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Letter to the Editor

Occurrence of *de novo* sustained monomorphic ventricular tachycardia induced after percutaneous transluminal alcohol septal myocardial ablation for hypertrophic obstructive cardiomyopathy

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Abstract

We report here a 75-year-old male with hypertrophic obstructive cardiomyopathy of *de novo* sustained monomorphic ventricular tachycardia (VT) after successful percutaneous transluminal alcohol septal myocardial ablation (PTSMA). In this case history, the necrotic induced by the PTSMA procedure might represent a region of slow conduction that is a circuit of re-entry and therefore stimulation might be spread around. Therefore, the basis of the sustained monomorphic VT was thought to be the presence of a focal necrotic area, itself a complication arising from the PTSMA procedures. In conclusion, the PTSMA procedure may have caused a *de novo* episode of ventricular arrhythmia.

Keywords: *De novo* sustained monomorphic ventricular tachycardia; Percutaneous transluminal alcohol septal myocardial ablation; Hypertrophic obstructive cardiomyopathy

Percutaneous transluminal alcohol septal myocardial ablation (PTSMA) is appropriate for patients who are refractory to medical treatment for hypertrophic obstructive cardiomyopathy (HOCM) [1–5]. However, abnormalities of conduction system have been reported as common complications of PTSMA [6]. We report here a case of *de novo* sustained monomorphic ventricular tachycardia (VT) after successful PTSMA. The induction of the sustained VT may be due to a re-entry circuit occurring in the necrotic sites caused by PTSMA procedure.

1. Case Report

A 75-year-old male experienced dyspnea with chest pain on effort and had cold sweats at rest 3 months ago. He also experienced blackouts accompanied with chest pain when going up stairs. Since then, he had often experienced episodes of similar symptoms with loss of consciousness and thus he presented at our hospital. Results of a transthoracic echocardiography were consistent with the features of HOCM, such as thickening of interventricular septum (IVS) 15mm, the presence of systolic anterior motion of the mitral valve, severe mitral regurgitation, and a 30 mm Hg pressure gradient between the outflow tract of the LV and aorta at rest with good LV systolic function. A dobutamine-loaded echocardiography showed that the pressure gradient between the outflow tract of the LV and aorta increased up to 109 mm Hg with a decrease of systemic systolic blood pressure and clinical symptoms of chest pain. After the administration of atenolol and cibenzoline, the pressure gradient decreased to 7 mm Hg and his symptoms disappeared. Since, syncope occurred repeatedly, even with medication such as atenolol and cibenzoline, he underwent

