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CASE REPORT

Kill two birds with one stone: curing accessory pathways and premature ventricular contractions with one ablation

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Key Clinical Message

Radiofrequency catheter ablation has been used for treating cardiac arrhythmias, such as premature ventricular contractions and accessory pathway. We report two cases with successful ablation of left-sided accessory pathways and premature ventricular contractions from mitral annulus with one ablation. To our knowledge, no similar reports have been found so far.

Keywords

Left-sided accessory pathways, premature ventricular contractions, radiofrequency catheter ablation

Premature ventricular contractions (PVCs) and accessory pathway-induced atrial-ventricular recurrent tachycardia (AVRT) are two common types of arrhythmias. Radiofrequency catheter ablation (RFCA) has been established as an effective and curative therapeutic strategy in treating these disorders. PVCs usually originate from different parts of ventricles, such as outflow tract, apex, and valve annulus. In some cases, PVCs originate from valve annulus could be located by means of Wolff–Parkinson–White syndrome manifest accessory pathway's algorithm. Here, we report two cases with successful ablation of left-sided accessory pathways and PVCs from mitral annulus with one ablation.

Electrophysiology Study

Informed consents were signed by each patient before cardiac electrophysiology procedure. After ceasing of antiarrhythmic drugs for more than five half-life periods, an intracardiac electrophysiology examination was performed under fasting state with local anesthesia. Heparin was used for anticoagulation during the whole procedure. One quadripolar and one decapolar diagnostic catheters (St. Jude Medical, St. Paul, MN 55117-9983, USA or Japan Lifeline Co., Ltd, Tokyo 140-0002, Japan) were, respectively, inserted into right ventricular apex and coronary sinus (CS) through right femoral vein and left subclavian vein. Programmed CS stimulation with extrastimuli was used for inducing tachycardia. The selection of the target sites for ablation was determined by the shortest AV/VA interval during sinus rhythm, AVRT, or right ventricular apex pacing with a 4-mm standard tip ablation catheter. The energy of RFCA was delivered by power of 35W and maximum temperature of 65°C.

Case 1

A 62-year-old man without structural heart disease was referred to our institution because of the long-standing episodes of palpitation. The standard 12-lead electrocardiogram showed frequent PVCs, and no other baseline abnormality was found (Fig. 1A). With Programmed CS stimulation, left concealed accessory pathway–mediated AVRT could be induced (Fig. 1B), and ventricular-atrial infusion was located at CS 5-6 which was also confirmed by RV pacing (Fig. 1C). The target site for ablation showed atrial:ventricular ratio as 1:4. Delivery of radiofre-

quency energy with an ablation catheter (35 W, 50–60°C, 60 sec) at the target site resulted in ventricular-atrial disassociation with right ventricular apex pacing, and no AVRT could be induced after ablation. Surprisingly, the spontaneous PVCs also disappeared. To confirm the origination of the PVCs, mechanical stimulation (Fig. 1D) with ablation catheter and pacing (Fig. 1D) the ablation catheter a little further into the left ventricle were used to compare the morphology of spontaneous and induced PVCs. As shown in the figures, the morphology of those PVCs was almost the same. The patient has been free from the same PVCs without any medications during 6-month follow-up.

Case 2

A 54-year-old man without structural heart disease presented a history of recurrent palpitations of 2 years was admitted to our laboratory. The standard 12-lead electrocardiogram showed pre-excitation syndrome and frequent PVCs (Fig. 2A). The morphology of PVCs was very similar with the QRS waves within pre-excitation electrocardiogram. During electrophysiology examination, it was revealed that the ventricular insertion was located at left posterior septum where local potential advanced surface QRS waves for about 20 ms with obvious QS-pattern

