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Case Report

Successful catheter ablation of premature ventricular contractions originating from the anterior fascicle of the left bundle branch in a patient with hypertrophic cardiomyopathy

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ABSTRACT

A 73-year-old man with hypertrophic cardiomyopathy was referred for an electrophysiologic study and catheter ablation of premature ventricular contractions (PVCs)/nonsustained ventricular tachycardia (NSVT). The QRS morphology of the PVCs was right bundle branch block with an inferior axis. Transthoracic echocardiography showed left ventricular hypertrophy and a normal left ventricular ejection fraction without any obstruction in the left ventricle (LV). Intracardiac mapping showed that the earliest activation site of the PVCs was within the anterior portion of the basal LV, where a Purkinje potential preceded the QRS onset by 32 ms. Radiofrequency application at that site terminated the PVCs. The PVCs/NSVT did not recur during 10 months of follow-up.

1. Introduction

Sudden cardiac death, the most devastating feature of the natural history of hypertrophic cardiomyopathy (HCM), can be the first clinical manifestation of the disease in previously asymptomatic patients with HCM [1]. These observations have led to the use of antiarrhythmic treatment or radiofrequency (RF) ablation in patients with HCM and premature ventricular contractions (PVCs)/ventricular tachycardia (VT). Monomorphic VT in the presence of structural heart disease is mainly due to reentry involving the bundle branches or myocardial scar regions [2]. In contrast, episodes of repetitive idiopathic right and left ventricular outflow tract VT are attributable to abnormal automaticity or triggered activity of the ventricular myocardium [2]. Evidence shows that PVCs originating from the distal Purkinje arborization play an important role in the initiation of malignant arrhythmias such as polymorphic VT and ventricular fibrillation (VF) in patients with or without structural heart disease [2]. In the clinical setting of HCM, the mechanism underlying VT is often scar-related reentry [3,4]. However, here, we report the case of a patient who had HCM and developed symptomatic repetitive monomorphic PVC and nonsustained VT (NSVT), the mechanism of which was suggested to involve the Purkinje fibers.

2. Case report

A 73-year-old man with HCM experienced palpitations for 6 months. A 12-lead electrocardiogram (ECG) obtained at a medical clinic showed a first-degree atrioventricular block (PR interval, 260 ms), left ventricular hypertrophy (LVH) with strain-type negative T waves in V2–6 leads, and monomorphic PVCs. The PVCs had a right bundle branch block (RBBB) morphology and an inferior axis (Fig. 1A), suggesting that the origin was the anterior portion of the basal left ventricle (LV). A Holter ECG showed a total of 5101 PVCs (6% of the total beats) and 9 VT runs. Oral administration of a β-blocker did not suppress the PVCs. A signal-averaged ECG showed negative late potentials. Transthoracic echocardiography showed LVH with normal LV systolic function (LV ejection fraction, 70.6%; interventricular septum thickness, 15 mm; posterior wall thickness, 12.7 mm). The patient had no history of hypertension.

Cardiac magnetic resonance imaging showed global LV hypertrophy and mild hypokinesis of the apex (Fig. 2A). Late gadolinium enhancement (LGE) was observed in the LV apex region (Fig. 2B). The coronary angiography findings were normal, and left ventriculography showed no obstruction of the LV outflow or middle portions. In an electrophysiologic study, three diagnostic

