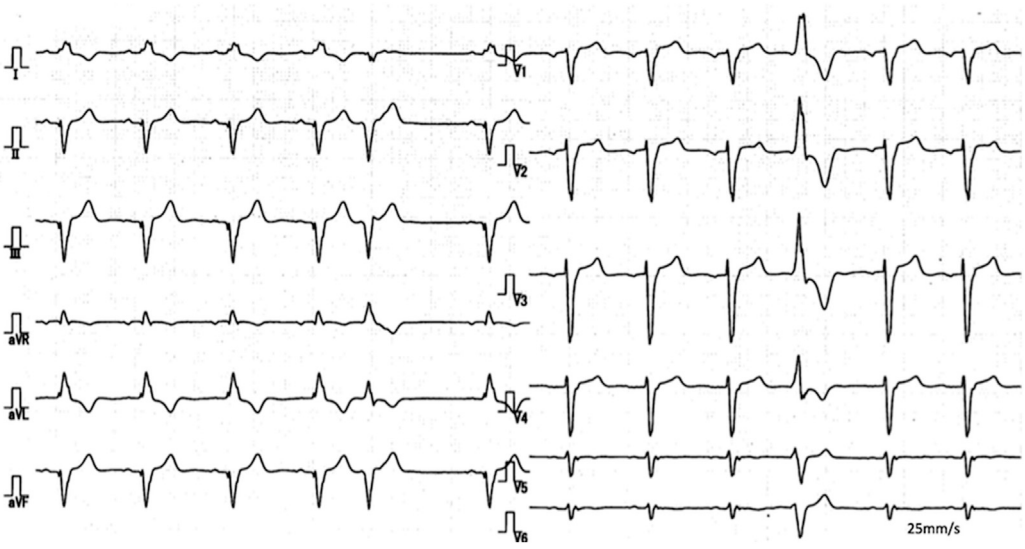

Fig. 1. 12-lead ECG during clinical PVC (ECG: electrocardiogram, PVC: premature ventricular contraction).

achieved at the midpart lateral of the morphologic RV (Fig. 2). At this site, 41 ms-ventricular potential preceding the surface QRS was observed using the ablation catheter during clinical PVC (Fig. 3). The good contact force was obtained at this site, and radiofrequency energy (40 °C, 25 W) was applied at this site, which resulted in elimination of clinical PVC. Radiofrequency energy applied ten times in total, for 45 s each time. We used Isprel (100 μg/h) to induce PVC and waited 40 min, but clinical PVC was not induced. Total procedure time was 2 h and 20 min. Fluoroscopy time was 27 min and total ablation time was 7 min and 30 s. After the ablation, by integrating ablation site and geometry of the morphologic RV and the MB using CARTO system, we diagnosed that the clinical PVC was originated from the MB of morphologic RV (Fig. 4). After the procedure, the patient remained symptom-free and twenty four-hour Holter ECG did not show clinical PVC recurrence at 6 months follow-up, but increase of systemic ejection fraction was not observed.

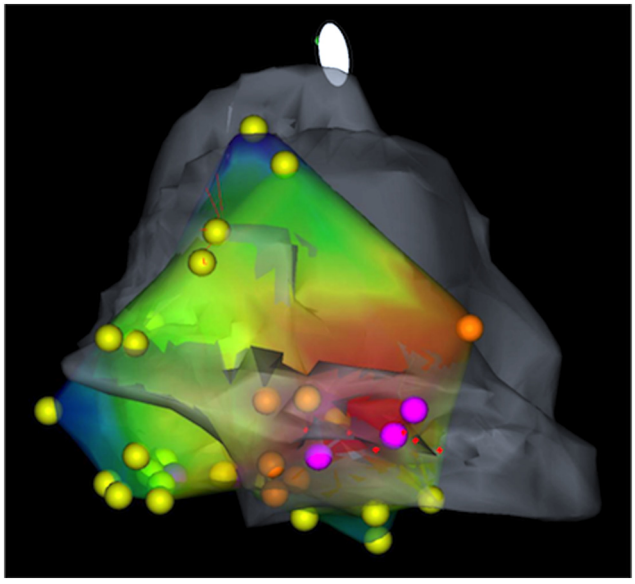

Fig. 2. Pacemap and geometry constructed by ICE. Pink tags show the sites of perfect pacemapping. Light gray geometry shows the morphologic RV. (ICE: intracardiac echocardiogram, RV: right ventricle).

Discussion

In this case, the origin of clinical PVC was the MB of the morphologic RV in CCTGA. Ventricular arrhythmias from MB are rare. Sadek et al. reported that in 394 patients underwent ablation for ventricular arrhythmias, only 10 patients (2.5%) presented with ventricular arrhythmias from MB [3]. Kasar et al. reported that most of rhythm disturbances in CCTGA patients are supraventricular arrhythmia and atrioventricular block [4]. There are two case reports of catheter ablation for ventricular tachycardia of CCTGA patient [5,6]. One is bundle branch reentrant ventricular tachycardia, and the other is reentrant tachycardia with slow conduction zone existed around the suture of VSD. To our knowledge, there has never been a case report describing PVC originated from MB of CCTGA patient.

