Two Cases of Polymorphic Ventricular Tachycardia Induced by the Administration of Verapamil against Paroxysmal Supraventricular Tachycardia

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Abstract

Verapamil is widely used for the termination of paroxysmal supraventricular tachycardia (PSVT) with little proarrhythmic effect. We describe two cases of PSVT that changed to non-sustained polymorphic ventricular tachycardia after administration of verapamil. Electrophysiological study revealed atrioventricular nodal reentrant tachycardia in the first case, and atrioventricular reentrant tachycardia due to a concealed left lateral accessory pathway in the second case. Catecholamine-induced automaticity was one of the possible mechanisms of VT in the first case, but the mechanism is unknown in the second case. (Internal Medicine 41: 445-448, 2002)

Key words: calcium antagonistic drug, electrophysiological study, proarrhythmic effect

Introduction

Paroxysmal supraventricular tachycardia (PSVT) is a common tachyarrhythmia. After simple vagal maneuvers have been tried and adenosine given, intravenous verapamil is the next treatment of choice for terminating AV nodal reentry (AVNRT), or orthodromic AV reciprocating tachycardia associated with the Wolff-Parkinson-White syndrome (1). Verapamil is considered as a safe regimen for these arrhythmias.

Verapamil has adverse effects such as hypotension, bradycardia, atrioventricular block, and asystole. We report here on two cases of PSVT that changed to polymorphic ventricular tachycardia (VT) after the administration of verapamil. A few reports (2–4) referred to this proarrhythmic effect previously.

Case Report

Case 1

A 53-year-old woman complained of palpitation and chest discomfort. Physical findings were normal except for the blood pressure that was 150/90 mmHg. The ECG during sinus rhythm showed no abnormalities but complete right bundle branch block and the ECG during palpitation showed supraventricular tachycardia (Fig. 1). After the intravenous administration of verapamil 3 mg for 5 minutes, PSVT directly changed to polymorphic ventricular tachycardia and spontaneously terminated (Fig. 2 upper panel). The QT interval of ECG did not change before (3 10 ms) or after administration of verapamil (3 10 ms). Blood pressure increased from 120/66 mmHg (during PSVT) to 140/74 mmHg after administration of verapamil.

In an electrophysiologic study, a common type of atrioventricular nodal reentrant tachycardia (CL=320 ms) was inducible with an atrial extrastimulation in the presence of isoproterenol. During AVNRT, spontaneous triplet of ventricular premature contractions appeared, but AVNRT did not terminate (Fig. 3). After catheter ablation of the slow pathway, AVNRT could not be induced with programmed stimuli. VT could not be induced, but the number of spontaneous ventricular premature contractions increased with infusion of isoproterenol.

Case 2

A 60-year-old man complained of palpitation. Physical findings were all within normal limits. The ECG during sinus rhythm showed no abnormalities and the ECG during palpitation showed narrow QRS tachycardia (Fig. 4). After the intravenous administration of verapamil 1.5 mg for 4 minutes, PSVT changed to polymorphic ventricular tachycardia that spontaneously terminated (Fig. 2 lower panel). Blood pressure did not decrease after administration of drug. The QT interval of ECG did not change before (260 ms) or after administration of verapamil (260 ms). Blood pressure increased from 110/60 mmHg (during PSVT) to 140/72 mmHg after administration...
of verapamil.

In an electrophysiologic study, an orthodromic atrioventricular reentrant tachycardia (CL 340 ms) was inducible with an atrial extrastimulation. Intracardiac ECG with the shortest VA interval during right ventricular pacing was recorded in the left lateral portion of the mitral annulus. After the catheter ablation of a concealed left postero-lateral accessory pathway, PSVT could not be induced. After the ablation, extra and burst stimuli were given to the right atrium and ventricle with isoproterenol infusion or after administration of verapamil (3 mg), but clinical polymorphic ventricular tachycardia could not be induced.

**Discussion**

Paroxysmal supraventricular tachycardia (PSVT) is a common tachyarrhythmia and there are several methods to terminate this arrhythmia. According to Miller and Zipes (1), intravenous verapamil is recommended as treatment to terminate sustained sinus nodal reentry, AV nodal reentry, or orthodromic AV reciprocating tachycardia associated with Wolff-Parkinson-White syndrome. Verapamil terminates 60% to more than 90% of episodes of PSVT within several minutes. It is also considered to have little proarrhythmic effect, and there have been few reports that PSVT changed to polymorphic VT after the administration of verapamil which was used for termination of PSVT (2-4).