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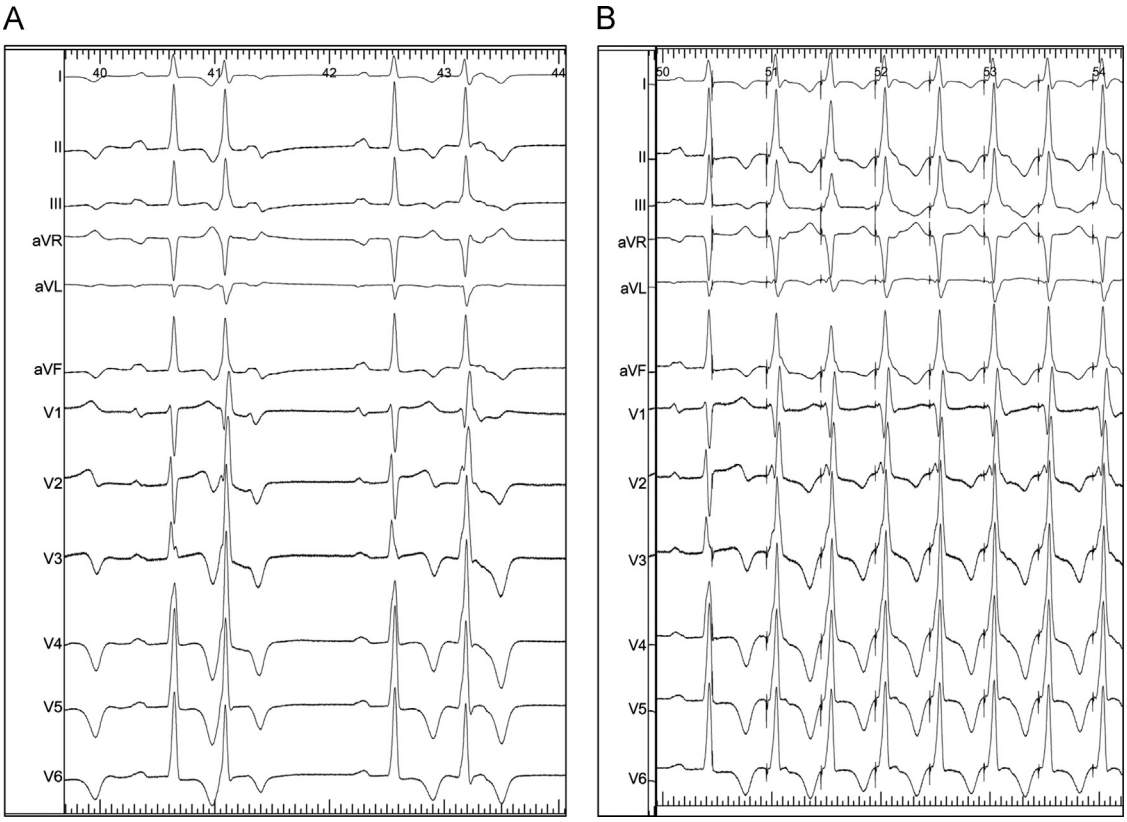

Fig. 1. (A) Twelve-lead electrocardiogram (ECG) obtained in sinus rhythm and premature ventricular contractions (PVCs). The QRS morphology of the PVCs was monomorphic with right bundle branch block and an inferior axis. (B) Twelve-lead ECG during pace mapping. The pace mapping results were 92% identical to the clinical PVCs.

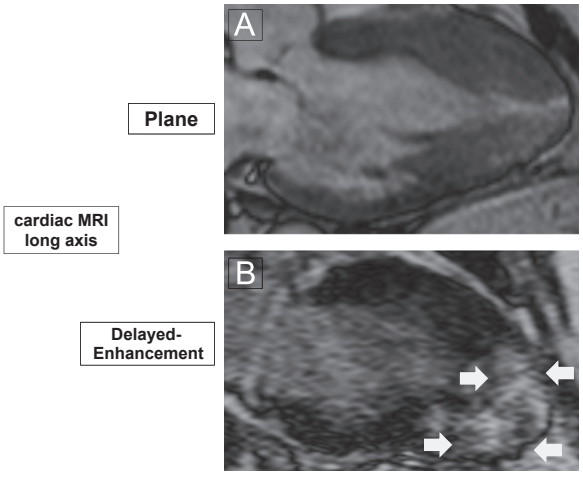

Fig. 2. Plane (A) and delayed-enhanced (B) cardiac magnetic resonance images (MRI). Late gadolinium enhancement was observed in the middle portion of the hypertrophic myocardial region of the left ventricular apex (arrows).

catheters were inserted from the femoral veins and placed in the high right atrium, His bundle position, and right ventricular apex. Endocardial bipolar electrograms were recorded at a filter bandwidth of 30–500 Hz. Electrograms were simultaneously recorded using the 12-lead surface ECG and stored in a computer system (LabSystem PRO; Bard Electrophysiology).