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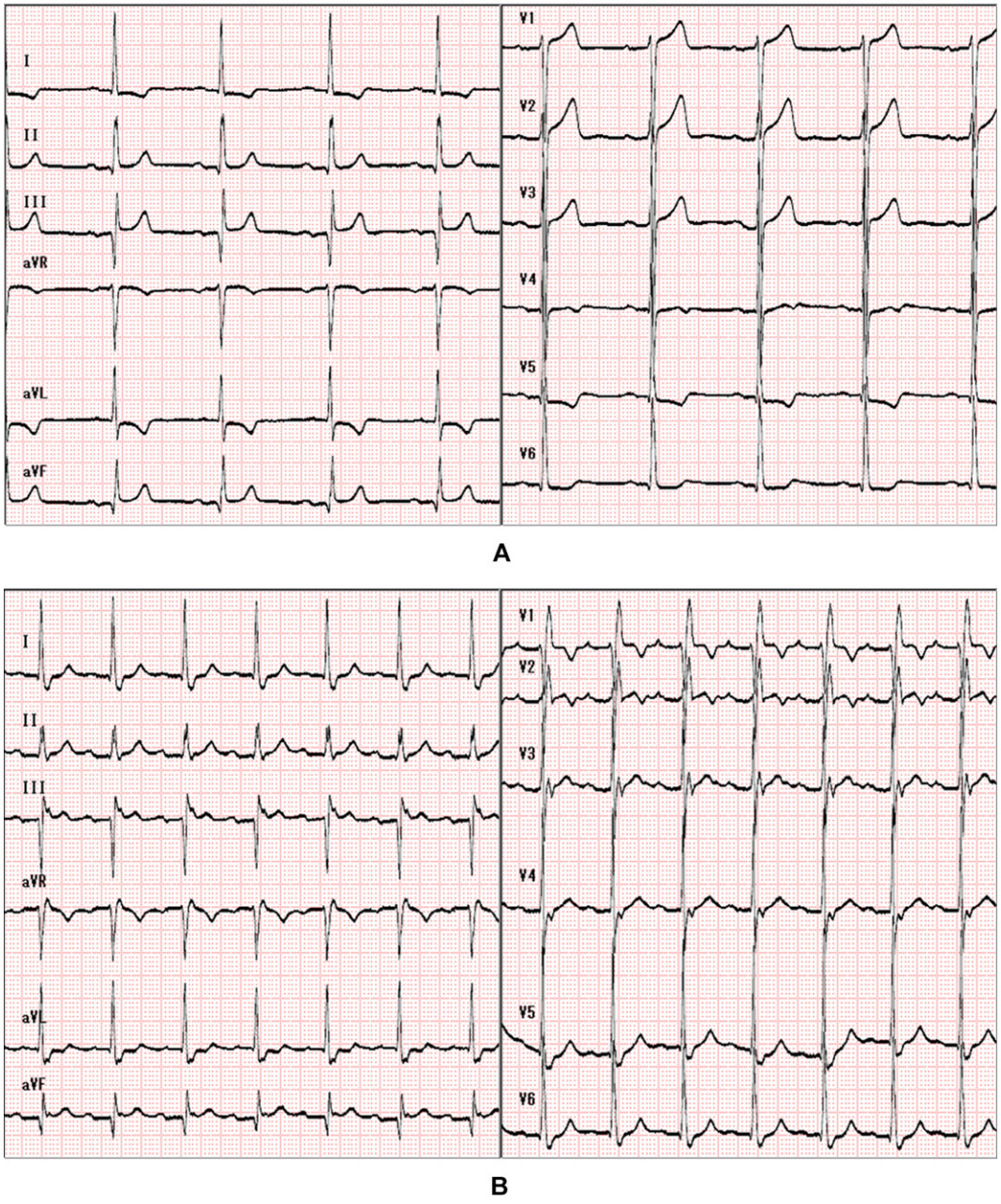

Fig. 1. Electrocardiograms before (A) and after (B) percutaneous transluminal alcohol septal myocardial ablation (PTSMA). A. Before PTSMA, one degree atrioventricular block and strain pattern ST decline were observed in the aVL, V5 and, V6 leads. B. After PTSMA, complete right bundle branch block appeared.

PTSMA. Before PTSMA, an electrocardiogram (ECG) revealed normal sinus rhythm, with normal axis deviation and LV hypertrophy pattern (Fig. 1A). The Holter ECG revealed no sustained VT and a coronary angiography revealed normal coronary arteries. The guide wire was first introduced to the 1st septal branch. After balloon inflation and injection of contrast, 3.0 ml of ethanol was injected slowly into the 1st septal branch artery over 3 min (Fig. 2). Echocardiography showed limited brightness in the septal region and complete right bundle branch block (CRBBB) had appeared on the ECG (Fig. 1B).

After PTSMA, he had no trouble and was discharged at 21 days after PTSMA. At 389 days after discharge, he suddenly lost consciousness whilst traveling in the train. Transthoracic echocardiography and an ECG revealed no remarkable changes from ones before discharge. Single-averaged ECG revealed positive findings of a late potential of the LV, which was not observed before PTSMA (Fig. 3). Since the loss of consciousness may be caused by ventricular arrhythmia, an electrophysiological study was performed. Fractionated potential was present on the Bundle of His on his ECG and ventricular programmed stimulations gave rise