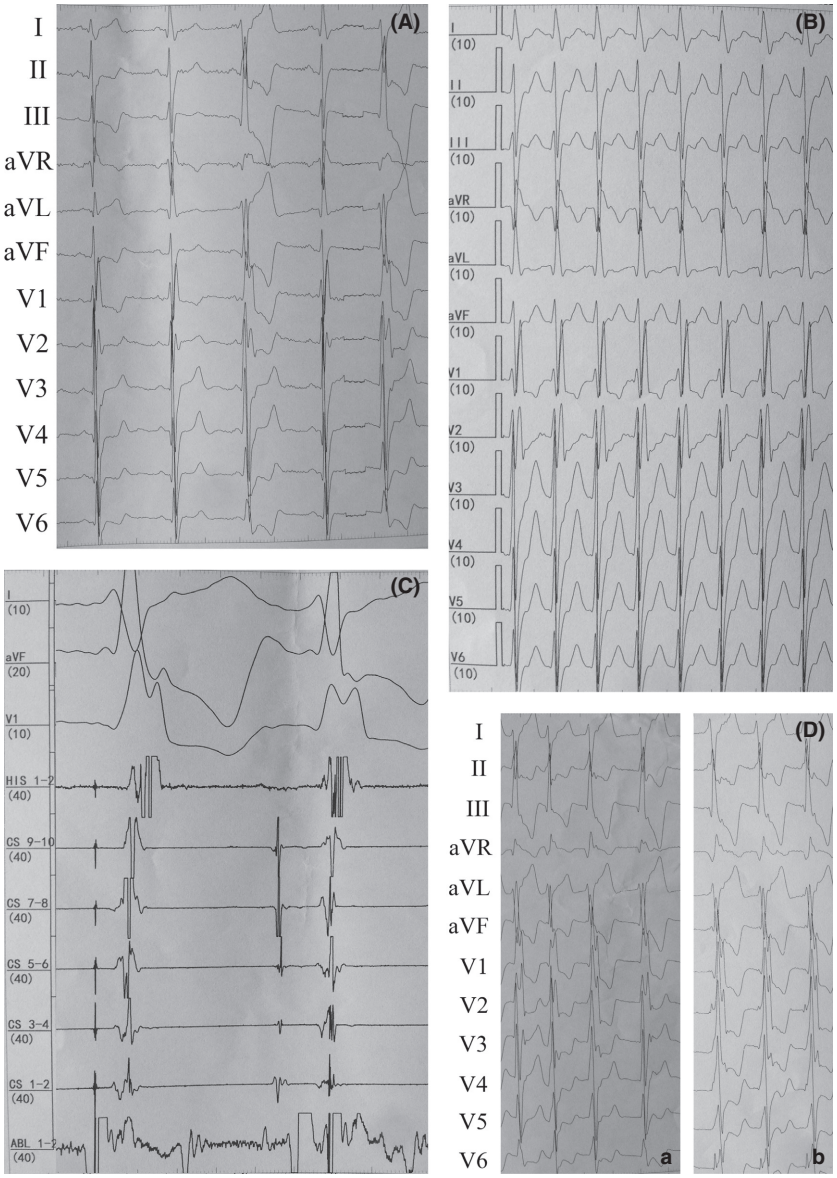

Figure 1. (A) Premature ventricular contractions shown by the standard 12-lead electrocardiogram; (B) tachycardia; (C) intracardiac electrogram of ablation target; (D) premature ventricular contractions with mechanical stimulation at ablation site (a) and pacing at ablation site (b).