A spontaneous clinical PVC with an RBBB QRS morphology and inferior axis was observed. LV mapping and catheter ablation were performed with a 7-Fr deflectable quadripolar catheter (4 mm distal tip electrode, 1–7–4 mm interelectrode spacing, and embedded with a thermistor for the CARTO system [Biosense Webster Inc., Diamond Bar, CA, USA]). LV mapping during sinus rhythm identified a low voltage zone only in the anterior-to-lateral portions of the basal LV (Fig. 3A). Intracardiac ECG recorded a sharp presystolic Purkinje potential that preceded the earliest ventricular activation on the surface ECG by 32 ms during the PVC (Fig. 3B). During sinus rhythm, this site exhibited a small Purkinje potential just before the local ventricular activation (Fig. 3B) near the border zone around the low-voltage area (Fig. 3A). Pacing from that site exhibited a 11/12 pace match with the clinical PVC (Fig. 1B). RF delivery (60 °C, 50W, 60s) at that site abolished the PVC without any recurrence during rapid ventricular pacing, and no recurrence of the PVCs/NSVT was observed for 10 months without the use of any antiarrhythmic drugs or β-blockers.

3. Discussion

Here, we reported a case of PVCs/NSVT in a patient with nonobstructive HCM. This patient presented with PVCs/NSVT with a RBBB and inferior axis morphology that was successfully cured by targeting of the proximal portion of the anterior left bundle branch. HCM-related VT has been shown to result from scar-related reentry, the circuits of which originate from the LV-right ventricle junction around the interventricular septum and sometimes involve an LV aneurysm [4]. In dilated-phase HCM, the VT reentrant circuits are located in the basal septum or basal anterior-to-anterolateral LV area [5]. In this scar-related reentry in HCM, targeting the channel by using a combination of voltage-based substrate late/fractionated potential mapping during sinus rhythm and activation and entrainment mapping during VT effectively eliminates VT [3].

Regardless of the origin of the HCM-related VT, the VT circuits often involve the epicardium and endocardium [3,4]. Such arrhythmogenic substrates and increased LV thickness cause

