Idiopathic Ventricular Fibrillation in a Patient with Wolff-Parkinson-White Syndrome

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SUMMARY

Ventricular fibrillation in a patient with ventricular preexcitation is usually due to atrial fibrillation with an extremely rapid ventricular rate from which it degenerates. We present a case with Wolff-Parkinson-White syndrome and coexistent idiopathic ventricular fibrillation. The patient, a 23-year-old male, had had a cardiac arrest four years earlier. In electrophysiological study, the accessory pathway was located in the left posteroseptal region and successfully eliminated with radiofrequency catheter ablation. After the ablation procedure, ventricular fibrillation was induced with programmed ventricular stimulation. A dual chamber implantable cardioverter defibrillator was implanted in the patient.

Key words: Wolff-Parkinson White syndrome, Idiopathic ventricular fibrillation

Patients with Wolff-Parkinson-White (WPW) syndrome usually complain of palpitations, sometimes associated with chest discomfort, dyspnea, light-headedness, or syncope. Sudden cardiac death is rare but it may be the first manifestation of this syndrome. The estimated sudden death incidence in ventricular preexcitation is nearly 0.1% a year and it usually occurs as a result of atrial fibrillation with an extremely rapid ventricular rate due to conduction over the accessory pathway that degenerates into ventricular fibrillation (VF).1,2)

In this report, we present a case with WPW syndrome and coexistent idiopathic VF whose mechanism is not related to preexcitation.

CASE REPORT

A 23-year-old Turkish man presented with palpitation episodes that had continued over the last 5 years. He had a history of cardiac arrest and cardiopulmonary resuscitation that occurred 4 years earlier, but no detailed information or...
electrocardiogram was obtained. He had no family history of sudden cardiac death. He was on sotalol therapy (160 mg/day) at time of presentation. Physical examination and routine laboratory measurements were within normal limits. A surface electrocardiogram was consistent with WPW syndrome due to a left sided accessory pathway. Delta waves were positive in V1 and negative in lead III (Figure 1). Chest x-rays and an echocardiogram showed no pathological findings.

The patient underwent electrophysiological study 1 week after withdrawal of the sotalol. Three sheaths (two 6 Fr and one 7 Fr) were introduced into the right femoral vein after local anesthesia with 1% lidocaine. Two quadripolar catheters were placed in the high right atrium and His bundle region and one steerable quadripolar catheter was placed into the coronary sinus. A spontaneous VF occurred during the positioning of the coronary sinus catheter and was reverted to sinus rhythm after defibrillation with 360 joules. In the electrophysiological study, the antegrade effective refractory period of the accessory pathway was measured as 340 msec. Orthodromic atioventricular reentrant tachycardia was induced with a cycle length (TCL) of 360 msec by programmed atrial stimulation. The

Figure 1. Surface electrogram with preexcitation due to left sided accessory pathway.
findings of mapping in the coronary sinus during orthodromic tachycardia and sinus rhythm were consistent with an accessory pathway located in the left posteroseptal region. A 7 Fr sheath was then introduced into the right femoral artery through which a 4 mm tip ablation catheter (Marinr, Medtronic, Inc., Minneapolis, MN, USA) was placed into the left ventricle by a retrograde transaortic approach. Accessory pathway conduction was terminated by the 13th radio-frequency application to the posteroseptal site of the mitral annulus. The ECG obtained after ablation was free from preexcitation and the corrected QT interval was 400 msec (Figure 2). No tachycardia could be induced with pacing from either the atrium or coronary sinus after ablation. Programmed ventricular stimulation was performed in order to confirm the elimination of retrograde accessory pathway conduction. During this procedure, VF was induced with double extrastimuli (500/250/200 msec) given from the right ventricular apical region (Figure 3). Total procedure time was 130 minutes; total radio-frequency energy duration was 270 seconds and fluoroscopy time was 30 minutes. Coronary angiography showed normal coronary anatomy which excluded coronary artery disease as a possible cause of VF. The patient underwent a second electrophysiological study two weeks later in which VF was reinduced by programmed ventricular stimulation (500/300/280 msec). VF was accepted as idiopathic because it was not

Figure 2. Surface electrogram without preexcitation pattern after successful ablation.
related to accessory pathway conduction and no underlying structural heart disease could be detected. Although neither ST segment elevation nor a right bundle branch block pattern was present in the surface ECG, pharmacologic provocation with propafenone (140 mg IV) was attempted to eliminate the possibility of Brugada syndrome as a cause of VF. No ST segment elevation was observed in the right precordial leads. In light of these findings, a dual chamber implantable cardioverter defibrillator (ICD) was implanted. The patient was symptom-free during an 18-month follow-up.

**DISCUSSION**

In 5 to 10% of patients resuscitated from out-of-hospital cardiac arrest due to VF, no underlying heart disease can be found despite extensive investigation. This arrhythmia is referred to as "idiopathic VF". In 1992, Brugada and Brugada identified a subgroup of patients with idiopathic VF who have a unique ECG pattern characterized by right bundle branch block with ST segment elevation in the right precordial leads. In patients with suspected Brugada Syndrome, unmasking the temporarily normalized ECG by sodium channel blockers is recommended. Brugada's sign is a dynamic pattern that may not always exist in a
surface ECG so pharmacologic provocation must be done for an exact diagnosis. The antiarrhythmics ajmaline, procainamide and flecainide have been used for this purpose.\textsuperscript{6) We performed pharmacological provocation with intravenous propafenone and no classical ECG criteria of the Brugada syndrome were observed. Although the data concerning the use of propafenone as a provocative agent for this syndrome are not sufficient, Matana, \textit{et al}\textsuperscript{7) reported an unmasking effect of propafenone on the concealed form of Brugada syndrome.

It has been shown that the risk of sudden death in people with WPW syndrome is related to the shortest preexcited R-R interval during atrial fibrillation. A shortest R-R interval of less than 250 msec during atrial fibrillation identifies a patient who is at greater risk of sudden death due to a rapid ventricular rate should atrial fibrillation develop.\textsuperscript{2) In our case, the antegrade effective refractory period of the left posteroseptal accessory pathway was 340 msec and atrial fibrillation could not be induced by either programmed atrial stimulation or atrial pacing by which the only induced arrhythmia was orthodromic tachycardia. In consideration of all these findings, we believe that the cardiac arrest that occurred four years ago was not related to a tachyarrhythmia due to accessory pathway conduction.

Implantation of an ICD is advocated by most investigators worldwide as a first-line therapy for patients with idiopathic VF. We implanted an ICD in our patient after successful catheter ablation of the accessory pathway.

Cardiac arrest in a patient with ventricular preexcitation is usually due to atrial fibrillation with a rapid ventricular rate and further degeneration into VF. These patients can be treated successfully by ablation of the accessory pathway. But, as in our case, an independent cause of cardiac arrest which can coexist with preexcitation such as idiopathic VF should always be considered.

REFERENCES