The morphology of clinical PVC was right bundle branch block pattern, superior axis, negative polarity in I lead, and rS pattern in V6 lead. We suggested that this morphology showed that the origin of PVC was mid lateral of systemic LV (morphologic RV), in where the moderator band was located. We think that conventional ECG diagnosis is useful even in CCTGA patient. In addition, as a previous report showed [3], narrow QRS morphology is consistent with MB origin of the PVCs.

It has been reported that successful rate of catheter ablation for PVC from MB was not high. Mouhannad M. Sadek et al. has reported that 6 of 10 patients who underwent catheter ablation for PVC from MB needed second procedure [3]. We could succeed catheter ablation for PVC from MB, even in CCTGA patient. We suggested that the main reasons for successful ablation in this case were good contact force and stability of ablation catheter through retrograde approach for morphologic RV.

We selected transaortic approach for ablation of morphologic RV because of the medical history of systemic atrioventricular tricuspid valve replacement. As long as non-structural heart disease, catheter ablation for PVC from RV was performed by antegrade approach. Therefore, this case was rare in the context of retrograde approach of right ventricular for catheter ablation.

Conclusion

We present a rare case of VPC originating from the MB of morphologic RV in CCTGA, which was eliminated by transaortic catheter ablation. Even in CCTGA, catheter ablation of PVC from the MB can be performed safely and accurately.

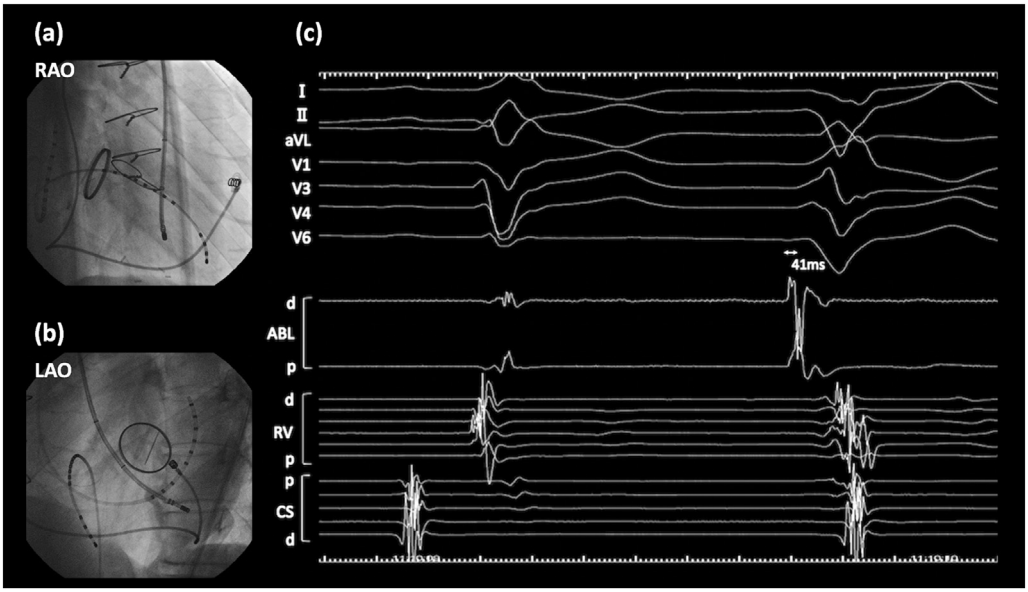

Fig. 3. Catheter ablation at midpart lateral of morphologic RV [RAO view (a), LAO view (b), at this site, a preceding ventricular potential is observed at the onset of clinical PVC (c)] (RAO: right anterior oblique, LAO: left anterior oblique, RV: right ventricle, PVC: premature ventricular contraction).

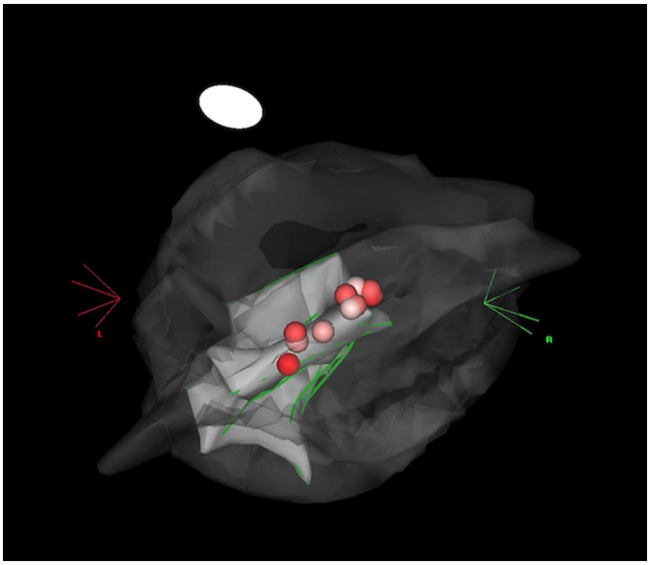

Fig. 4. The sites of ablation on the shell constructed by ICE integrated in CARTO system. Red tags show ablation sites. Light gray geometry shows the morphologic RV. Dark gray geometry shows the MB. (ICE: intracardiac echocardiography, RV: right ventricle, MB: moderator band).

Declaration of Competing Interest

None.

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