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Case Report

Ablation of idiopathic ventricular fibrillation targeting short coupled ventricular premature contractions originating from a right ventricular papillary muscle

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ABSTRACT

We describe a 38-year-old male who experienced several episodes of syncope after having ventricular fibrillation. The electrocardiographic monitoring after his hospitalization revealed repetitive polymorphic ventricular tachycardias. All polymorphic ventricular tachycardias were consistently initiated by a short-coupled monomorphic ventricular premature contraction (VPC). This VPC was suggested to originate from the inferoposterior region of the right ventricle (RV). Radiofrequency catheter ablation targeting the VPC was successfully performed, and the CARTO merge system (Biosense Webster Inc., Diamond Bar, CA, USA) revealed that the culprit region was the root of the posterior papillary muscle of the RV. A subsequent follow-up of 15 months has been uneventful.

<Learning objective: This is a case report of idiopathic ventricular fibrillation (IVF) triggered by a ventricular premature contraction (VPC) from the posterior papillary muscle of the right ventricle. We can learn about the relationship between the anatomical structure and the possible mechanisms of the short-coupled variant of Torsade de Pointes.>

Introduction

The short-coupled variant of Torsade de Pointes is a rarely described cause of idiopathic ventricular fibrillation (IVF). Further, the papillary muscles and Purkinje network sometimes play a key role in the initiation and maintenance of IVF [1]. In addition, radiofrequency catheter ablation (RFCA) targeting the initiating triggers of IVF has been shown to be effective [2–4]. However, it is difficult to identify the culprit area and investigate the detailed mechanism of IVF, especially in the right ventricle (RV), because recording of stable electrical potentials on the papillary muscle is difficult and the Purkinje potentials along the papillary muscle could hardly be recorded because these structures are densely surrounded by a massive complex myocardium [4]. We describe here a case of IVF from the root of the posterior papillary muscle of the RV, which was able to be clarified in detail and eliminated by RFCA.

Case report

A 38-year-old male resuscitated from ventricular fibrillation (VF) (Fig. 1A) was referred to our hospital. He had no family history of sudden cardiac death. The initial 12-lead electrocardiogram (ECG) showed sinus rhythm (SR) with normal QRS and QT intervals without any early repolarization. A physical test, laboratory examination, echocardiography, magnetic resonance imaging, and cardiac computed tomography (CT) revealed no cardiac abnormalities. With these clinical data, we diagnosed this patient with IVF.

Continuous ECG monitoring after admission revealed repetitive polymorphic ventricular tachycardias (pVTs) with the peak of the QRS complexes twisting around the iso-electric baseline. The pVTs were consistently initiated by an early coupled monomorphic ventricular premature contraction (VPC). The VPC morphology exhibited a superior axis and left bundle branch block, suggesting an inferoposterior wall origin in the RV (Fig. 1B). Before an implantable cardioverter defibrillator (ICD) implantation, RFCA targeting the VPC was performed to reduce the shock therapies.

At first, 3 multipolar catheters and a 4-mm-tip mapping catheter were inserted percutaneously. Three-dimensional

