Successful Catheter Ablation for Paroxysmal Atrial Fibrillation Originating from Superior Vena Cava in a Patient with Brugada Syndrome

Toshiya Kurotobi MD PhD, Hiroshi Ito MD PhD, Katsuomi Iwakura MD PhD, Shigeo Kawano MD PhD, Atsunori Okamura MD PhD, Koichi Inoue MD PhD, Hiroyuki Nagai MD, Kensi Fujii MD PhD

Division of Cardiology, Sakurabashi Watanabe Hospital, Osaka, Japan

This case report describes the treatment of atrial fibrillation (AF) in a patient with Brugada syndrome. We found the triggers of AF in the superior vena cava during electrophysiological study and the isolation of superior vena cava with catheter ablation successfully eliminated the recurrence of AF. This case suggests that abnormal myocardial repolarization due to Na\(^{+}\)-channel abnormality is not always the etiology of AF in patients with Brugada syndrome. In case of the focal trigger related to the initiation of AF, catheter ablation can be an effective therapeutic strategy.

(J Arrhythmia 2005; 21: 470–474)

Key words: Atrial fibrillation, Superior vena cava, Catheter ablation, Brugada syndrome

Introduction

It is reported that myocardial abnormal repolarization due to Na\(^{+}\)-channel abnormalities is associated with the occurrence of atrial fibrillation (AF) and ventricular fibrillation (VF) in patients with Brugada syndrome (BS).\(^{1,2}\) Recently, Haissaguerre et al. suggested ablation of the foci of premature beats preceding the ventricular tachyarrhythmias is associated with better clinical outcomes in patients with BS.\(^{3}\) It remains unknown that such focal triggers also contribute to AF in patients with BS.

Case Report

A 55-year-old man complained of paroxysmal palpitation for 15 years. He was diagnosed with paroxysmal AF 5 years ago, and received disopyramide. He was referred to our hospital due to sudden and severe palpitation and dizziness on August 17th, 2004. In the ambulance, the ECG showed monomorphic wide QRS tachycardia (Figure 1). Just before the hospital arrival, wide QRS tachycardia turned to narrow QRS morphology and 12-lead ECG showed coved-type ST segment elevation in V\(_1\) and V\(_2\) leads (Figure 2). Atrial rhythm suspected of atrial flutter spontaneously changed to sinus rhythm. Chest X-ray finding and laboratory data were normal. Echocardiographic findings were also normal (left atrial dimension = 32 mm, left ventricular ejection fraction = 69\%). After the intravenous administration of pilscainide, further elevation of coved-type...
ST segment was noted in V₁–V₃ (Figure 3) and he was diagnosed with BS. He had no family history of sudden cardiac death. He had experienced syncope once when he was 53 years old.

After obtaining written informed consent, we conducted the electrophysiological study. After intravenous isoproterenol infusion (3.0 mcg/min), we found the premature beats originated from the superior vena cava (SVC), followed by atrial tachycardia (Figure 4). Atrial tachycardia spontaneously shifted to AF (Figure 5). We found local high frequency and centrifugal activation within SVC and pulmonary veins. The activation patterns were periodical. Thus, we conducted electrical segmental isolation for the ostium of the SVC and for each pulmonary vein. The isolation procedure for SVC breakthrough site was accomplished by two radiofrequency energy applications targeted at 50 degrees within 30 W (Japan Lifeline, Abraze, Tokyo). Although the abnormal activation remained within SVC, sinus rhythm was maintained after the electrical SVC isolation (Figure 6). We could not induce ventricular tachycardia or atrial flutter with the standard programmed electrical stimulation protocol. Programmed ventricular stimulation was performed consisting of single, double and triple extrastimuli during paced rhythm with basic cycle lengths of 600 and 400 ms; and burst pacing up to 240 beats per minute with and without isoproterenol. HV interval was not significantly prolonged (45 ms). Implantable cardioverter defibrillator (ICD) was not implanted because of patient’s rejection. The patient was discharged without the medications and maintained good condition without recurrence of AF and ventricular tachyarrhythmia (follow-up period: 12 months).

Discussion

To our knowledge, this is the first case report
documenting that AF foci was identified and was successfully treated with catheter ablation in a patient with BS. BS is an arrhythmogenic disease characterized by the right bundle branch block with ST-segment elevation in right precordial leads and with a high risk of life threatening ventricular tachyarrhythmia.\textsuperscript{4–7}\) AF also sometimes occurs in patients with BS.\textsuperscript{2,8}\) Na\textsuperscript{+} channel abnormality is...
common etiology of BS and it is generally considered that the altered action potential in atrial and ventricular cells should be a cause of AF and ventricular tachyarrhythmia, respectively, in patients with BS. Therefore, we have not considered the contribution of focal electrical abnormality to the AF and ventricular tachyarrhythmias. A recent study demonstrates the focal triggers have an important role in the development of ventricular tachyarrhythmias associated with BS, and the ablation led to a dramatic improvement in the subsequent clinical course. A recent study demonstrates the focal triggers have an important role in the development of ventricular tachyarrhythmias associated with BS, and the ablation led to a dramatic improvement in the subsequent clinical course.3) Bordachar et al. suggested that the preceding atrial premature beats are likely to be associated with the occurrence of AF on ambulatory ECG.
8) Therefore, we hypothesized that the focal triggers may play an important role in AF and was not caused solely by an abnormal myocardial substrate. Anti-arrhythmic agents may promote the occurrence of ventricular tachyarrhythmias, thus the catheter ablation for AF is an effective alternative treatment strategy in patients with BS. ICD therapy and atrial isthmus ablation may be required in this case, however the programmed electrical stimulation protocol failed to induce life threatening ventricular arrhythmia and atrial flutter without disopyramide. These electrophysiological findings suggest that wide QRS tachycardia and atrial flutter could result from the inadequate use of anti-arrhythmia drugs.

References