Idiopathic ventricular fibrillation triggered by two distinct foci

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Idiopathic ventricular fibrillation (VF) is often triggered by short coupled ventricular premature beats originating from the ventricular Purkinje system or from the myocardium of the right ventricular outflow tract. We present a case of electrical storm due to idiopathic VF triggered from two distinct arrhythmogenic foci within the right ventricle. Successful radiofrequency ablation of both foci was performed.

Case presentation

A 51-year-old man with a history of idiopathic ventricular fibrillation (VF) and subsequent implantation of cardioverter-defibrillator (ICD) was admitted for electrical storm. Baseline electrocardiogram (ECG) showed sinus rhythm with normal conduction intervals and negative T waves in inferolateral leads. Echocardiography and coronary angiography did not show any significant results. Frequent episodes of VF preceded by a short coupled (280 ms) ventricular premature beat (VPB) with left bundle branch block morphology (QRS of 130 ms) and superior left axis (Figure 1; asterisk) were recorded.

Radiofrequency ablation of the triggering ectopy was indicated. The voltage endocardial map of both the ventricles did not show any area of abnormal low voltage/scar. Activation mapping localized the earliest activation during triggering VPB in the right ventricular lower septum. At this site, the Purkinje potential preceded local ventricular activation by 28 ms during VPB, confirming participation of the Purkinje network as the source of the arrhythmia trigger (Figure 1, endocardial signals). The radiofrequency energy was applied using an irrigated tip catheter up to 30 W and up to 60 s per site, applications of energy eliminated the ectopy and prevented subsequent episodes of VF.

On the next day, another episode of polymorphic ventricular tachycardia was recorded and frequent isolated VPBs re-appeared. However, their ECG characteristics were different from the previously ablated VPBs (coupling interval of 320 ms, QRS of 140 ms, left inferior axis) (Figure 1; hash sign). Therefore, the second ablation procedure was performed. The source of this triggering ectopy was localized into the right ventricular outflow tract (RVOT), approximately four centimeters remote from the previously mapped ectopy (Figure 1, electroanatomical map). The Purkinje potentials were not present at this spot. Radiofrequency energy delivery in this area abolished ectopic activity. The patient was discharged on beta-blockers and is free of episodes of arrhythmia during 5-month follow-up.

Discussion

This is a unique case of idiopathic VF that originated from two distinct arrhythmogenic foci. To our knowledge, this is the first report documenting the trigger site both in the Purkinje network and in the myocardium of the RVOT in one patient.

Both the Purkinje network and RVOT myocardium have been recognized as a site of triggering foci in idiopathic VF. In a series of 27 patients, VPBs originated from the left ventricle in 10, from the right in 9, from both ventricles in 4, and from the RVOT in 4 patients. The long-term outcome of larger cohort of patients has recently been published by Knecht et al. It comprised 38 patients, in whom the triggering VPBs originated from the right (n = 16), the left (n = 14), or both (n = 3) Purkinje network systems. Four of seven patients with recurrence of VF presented with other morphologies of VPBs at a median of 4 months. This was attributed to evolution of the disease. In our patient, the triggering focus of VF was dormant at the time of ablation and became manifest soon after. Although ablation successfully suppressed both ectopic foci, the delayed manifestation of the second trigger confirmed that catheter ablation cannot replace in this indication an ICD implant.

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References