


Monomorphic ventricular tachycardia induced by tilt table testing in a patient with syncope and normal heart

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1 | CASE

A 62-year-old woman presented with recurrent syncopal episodes of head injuries. Although she had been under in-hospital management, including long-term electroencephalogram monitoring and several cardiac evaluations, the origin of her syncope remained unknown. She had a normal heart without a significant electrocardiogram (ECG) abnormality (Figure 1). Her treadmill exercise test was negative for ischemic heart disease and arrhythmias. Her syncopal episodes often occurred while she was in the standing position or if she was seated for a long period. The first syncopal episode occurred one day during the winter season. She waited for her family in a slightly warm place with her coat on. She suddenly lost consciousness without any pre-symptoms. At another time, she had a syncopal episode while she was a passenger in a car. These episodes were likely reflex syncope triggered by long durations of standing or sitting. The first diagnosis we made was vasovagal reflex syncope without any prodromes. She had several secondary injuries due to syncope; thus, we decided to perform a tilt table test (TTT). We performed TTT to induce reflex syncope. We selected the "Italian protocol" as TTT. After 3 minutes of tilting at 60°, premature ventricular contraction (PVC) appeared frequently, and monomorphic ventricular tachycardia (VT) occurred with similar symptoms as usual syncopal episodes (Figure 2). The patient would fall unconscious during tilting. The VT was stopped spontaneously during the tilt

table returned to spine position. Her consciousness was completely awake and alert after that. In addition, she did not have palpitations before or during VT. We performed TTT again, and a similar VT was also reproduced with similar symptoms. The VT had an ECG appearance with a left bundle branch block appearance and was positive in the inferior leads (Figure 3). After TTT, VT occurred when the patient was standing despite her presenting with a normal coronary angiogram. Oral intake of bisoprolol fumarate 2.5 mg/d reduced her VT. Her VT was induced only by the autonomous reflex of the sympathetic nervous system while she was in the standing position, but was excised by stress. Finally, we chose radiofrequency catheter ablation (RFCA) for treatment. The procedure was a success to terminate the VT. However, another VT occurred in very early period after the RFCA. We selected medication as an additional treatment for the VT. We performed TTT under oral intake bisoprolol and sotalol hydrochloride. The TTT did not induce VTs and syncope. After more than 3 years of follow-up, she did not have any syncopal episode or palpitation with the drugs.

2 | DISCUSSION

We report TTT-induced VT in a healthy woman with a normal heart. TTT-induced arrhythmia is rare: the most common arrhythmia is atrial fibrillation, with very few reports of VT. In several reports,

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Twelve Lead Electrocardiogram at the first visit.

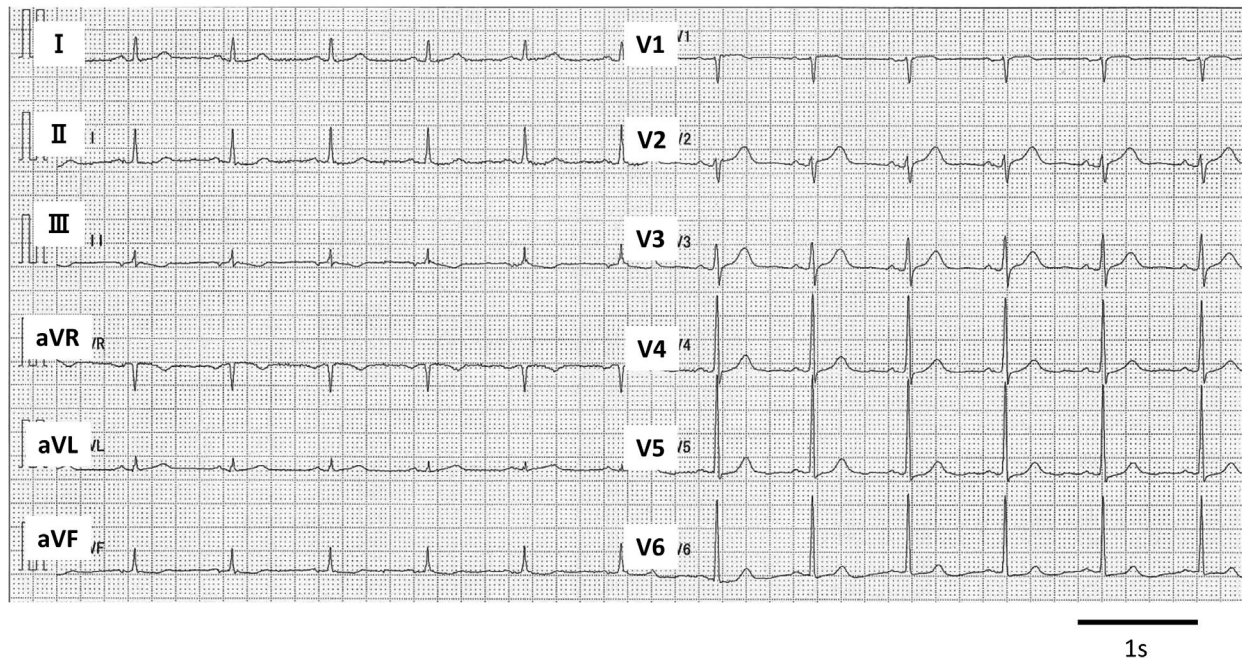


FIGURE 1 Electrocardiogram (ECG) at the first visit. The ECG showed a sinus rhythm and heart rate of 73 beats per minute. Slight ST-segment depression observed in inferior leads. Her coronary angiogram presented normal