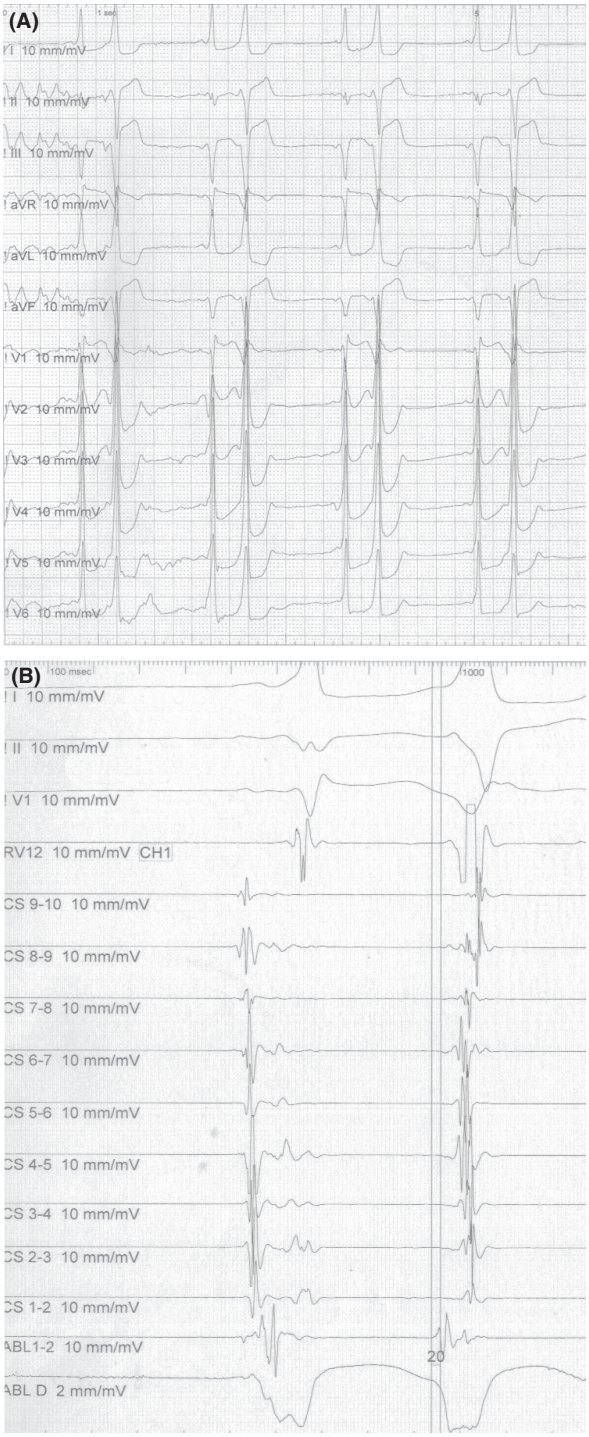

Figure 2. (A) Pre-excitation syndrome and frequent premature ventricular contractions shown by the standard 12-lead electrocardiogram; (B) intracardiac electrogram of ablation target.

unipolar potentials (Fig. 2B). Ablation at this site blocked the accessory pathway with pre-exciting conduction and also eliminated the PVCs at the same time. The patient has been free from the same PVCs without any medications during a follow-up period of 9 months.

Discussion

Radiofrequency catheter ablation has been established as an effective and reliable treatment of PVCs for decades [1]. In general, most of the PVCs originate from the ventricular outflow tract or left ventricular inferoseptal site [2]. Less commonly, PVCs can originate from the mitral annulus [3], the tricuspid annulus [4], Purkinje-fascicular network, left ventricular papillary muscles, and the moderator band in the right ventricle [5]. Usually, the PVCs and ventricular tachycardia originating from the valve annulus can be located by the accessory pathway's algorithm based on their electrocardiogram characteristics and be compared with the target site pacing after ablation [6]. The anatomic relationships of accessory pathways and ventricular muscle may cause disturbance of electrical activity of focal cardiac muscle cells in specific patients. During the accessory pathways ablating, the abnormal ventricular insertion which caused focal electrical abnormality was also ablated. In the two procedures we presented, the intracardiac electrogram confirmed that both accessory pathways and PVCs originating from the same site of the heart. As report above, the morphology of pacing induced PVCs was almost the same as spontaneous ones. However, the exact mechanism of this phenomenon is still unknown. Many patients were reported to have accessory pathways during electrophysiology examination. However, reports of individuals with PVCs originating from the same anatomic sites as accessory pathways were rare. Possibly there are some underlining mechanism and network between PVCs and accessory pathway which could not be recognized at this time. Further clinical studies are surely needed to explain this phenomenon.

Conflict of Interest

None declared.

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