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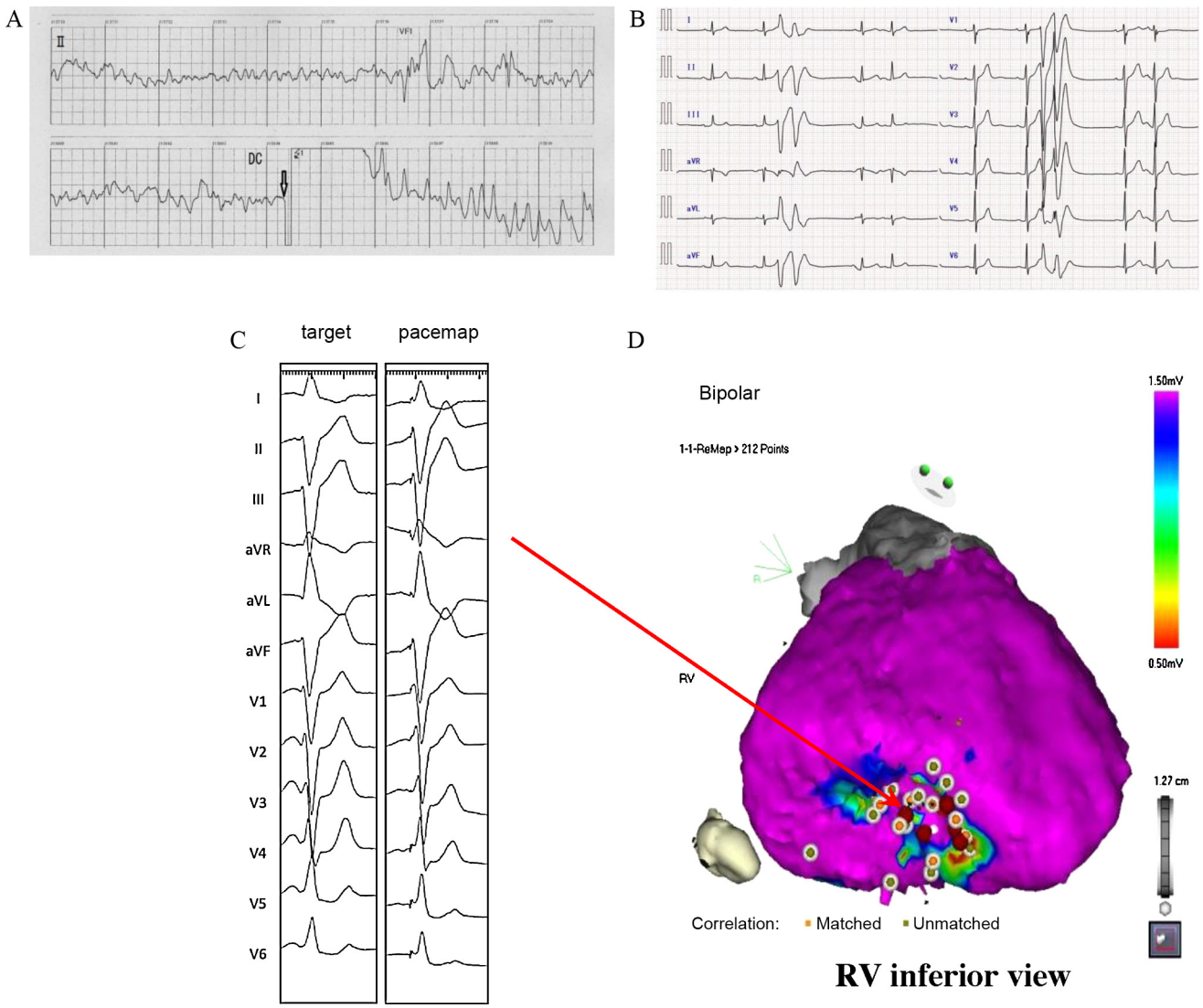

Fig. 1. (A) The patient received defibrillation for ventricular fibrillation. (B) The ventricular premature contractions (VPCs) that initiated the ventricular tachycardia (VT) were monomorphic. (C) Pace map of the right ventricle (RV) on the activation map. The best pace maps of the VPCs were obtained from the posterior region of the RV. (D) The voltage map of the RV. It shows a spotty low-voltage area around the targeted area in the posterior RV. The orange points indicate the matched points and the red points indicate where radiofrequency energy was applied.

mapping was performed using the electro-anatomic CARTO® mapping system (Biosense Webster Inc., Diamond Bar, CA, USA).

During the electrophysiological study, VPCs were rare. Therefore, after creating a voltage map of the entire RV, a pace map was performed on the RV. At the mid-portion of the RV inferior wall, the pace map was associated with an identical morphology to the target VPC (12.0/12.0) (Fig. 1C). The voltage map revealed a spotty low-voltage area around the targeted site (Fig. 1D). While the catheter was fixed at that site, we could record an intracardiac electrogram of a spontaneous episode of triplet. Although no evident Purkinje potentials were seen in SR, the ventricular electrogram split into two discrete local ventricle components with progression to VPCs. In the third VPC, a Purkinje-like prepotential preceded the local electrogram of the QRS by 70 ms (Fig. 2A). At that site, radiofrequency (RF) energy with a target temperature of 55 degrees Celsius and maximum power of 35 W was delivered. During the application of RF energy, nonsustained pVT flared up initiated with the same morphologic QRS (Fig. 2B). However, the pVT was no longer inducible after 5 applications.

Ultimately, it was revealed that the culprit region was located at the root of posterior papillary muscle of the RV, by observing the structure with multi-sliced CT combined with CARTO merge and the fluoroscopic image (Fig. 3).

This patient received an ICD before discharge owing to uncertainty about the long-term prognosis of ablation of IVF. The subsequent follow-up of 15 months has been uneventful; in addition, a genetic screening test did not detect any major channel disturbances including any mutations of the KCNQ1, KCNH2, and SCN5A genes.

Discussion

We reported a case of IVF originating from the root of the posterior papillary muscle of the RV. Further, RFCA successfully eliminated this arrhythmia.

IVF is rarely caused by short-coupled VPCs and RFCA targeting those VPCs is effective [2–4]. Regarding the mechanism of IVF without any apparent structural heart disease, Suwa et al. originally reported that there were fibrotic changes and local myocardial scarring on voltage maps of ventricular false tendons even in