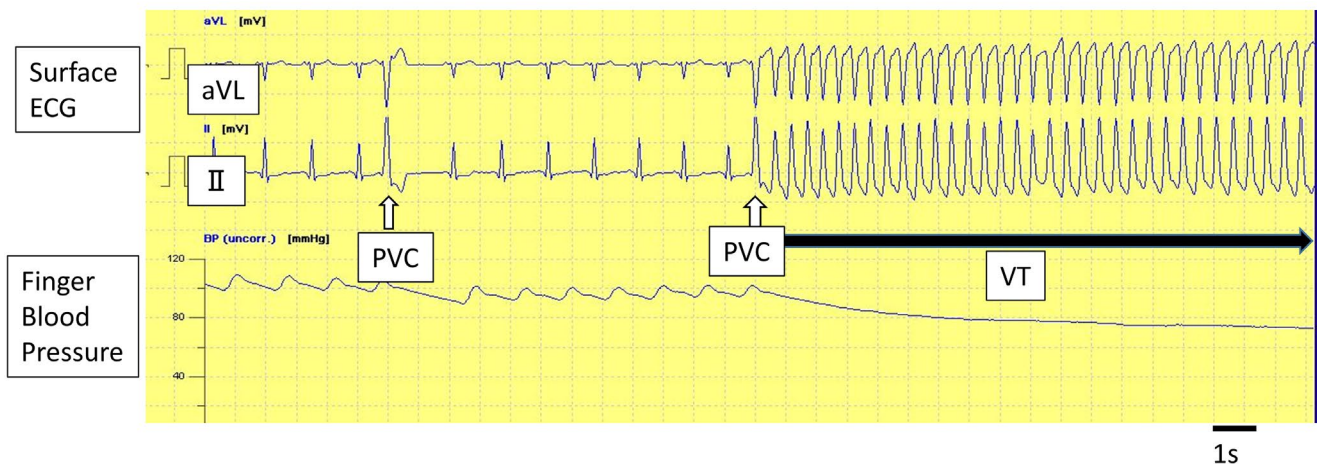


FIGURE 2 Premature ventricular contraction appeared frequently (White arrows) and monomorphic ventricular tachycardia with about 220 beat / minute occurring. The symptoms was similar usual syncopal episodes (Black arrows). Her finger blood pressure was undetected due to lower blood pressure during the ventricular tachycardia

VT was induced by TTT in patients with an abnormal heart and/or abnormal ECG. In other reports, VT was induced during TTT with an isoproterenol (ISO) infusion. Our patient had no abnormalities in the ECG, ultrasonic echocardiography, or coronary arteries. We also performed TTT without any drugs, including ISO. The TTT recommendation level in a recent guideline was changed to “low.” However, TTT is useful for selected patients, as in our case. TTT is one of the options used to verify diagnosed reflex syncope or

another arrhythmic syncope. In this case, the syncopal episodes were prolonged by standing, sitting, and hot temperatures.

Our patient did not have any heart disease. We suspected reflex syncope even though there were no prodromal symptoms or injuries. Thus, we selected TTT before implanting an insertable cardiac monitor or other long-lasting ECG monitoring devices. In another case report written in 1995, orthostatic VT was induced by an infusion of ISO. In our case, VT was induced by reflex sympathetic activation triggered by the

Twelve Lead Electrocardiogram at the Induction of Ventricular Tachycardia

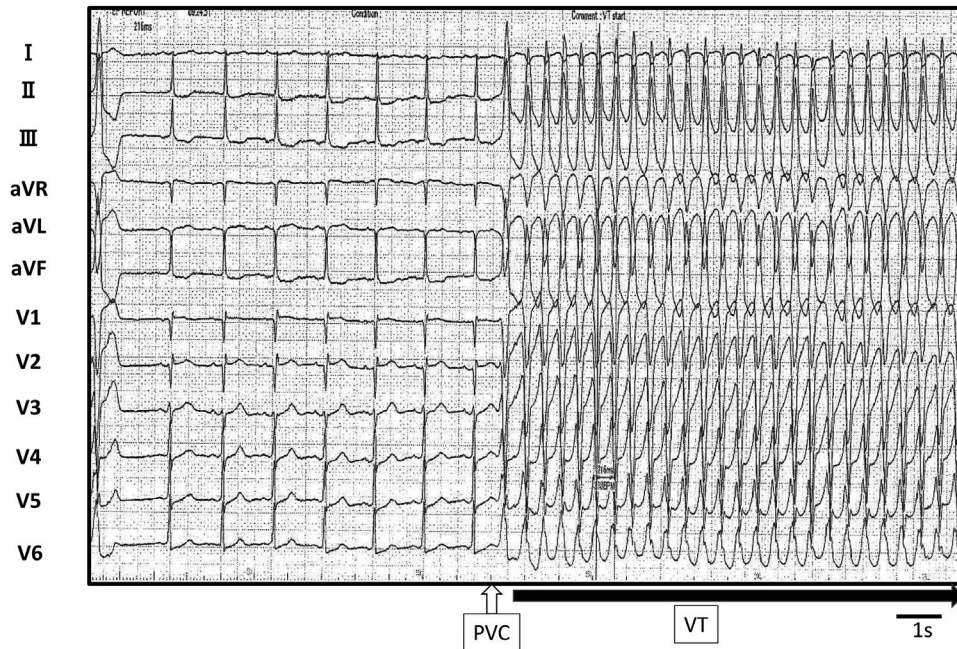


FIGURE 3 12-lead electrocardiogram appearance of ventricular tachycardia, which showed a left bundle branch block appearance and which was positive in the inferior leads

orthostatic position, but not exercise stress. The difference in autonomic tone may be due to an increase in exercise stress and orthostatic stress.

3 | CONCLUSION

We experienced a case in which monomorphic VT was induced by TTT examination in a naturally healthy woman who complained of syncope. VT was induced only by orthostatic stress.

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CONFLICT OF INTEREST

Authors declare no conflicts of interest for this article.

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