Case Report

The main trunk of the left bundle branch is not part of the re-entry circuit of verapamil-sensitive idiopathic left ventricular tachycardia

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1. Introduction

Idiopathic left ventricular tachycardia (ILVT) with a right bundle-branch block (RBBB) configuration and superior or left axis is a distinct entity that arises in the left ventricle, mostly because of a reentry mechanism and is usually verapamil sensitive \cite{1,2,3,4,5,6}. Although this type of tachycardia has been suggested to originate from the Purkinje network \cite{1,5}, its precise re-entry circuit and slow conduction zone remain unclear. However, radiofrequency (RF) catheter ablation has been reported to successfully eliminate this arrhythmia from the left-hand side of the interventricular septum, where the earliest Purkinje potential (PP) and late diastolic potential are recorded during ventricular tachycardia (VT). In addition, catheter ablation targeting of the left posterior fascicle was shown to be another therapeutic strategy for curing ILVT \cite{7}. These observations raise the possibility that anterior or posterior fascicles compose a critical part of the re-entry circuit of ILVT. However, there have been no reports describing the role of the main trunk of the left bundle branch in the mechanism of ILVT. In this report, we present results indicating the role of main trunk of the left bundle branch in the re-entry circuit of ILVT in a patient.

2. Case report

A 19-year-old man was referred to our hospital in June 2006 with a 3-year history of paroxysmal palpitations. An ambulatory electrocardiogram monitor showed a regular wide QRS tachycardia. The patient was admitted to our hospital for an electrophysiological study and RF catheter ablation. An initial physical examination revealed normal findings and the laboratory parameters were within normal limits. A standard 12-lead electrocardiogram obtained during sinus rhythm showed no abnormalities; other examinations, including a chest roentgenogram and echocardiogram, showed no evidence of structural heart disease or other abnormalities. After obtaining written informed consent, we performed an electrophysiological study and catheter ablation for the regular wide QRS tachycardia. Under fluoroscopic guidance, a 4-Fr quadripolar electrode catheter, two 5-Fr decapolar electrode catheters, a 5-Fr quadripolar electrode catheter (Irvine Biomedical Inc., Irvine, CA, USA), and a 2.5-Fr micro-sized mapping catheter with 8 electrodes (Cardima Inc., Fremont, CA, USA) were introduced into the high right atrium, His-bundle region, left ventricular septum, right ventricular apex, and coronary sinus, respectively, through the femoral veins and artery. The intracardiac electrocardiogram showed an atrial-His interval of 85 ms and a His-ventricular interval of 47 ms at baseline. Right atrial burst pacing and right and left ventricular burst pacing easily induced tachycardia with a QRS duration of 125 ms, an RBBB pattern, inferior axis, and a cycle...
length of 320 ms (Fig. 1). Ventriculoatrial dissociation was observed during tachycardia and the diagnosis of VT was subsequently established. Ventricular stimulations from the right ventricular apex with constant cycle lengths 10, 20, and 30 ms shorter than that of the VT cycle length were performed during VT and manifest entrainment was confirmed. In addition, a PP preceding ventricular activation and dull mid-diastolic potential preceding PP (pre-PP) were recorded simultaneously during the VT at the mid-septum of the left ventricle, which is characteristic of verapamil-sensitive ILVT. During left ventricular outflow mapping, we observed a slight spontaneous conversion of the QRS morphology during VT with prolongation of the QRS duration from 125 ms to 143 ms and an identical inferior axis and tachycardia cycle length. A reverse morphological change of the QRS was observed as well. Intracardiac electrograms showed that there were no differences in the left ventricular activation sequences between VT with narrow QRS duration and that with wide duration (Fig. 2). RF catheter ablation and left ventricular mapping were performed using a 7-Fr quadripolar ablation catheter with a 4-mm distal electrode, an embedded thermistor, and a deflectable tip (Marinos®, Medtronic Inc., Minneapolis, MN, USA). Left ventricular endocardial mapping at the left ventricular outflow tract accidentally produced a complete left bundle-branch block (CLBBB), and thereafter, only VT with a wide QRS duration of 143 ms was induced (Fig. 3). In addition, no conversions of the QRS morphology and no changes of the VT cycle length were observed. The earliest ventricular activation during VT was found at the apical septum of the left ventricle, and RF catheter ablation at the basal third of the mid-septum, where pre-PP and PP were recorded during tachycardia, successfully eliminated the VT. After ablation, a new diastolic potential appeared during sinus rhythm at the lower and more basal site compared with the successful ablation site; this has been observed in some patients with verapamil-sensitive ILVT after successful ablation [8].

Our study patient underwent follow-up without any antiarrhythmic medication and has not experienced VT recurrences during the 60-month follow-up period.

3. Discussion

The major finding in this case is that the incidental occurrence of CLBBB in a patient with verapamil-sensitive ILVT had no effect on the inducibility, maintenance, or cycle length of the VT. On the
other hand, it changed the QRS morphology of the VT with a wider QRS duration. These findings indicate that the main trunk of the left bundle branch is not a critical component of the re-entry circuit of verapamil-sensitive ILVT.

The mechanism of verapamil-sensitive ILVT has been suggested as re-entry with an excitable gap and a slow conduction, because the VT can be induced, entrained, and terminated by programmed ventricular or atrial stimulation [1–6]. In addition, previous studies have suggested that the re-entry circuit is confined to the posterior Purkinje system and that the ventricular myocardium is involved in the circuit [5–7]. Previous studies have shown successful catheter ablation achieved at the site of the earliest recorded PP or pre-PP, particularly at the inferoposterior left ventricular septum during sustained VT [5,9], and at the site of abnormal potential in the mid-inferior septum within the posterior fascicular network during sinus rhythm [6]. In our case, catheter ablation could eliminate VT and was performed at the basal third of the mid-septum, where pre-PP and PP were recorded during tachycardia. However, the entire re-entrant circuit and slow conduction zone remain unclear, and there have been no reports describing patients with verapamil-sensitive ILVT in the presence of CLBBB. To the best of our knowledge, this is the first report of verapamil-sensitive ILVT, which was not influenced by the incidental occurrence of CLBBB.

The slight morphological change of the VT with QRS widening occurred transiently during LV outflow mapping and was perpetuated by the occurrence of CLBBB. However, the LBBB did not affect other VT characteristics, such as left ventricular activation pattern, cycle length, VT induction, or maintenance. This suggests that the conduction block of the main trunk of the left bundle branch did not have any effects on VT circuit, and it changed the ventricular activation pattern by prolonging the conduction time from the VT circuit to the right ventricle. This might also raise the possibility that the re-entrant circuit of verapamil-sensitive ILVT was localized within the left ventricle.

In the present case report, we showed that the left main trunk of the His-Purkinje system is not a critical component of the re-entry circuit of verapamil-sensitive ILVT.

Conflict of interest

No conflict of interest declared.

References