CASE REPORT

Reversible left ventricular dyssynchrony and dysfunction resulting from right ventricular pre-excitation

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We report observations in a 7-year-old girl with right ventricular pre-excitation due to an accessory atroventricular pathway (AP), and depressed cardiac function. Echocardiographic findings consisted of left ventricular (LV) dilatation with asynchronous ventricular wall motion and diminished LV ejection fraction. Electrophysiological study revealed a para-Hisian AP. She underwent successful AP ablation, after which asynchronous ventricular wall motion disappeared. Cardiac size and function were normal after 3 months follow-up. These findings suggest that ventricular pre-excitation leading to asynchronous ventricular motion was a possible cause of LV dilation and dysfunction and that catheter ablation reversed undesirable cardiac remodelling in this patient.

KEYWORDS
Pre-excitation; Accessory pathway; Catheter ablation; Cardiomyopathy

Introduction

Supraventricular tachycardia (SVT) is very common in patients with accessory atroventricular pathways (APs), and in occasional patients, the tachycardia can be incessant and induce a cardiomyopathy. On the other hand, although occasional episodes of SVT are not usually associated with the development of ventricular dysfunction, the possibility exists that abnormal ventricular activation resulting from antegrade conduction over an AP (ventricular pre-excitation) could cause sustained atrial-ventricular, inter-ventricular, and intraventricular dyssynchrony. In this report, we present observations in a patient with right ventricular pre-excitation and depressed cardiac function whose left ventricular (LV) function substantially improved after successful AP catheter ablation.

Case report

A 7-year-old girl was referred to our hospital after two fainting episodes during vigorous exercise within the previous 6 months. She felt palpitation before each fainting episode. There was no family history of cardiomyopathy and no history of her having had a viral illness in the recent past. The troponin I was in normal range. An electroencephalogram was obtained as part of the evaluation for possible seizure disorder, and the result was negative. Her electrocardiogram (ECG) revealed a short PR interval (80 ms) and long QRS duration (180 ms) (Figure 1, left panel). A delta wave was apparent, and the overall QRS appearance suggested a left bundle branch block (LBBB) appearance, compatible with right ventricular pre-excitation. Echocardiogram (Echo) revealed LV dilatation (LVd = 52 mm), reduced LV ejection fraction (LVEF = 46%), and moderate mitral valve regurgitation. Tissue Doppler imaging (TDI) revealed asynchronous LV wall motion.

The patient underwent electrophysiology study and an AP ablation procedure on the fifth hospital day. Electrophysiology study showed a para-Hisian AP (Figure 2) with both antegrade and retrograde conduction properties. The AP did not exhibit decremental conduction, and effective refractory periods were identical (300 ms) in both antegrade and retrograde directions. An orthodromic SVT with LBBB (aberrant conduction) was induced by right ventricular apex programmed stimulation (Figure 3). During the SVT episode, her symptoms were similar to those experienced during spontaneous tachycardia.

A sharp QS deflection preceding the surface ECG delta wave onset by 20 ms was present on the unipolar electrogram at the successful ablation site. The delta wave disappeared during radiofrequency energy delivery (temperature setting was 55°C, and the energy titrated from 10 to 30 W), and a low-amplitude His deflection was recorded on the bipolar electrogram (Figure 4). After ablation, SVT could not be induced in the drug-free state or during isoproterenol infusion. Normal atrial-ventricular conduction was documented at the end of the procedure. Prior
to discharge, an Echo showed that the L VEF had slightly improved (53%), and TDI indicated that asynchronous ventricular wall motion had disappeared. She was discharged without any medication.

At the 6 month follow-up visit, she remained free of symptoms. A delta wave was no longer observed on the ECG recording (Figure 1, right panel), and echocardiographic findings revealed that both her cardiac size and L VEF had returned to normal (L VDd = 44 mm, L VEF = 63%).

