Idiopathic Ventricular Fibrillation Initiated by a Short-coupled Ventricular Premature Beat

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SUMMARY
We report the case of a 38-year-old man who had idiopathic ventricular fibrillation (VF) which initiated by abnormally short-coupled ventricular premature beats. VF was successfully prevented by the combination of pilsicainide, propranolol, and verapamil. In particular, the effects of pilsicainide are assumed to exert an important effect in controlling this arrhythmia. Class Ic drugs may be effective for preventing VF initiated by short-coupled VPBs. (Jpn Heart J 1996; 37: 265–269)

Key words: Sudden death Anti-arrhythmic drugs Pilsicainide

Idiopathic ventricular fibrillation (VF) is a rare but important cause of sudden cardiac death in young patients.1) It is characterized by a normal surface electrocardiogram (ECG) that demonstrates a sinus rhythm and absence of any structural heart disease. Recently, Leenhardt et al2) have reported a subgroup of patients with idiopathic VF that was followed by Torsade de Pointes and triggered by a very short-coupled ventricular premature beat (VPB). No QT prolongation was recognized during sinus rhythm. The authors state that this entity should be identified as a risk factor for sudden cardiac death in young adults. We describe a case with frequent episodes of VF initiated by a short-coupled VPB that were successfully controlled using a combination of anti-arrhythmic medications.

CASE REPORT

A 38-year-old man was admitted to a referring hospital with a chief com-
plaint of syncope of several minutes duration. His past and family histories were unremarkable. Physical examination revealed no abnormalities. The electrolyte concentrations were also within normal limits. ECG demonstrated atrial fibrillation with frequent short-coupled VPBs (Figure 1). Although atrial fibrillation was spontaneously restored to sinus rhythm, abnormally short-coupled VPBs were still observed. The coupling interval of the VPB was 0.24 seconds. No evidence of QT prolongation or abnormal ST-T changes was recognized. After admission, the patient suddenly developed another episode of syncope during a conversation. The ECG at the time of syncope showed polymorphic ventricular tachycardia and VF which was initiated by short-coupled VPBs (Figure 2).

Although DC cardioversion transiently converted the VF to sinus rhythm, it recurred despite intravenous injections of several anti-arrhythmic drugs including lidocaine, mexiletine, propranolol and verapamil. Therefore, frequent DC cardioversion was required to maintain sinus rhythm. The patient lost consciousness during the episodes and was ultimately placed on an artificial respirator. Following administration of pilsicainide (50 mg) via a nasogastric tube, the VF episodes were terminated and prevented despite the appearance of short-coupled
VPBs. The patient was then transferred to our hospital for further evaluation. The combination of pilsicainide (200 mg/day), mexiletine (400 mg/day) and propranolol (40 mg/day) previously prescribed by the referring hospital suppressed the VPBs for several days following transfer.

Thereafter, the patient recovered consciousness and his respiratory condition improved. Mexiletin was ultimately discontinued due to gastrointestinal symptoms. However with recovery and increased activity, the number of short-coupled VPBs gradually increased. Exercise clearly led to an increase in the number of VPBs. We therefore added 200 mg/day of verapamil to the medical regimen. The combination of pilsicainide, propranolol and verapamil (200 mg/day) was well tolerated. This combination was very effective and suppressed the VPBs even during treadmill exercise testing. The coupling interval of VPB was also prolonged to 0.30 seconds. On Holter ECG, only 9 VPBs per day were observed. Cardiac scintigraphy and other examinations could not be performed due to the patient's refusal. The patient has done well during a follow-up period of over 1 year. Isolated VPBs have been observed sporadically during follow-up Holter ECG monitoring.

**DISCUSSION**

Idiopathic VF is known to be a cause of sudden cardiac death in young adults. A subgroup of idiopathic VF, characterized by short-coupled ventricular premature beats, has been reported.\(^1,3-6\) Patients in this group have no structural heart disease and the ECG commonly reveals short-coupled VPBs that may trigger a ventricular tachyarrhythmia. Leenhardt et al\(^3\) have described 14 such cases and compared the clinical characteristics with 9 previously reported cases. Patients with this new syndrome have abnormally short-coupled VPBs on the surface ECG.

Although our patient refused further evaluation, he had no prior history of
heart disease. Furthermore, echocardiography performed on admission revealed no evidence of structural heart disease. This patient had electrocardiographic features similar to those described by Leenhardt et al.\textsuperscript{2) and was therefore considered to possess this syndrome.

Leenhardt et al.\textsuperscript{2) postulated that the mechanism of the short-coupled VPBs involved delayed after depolarization (DAD) that invoked the first beat of Torsade de Pointes. Consistent with these observations, verapamil was the sole medication effective for preventing episodes of VF. This concept is based on the fact that verapamil is effective in suppressing DAD. A low parasympathetic drive has been proposed as another mechanism for the short-coupled VPBs. In the present case, it is difficult to define the precise mechanism because the patient refused detailed evaluation. Pilsicainide was able to prevent recurrent VF during the initial admission. The strong sodium channel blocking action of this class Ic drug and suppression of conduction velocity may inhibit the short-coupled VPBs. In addition, verapamil may reduce VPBs during exercise. Therefore, triggered activity such as DAD may play an important role in the mechanism of the short-coupled VPBs. Although the pathogenesis of VF remains unknown, the clinical efficacy of these medications may be explained by a synergistic effect of the combination of these drugs.

Leenhardt et al.\textsuperscript{2) have reported that verapamil is the only drug effective for preventing VF in this patient population, however, it was not able to prevent sudden death. Therefore, the authors recommend use of an automatic implantable cardioverter defibrillator (ICD) in this group of patients. However, drug therapy for idiopathic VT has been reported to be effective in some patients. Moe\textsuperscript{3) reported the efficacy of quinidine for preventing VF. Ledwich and Fay\textsuperscript{4) have described the use of propranolol for preventing VF despite the appearance of R on T type VPBs. Belhassen et al.\textsuperscript{5) also have reported the efficacy of the combination of amiodarone (400 mg/day) and quinidine (1500 mg/day). Our patient was treated successfully by the combination of pilsicainide, propranolol and verapamil. In particular, pilsicainide is assumed to exert an important effect in controlling this arrhythmia. Thus, class Ic drugs might be effective for preventing VF triggered by short-coupled VPBs. However, the drug does not always prevent sudden-death events. Careful follow-up is necessary and consideration should be given to ICD implantation if the VPBs increase or if recurrent VF is observed.
REFERENCES