Progressive Facilitation of Antegrade Conduction via an Accessory Pathway in a Patient with Wolff-Parkinson-White Syndrome and Permanent Atrial Fibrillation

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Abstract

The case of a 64-year-old man with Wolff-Parkinson-White syndrome and permanent atrial fibrillation (AF) is reported. The patient was admitted due to electrocardiographic feature of AF with rapid conduction over the left-sided accessory pathway. Administration of pirmenol effectively suppressed the ventricular response via an accessory pathway. A transesophageal echocardiography detected an uncertain thrombus in the left atrial appendage. During the 33-month follow-up period, the ventricular response via an accessory pathway was progressively facilitated. Radiofrequency catheter ablation using a transseptal approach was performed during AF, resulting in complete elimination of the antegrade accessory pathway conduction.

Case Report

A 64-year-old man was admitted to our Institute in January 2001 due to an electrocardiographic feature of AF with rapid conduction over the left-sided accessory pathway (Fig. 1A). There was no information as to when the AF had initially begun. When admitted, oral administration of pirmenol (200 mg/day) had already been started and it effectively suppressed the antegrade accessory pathway conduction. An RFCA was planned after electrical conversion of AF. However because the transesophageal echocardiography (TEE) detected an uncertain thrombus formation (5 mm in diameter) in the left atrial appendage (LAA), it was postponed. The patient was treated at the outpatient clinic with combined administration of pirmenol and warfarin. However, the uncertain thrombus in LAA was refractory to treatment with warfarin. During the 33-month follow-up period, the ventricular response through the accessory pathway was progressively facilitated in spite of the administration of pirmenol at the same dosage (Fig. 1B). In October 2003, he was readmitted to hospital as his electrocardiogram (ECG) showed rapid ventricular response, with the RR intervals of less than 280 ms (Fig. 1B). Ambulatory 24-h ECG recording revealed an average daily heart rate of 129 beats/min. Echocardiography disclosed enlargement of the left ventricle (LV) (LV internal dimension at end-diastole; LVIDd =66 mm) and diffuse hypokinesis of the LV wall (LV ejection fraction; LVEF =34%). Because the uncertain thrombus in...
LAA was still detected by the TEE, we decided to perform RFCA during AF. A 5-French decapolar catheter with a 1-5-1 mm interelectrode spacing between each electrode pair (Daig Corp.) was inserted into the coronary sinus (CS). Two 5- and 4-French, four-pole catheters with 2-mm interelectrode spacing were placed at the His-bundle area (HB) and at the right ventricular apex (RVA), respectively. Access of a 7-French quadipolar mapping-ablation catheter (Navisitar™, Biosense-Webster) to the left atrium was achieved with an atrial transseptal puncture. Mapping of the mitral valve annulus was performed via a long sheath (8-French, Daig Corp.). Localization of the accessory pathway was evaluated by the QS complex on the unipolar electrogram and a rapid deflection preceding the onset of ventricular activation on the bipolar electrogram (accessory pathway potential). The QS complex was obtained over a wide area between the lateral and posterior regions. However, the accessory pathway potential was obtained exactly at the posterolateral region of the mitral valve annulus (Fig. 2). Antegrade accessory pathway conduction was successfully ablated immediately after the first RF pulse application. The rate of ventricular response through the atrio-ventricular node after the RFCA was about 80 beats/min. The patient was free of anti-arrhythmic drugs and has been treated with warfarin and beta-blocker (carvedilol, 5 mg/day). Carvedilol was administered in expectation of a reduction of LV internal dimension and an improvement in cardiac performance, combined with a reduction in the ventricular response through the atrio-ventricular node. One month after RFCA, LVIDd was reduced to 62 mm, with an improvement in LVEF of up to 52%, assessed by echocardiography. ECG showed AF rhythm with an appropriate ventricular response rate (Fig. 3). Ambulatory 24-h ECG recording revealed an average daily heart rate of 73 beats/min. During a follow-up period of 20 months, no recurrence of antegrade accessory pathway conduction was observed.
The most interesting finding of the present case is the progressive facilitation of the ventricular response through an accessory pathway. However, the mechanism for this observation remains unclear. Antegrade accessory pathway conduction can be facilitated by the progression of heart failure. However, during the follow-up period, the progression of

Discussion

The most interesting finding of the present case is the progressive facilitation of the ventricular response through an accessory pathway. However, the mechanism for this observation remains unclear. Antegrade accessory pathway conduction can be facilitated by the progression of heart failure. However, during the follow-up period, the progression of
heart failure was not evident, by symptoms, physical examination, chest X-ray, or echocardiography, although we did not assess the plasma levels of norepinephrine and brain natriuretic peptide. Supposing that heart failure was substantially progressed, it is difficult to explain the selective facilitation of antegrade conduction via an accessory pathway but not through the atrio-ventricular node. In fact, the rate of ventricular response through the atrio-ventricular node after the RFCA was about 80 beats/min, which was a little faster than that before the progression of antegrade accessory pathway conduction. This observation also appears to deny the hypothesis that the gradual decrease in antegrade conductivity through the atrio-ventricular node caused relative facilitation of accessory pathway conduction. During the follow-up period, the patient was continuously treated with 200 mg/day of pirmenol. Therefore, our observation might be explained by a gradual decrease in the sensitivity of the accessory pathway to pirmenol, although we found no reports on the gradual reduction in the suppressive effects of pirmenol on the accessory pathway. Nevertheless, it may be postulated that the serum concentrations of pirmenol were reduced for some reason, including activation of its metabolizing enzyme. On the other hand, recent evidence has shown that the high frequent depolarization of atrial cardiomyocytes results in a shortening of the refractory period with alterations of ionic currents (5, 6). In the present case, it can be postulated that the frequent depolarization of accessory pathway myocytes, as observed from June 2002 (Fig. 1B), altered the electrophysiological characteristics to enhanced excitability and shortening of the refractory period, resulting in excessive conduction via an accessory pathway.

Regarding therapeutic strategies, we chose the RFCA with a transseptal approach. During the procedure, we were careful to prevent the catheters from entering the LAA. When compared to a transaortic approach, the transseptal approach has the advantage of smoother mapping of the mitral valve annulus. Also, there was the possibility that during the transaortic approach contact of the ablation catheter with the endocardial left ventricle might accidentally induce ventricular fibrillation. There have been a few reports of RF catheter ablation of an accessory pathway during AF. Hindricks et al (7) reported 19 patients with Wolff-Parkinson-White syndrome who had ongoing AF with rapid antegrade conduction over the accessory pathway during RFCA. In these patients, however, only one had permanent AF, whose antegrade accessory pathway conduction recurred one day after the
Regarding the electrophysiological criteria for catheter ablation, Hindricks et al (7) reported that the presence of an accessory pathway potential is important, while a QS configuration of the unipolar electrogram could be recorded over a wider area. In the present case, a QS configuration was recorded consistently over a wide area, while the accessory pathway potential was obtained just at the point where the RFCA succeeded.

There are several uncertainties in this report. First, the serum concentration of pirmenol was not monitored and, secondly, a transaortic approach might have been better to completely avoid reaching the LAA.

The association of Wolff-Parkinson-White syndrome and permanent AF is rare. To our knowledge, this is the first report demonstrating progressive facilitation of antegrade conduction through the accessory pathway in patients with Wolff-Parkinson-White syndrome.

References