In the first case, the interval from R wave of PSVT to R wave of first non-SVT beat (350 msec) was shorter than the cycle length of supraventricular tachycardia (SVT) (410 msec). First non-SVT beat was wide QRS and considered as premature ventricular contraction (PVC). After PVC was triggered, the rhythm in this case changed to polymorphic VT. Polymorphic VT such as Torsade de pointes (5) is often associated with long QT, but in our case, QT was not prolonged. After catheter

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**Figure 1.** Case 1 ECG. ECG in the left column was recorded at rest and showed normal sinus rhythm. ECG in the right column was recorded during palpitation attack and showed PSVT.

**Figure 2.** PSVT changed to polymorphic VT. Monitor ECG showed that PSVT changed to polymorphic VT. Upper panel: recording after administration of verapamil 3 mg in case 1. Lower panel: After administration of verapamil 1.5 mg, PSVT terminated, but 15 consecutive beats of polymorphic VT appeared in case 2. Monitor showed a part of the VT.
Verapamil Induced Polymorphic VT

Figure 3. Intracardiac ECG. Ventricular triplet occurred spontaneously during PSVT. Though the triplet showed decremental ventriculoatrial conduction through atrioventricular node, AVNRT did not terminate.

ablation, the number of spontaneous PVCs increased with infusion of isoproterenol, but VT could not be induced by programmed stimuli with even additional administration of verapamil. Spontaneous PVCs were considered as catecholamine sensitive, but the cause of polymorphic VT was unknown.

In the second case, the cycle length of SVT changed alternately in four beats preceding to VT and spontaneously SVT terminated with cessation of antegrade atrioventricular conduction in AV node. After termination of PSVT, 15 consecutive beats of polymorphic VT appeared. After catheter ablation, we could not induce VT with programmed stimulation even with infusion of isoproterenol or with administration of verapamil.

Unbalanced stimulation of the autonomic nerve might be a trigger of VT. There are some reports suggesting that polymorphic VT after termination of PSVT is related to vagal stimulation. Hellerstein et al (6) reported on the conversion of SVT by carotid-sinus massage, PVCs or short runs of VT followed the cessation of the SVT in 6 of 33 episodes. In another paper, similar ventricular arrhythmias followed by the administration of methacholine were reported (7). These reports suggested that ventricular arrhythmias were due to the vagal stimulation caused by the carotid-sinus manipulation or the drug.

However, in the present cases, the vagal stimulation was not likely the cause of VT, because our patients did not complain of a vagotonic reaction after termination of PSVT, such as bradycardia, hypotension, nausea and so on.

Conversely, VT in our cases was related to abnormal automaticity. VT in the first case was exaggerated by stimulation of the sympathetic nerve. One of the possible mechanism of polymorphic VT in our cases was considered to be abnormal automaticity, for some reason. First, it had irregular R-R intervals, and the number of premature ventricular beats increased with isoproterenol in the first case. Second, it could not be induced by the extra-stimulus method. However, triggered activity such as delayed after-depolarization was unlikely, because VT occurred in the presence of verapamil.

Previous reports have shown that ventricular ectopic activities are not observed after injection of verapamil in patients with sinus-rythm (8) and atrial fibrillation or flutter (3). Therefore, verapamil was not considered to induce VT by itself. However, verapamil has adverse effects such as negative chro-
notropic and negative inotropic effects (1). Calcium antagonistic drugs generally lower blood pressure and stimulate the sympathetic nerve as a reflex (9). If patients have the substrate of VT, PVC might trigger the VT under the stimulation of the sympathetic nerve.

The other possible mechanism of VT was hypoperfusion of the myocardium. It is caused by hypotension due to PSVT itself and additional infusion of verapamil. Ischemia of the myocardium might cause arrhythmogenesis, such as reentry, triggered activity and abnormal automaticity.

We describe two cases of PSVT that changed to polymorphic VT after the administration of verapamil. This proarrhythmic effect was considered to be rare, but it should be taken into consideration when intravenous verapamil is used.

References