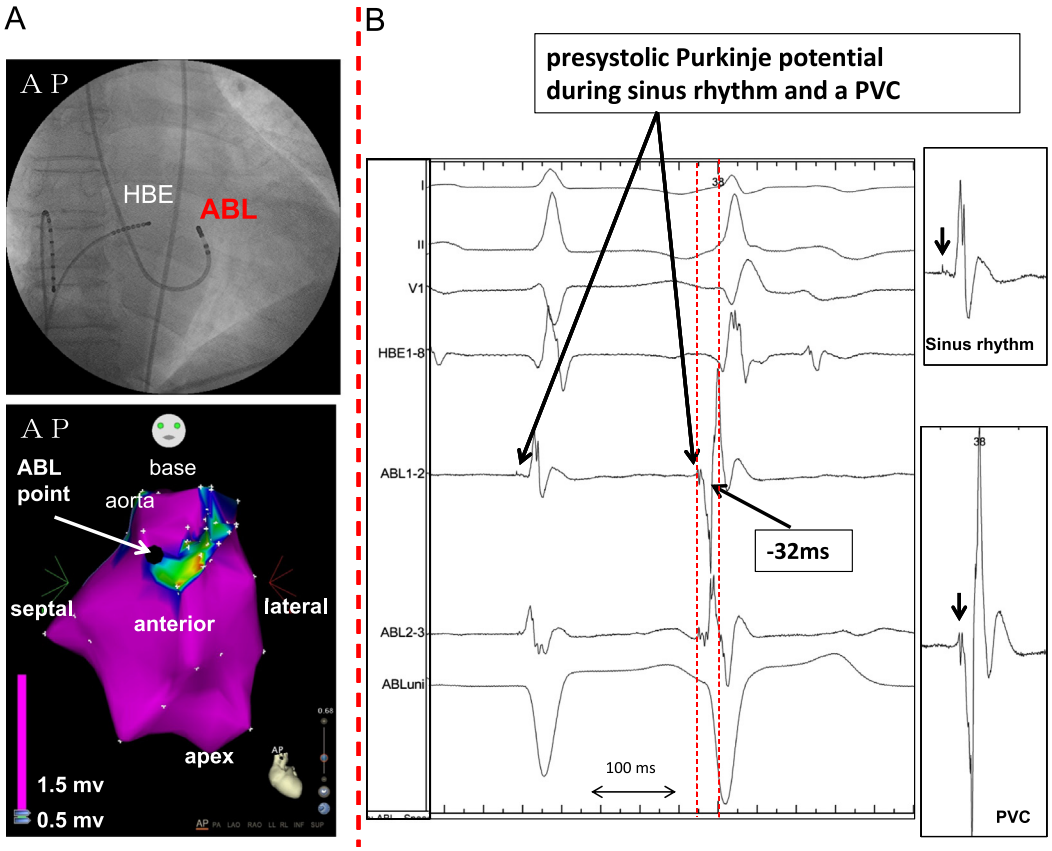

Fig. 3. Catheter position during the ablation (ABL) of premature ventricular contraction (PVCs) (A) and the intracardiac electrograms obtained during ablation (B). (A) ABL catheter position in the anteroposterior (AP) fluoroscopic view (top) and a CARTO image (bottom). The catheter was positioned in the anterior portion of the basal LV. (B) A sharp presystolic Purkinje potential was recorded; it preceded the earliest ventricular activation during surface electrocardiography by 32 ms during the PVC. During sinus rhythm (right upper panel), this site exhibited a sharp presystolic Purkinje potential just before local ventricular activation. HBE, his bundle electrogram and ABL, ablation.

difficulties in performing endocardial ablation of VT in patients with HCM [3,4]. In the present case, delayed-enhanced magnetic resonance imaging (MRI) showed an LGE in the LV apex (Fig. 2B), but intracardiac mapping showed a low voltage area only in the basal anterior-to-lateral LV area (Fig. 3A). In addition, a spike potential preceding the ventricular potential at that site during the PVC and sinus rhythm was reproducibly observed. Delivery of RF energy to that site terminated the PVCs/NSVT. These observations suggest that the PVCs/NSVT originated from the anterior fascicular Purkinje network area. Although the exact mechanism underlying the PVCs/VT remains unclear, the PVCs/VT occurred spontaneously, suggesting that its mechanism was triggered activity, abnormal automaticity, or (less possibly) unstable reentry within the Purkinje network.

One study reported the role of the Purkinje system with respect to the relationship between the septum and HCM-related VT. Santangeli et al. reported that patients with HCM also often have His-Purkinje conduction-related septal abnormalities, and bundle branch block often develops as part of the natural history of the disease [4]. Kim et al. reported that reentry within the Purkinje network adjacent to the papillary muscles played an important role in VF initiation and maintenance in a model using an isolated swine right ventricle [6]. Kuboki et al. and our group have reported cases wherein patients who had HCM showed torsades de pointes or monomorphic VT, the origin of which was suggested to be Purkinje fibers in the left bundle branch [2,7]. The present case report highlights the Purkinje network as the arrhythmogenic substrate even in HCM-related PVCs/NSVT; similar to other Purkinje-related PVCs/VTs, this condition can be cured by using endocardial ablation targeting the presystolic Purkinje potentials.

Conflict of interest

None.

Disclosures and finding sources

None.

References

[1] Wissner E, Stevenson WG, Kuck KH. Catheter ablation of ventricular tachycardia in ischemic and non-ischemic cardiomyopathy: where are we today? A clinical review Eur Heart J 2012;33:1440–50.
[2] Okumura Y, Watabnabe I, Ohkubo K, et al. Successful catheter ablation for incessant ventricular tachycardia in a patient with hypertrophic cardiomyopathy. Circ J 2007;71:1164–8.
[3] Dukkipati SR, d'Avila A, Soejima K, et al. Long-term outcomes of combined epicardial and endocardial ablation of monomorphic ventricular tachycardia related to hypertrophic cardiomyopathy. Circ Arrhythm Electrophysiol 2011;4:185–94.
[4] Santangeli P, Di Biase L, Lakkireddy D, et al. Radiofrequency catheter ablation of ventricular arrhythmias in patients with hypertrophic cardiomyopathy: safety and feasibility. Heart Rhythm 2010;7:1036–42.
[5] Ueda A, Fukamizu S, Soejima K, et al. Clinical and electrophysiological characteristics in patients with sustained monomorphic reentrant ventricular tachycardia associated with dilated-phase hypertrophic cardiomyopathy. Europace 2012;14:734–40.
[6] Kim YH, Xie F, Yashima M, et al. Role of papillary muscle in the generation and maintenance of reentry during ventricular tachycardia and fibrillation in isolated swine right ventricle. Circulation 1999;100:1450–9.
[7] Kuboki K, Ohkawa S, Chida K, et al. Torsades de pointes in a case of hypertrophic cardiomyopathy with special reference to the pathologic findings of the heart including the conduction system. Jpn Heart J 1999;40:233–8.