**Discussion**

The principal finding in this report was the resolution of cardiac chamber enlargement and normalization of LV function within a few months of successful elimination of right ventricular pre-excitation by radiofrequency ablation in a young patient who presented with a cardiomyopathic picture. In this case, a paroxysmal supraventricular tachyarrhythmia function was the most possible reason for fainting episode, especially, given the diminished LV function. However, the mechanism of her LV dilation and ventricular dysfunction was not so readily explained given the absence of evidence for familial disease, a recent illness (e.g. causing myocarditis), or a drug-induced cardiomyopathy. In addition, incessant tachycardia causing a tachycardia-induced cardiomyopathy seemed unlikely given no history of long-standing palpitation and the findings at EP study that excluded susceptibility to asymptomatic incessant tachycardia such as that associated with the permanent form of functional reciprocating tachycardia. On the other hand, since her ECG showed

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**Figure 1**  The manifest right-sided accessory pathway led to reversible left ventricular dyssynchrony and dysfunction. (Left panel) Before ablation, her electrocardiogram displayed an apparent delta wave which exhibited left bundle branch block with short PR interval (80 ms) and long QRS duration (180 ms). Echocardiogram showed left ventricular dilation with asynchronous wall motion and function reduction. (Right panel) After ablation, the electrocardiogram showed normal PR interval (160 ms) and QRS duration (80 ms). Her cardiac structure and function was normal on echocardiogram from the third month follow-up.
Constant LBBB-like pattern with short PR interval due to manifest right ventricular pre-excitation, it seemed reasonable to attribute the reversible LV dysfunction to that cause. Left bundle branch block causes asynchronous ventricular wall motion, which has the potential to progress to ventricular dysfunction and mitral regurgitation. In this regard, Blanc et al. recently demonstrated reversibility by LV-based pacing of an apparent cardiomyopathic picture triggered by long-standing LBBB in patients with no other evident cause for LV dysfunction. Presumably, effective ‘elimination’ of the LBBB pattern, and its associated intraventricular dyssynchrony (as we hypothesize in our patient), was the basis for LV functional improvement. In addition, it has become widely accepted on the basis of relatively recent clinical trials that long-term right ventricle apical pacing promotes heart failure progression due to the deterioration of LV function. Again, it seems likely that pacing results in a scenario essentially equivalent to LBBB-induced ventricular dyssynchrony and adverse effect on LV structure and function. Finally, there have been several reports describing patients with frequent (>20%) premature ventricular complexes from right ventricle outflow tract (ROVT-PVCs), LV dilation, and LVEF reduction. Successful catheter ablation of PVCs reversed their LV dilatation and dysfunction. It is presumed that the ROVT-PVCs, which manifested LBBB-like morphology, gave rise to LV dyssynchrony and dysfunction.

On the basis of these previous observations, it seems plausible that right ventricular pre-excitation in our patient led to interventricular and intraventricular dyssynchrony. The short PR interval (80 ms) also could give rise to atrial-ventricular dyssynchrony. After successful ablation, her PR interval and duration of QRS complex became normal; Echo verified that dyssynchrony wall motion had disappeared.

Conclusions

Given the previous reports regarding the potential impact of LBBB or LBBB-like activation pattern on ventricular function, as well as our observation in this patient, it seems possible that APs producing right ventricular pre-excitation leading to comparable ventricular dyssynchrony could be a possible cause of reversible LV dilatation and ventricular dysfunction. Consequently, in patients with a manifest right-sided AP and depressed cardiac function, catheter ablation

![Coronary sinus pacing. In this and subsequent figures, surface leads 1, aVF, and V1 are shown with intracardiac recording from high right atrium (HRA), His bundle (H), coronary sinus (CS, proximal; CSm, middle; CSd, distal), and ablation electrode (ABL, unipolar; ABLb, bipolar); S denotes stimulus artefact, H the His deflection, A the atrial deflection, and V the ventricular deflection. The His tracing showed the atrial-ventricular deflection fusion with a possible atrioventricular pathway potential in sinus rhythm, the normal A-H-V sequence was presented during CS pacing (S1S1 = 300 ms), because the effective refractory period of atrioventricular pathway was longer than the atrioventricular node and atrioventricular pathway block. This suggested that the atrioventricular pathway was near the His bundle (parahisian) with antegrade conduction. The surface electrocardiogram also showed left bundle branch block due to aberrant conduction during coronary sinus pacing.](image-url)
Figure 3  Orthodromic supraventricular tachycardia with left bundle branch block (aberrant conduction). The His tracing showed the shortest VA interval (or VA fusion) during supraventricular tachycardia episode (201 bpm). This supports the finding that the atrioventricular pathway originated from a para-Hisian site and exhibits retrograde conduction.

Figure 4  Successful ablation site of para-Hisian atrioventricular pathway. Pre-ablation, the QS deflection, which preceded surface electrocardiogram delta wave onset by 20 ms, showed on ABLu tracing; during ablation, delta wave disappeared, and a little H-deflection showed on ABLb tracing.
to eliminate pre-excitation may have beneficial effects on cardiac function beyond eliminating their susceptibility to recurrent tachyarrhythmias.

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References