

## Intramural Reentrant Ventricular Tachycardia in a Patient with Severe Hypertensive Left Ventricular Hypertrophy

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We describe the case of a patient with severe hypertensive left ventricular hypertrophy and sustained hemodynamically unstable ventricular tachycardia (VT). Entrainment was demonstrated in the electrophysiological study. Activation mapping and pacemapping identified the location of the intramural reentrant VT with the exit site close to the epicardium. However, VT persisted after ablation at the epicardial exit site. Successful ablation was performed endocardially at the corresponding position. (**Korean Circ J 2015;45(6):526-530**)

**KEY WORDS:** Catheter ablation; Hypertension; Hypertrophy, left ventricular; Tachycardia, ventricular.

### Introduction

Hypertension leads to a myriad of cardiovascular diseases, including left ventricular hypertrophy (LVH), congestive heart failure, and ischemic heart disease.<sup>1)</sup> The long-term increased afterload imposed on the left ventricle (LV) during hypertension contributes to manifestation of concentric hypertrophy, which could be an ominous sign, and an independent risk factor for sudden cardiac death (SCD),<sup>2)</sup> ventricular arrhythmias,<sup>3)</sup> myocardial ischemia,<sup>4)</sup> coronary artery disease,<sup>5)</sup> and heart failure.<sup>6)</sup> Although the presence of ventricular dysrhythmias in a hypertensive patient has a clinical prognostic value,<sup>7)</sup> ventricular electrical storm attributable to hypertensive LVH has not been reported previously. In addition, the electrophysiological properties and substrate characteristics of

hypertensive LVH remain unclear. Furthermore, a significantly thickened hypertrophic myocardium as a result of hypertensive remodeling could cause difficulty in radiofrequency energy penetration and prevent effective ablation from eliminating the intramural circuits. Therefore, the efficacy of radiofrequency catheter ablation of ventricular tachyarrhythmias in patients with hypertensive LVH remains unknown. In this report, we performed successful ablation of an intramural reentrant ventricular tachycardia (VT) circuit and abolished the electrical storm in a patient with severe hypertensive LVH.

### Case

The patient was a 60-year-old gentleman with a known history of adult dominant polycystic kidney disease, and it was associated with hypertension and chronic kidney disease. He had suffered from intracranial hemorrhage as a result of a berry aneurysm three years ago. He complained of palpitation, chest discomfort, and several episodes of near-syncope, and these symptoms had worsened in the last one month. In the emergency room, he developed sustained hemodynamically unstable VT, which showed a right bundle branch block configuration, positive precordial concordance, and inferior axis (Fig. 1A). The presence of a pseudo-delta wave (138 msec), high maximum deflection index (0.62), q wave in lead I, and alternans in tachycardia cycle length (442 msec and 589 msec) with variation of QRS morphologies suggested the presence of different epicardial exit sites near the basal lateral region of the perivalvular mitral valve.<sup>8)</sup> The electrical storm of sustained VT was refractory to

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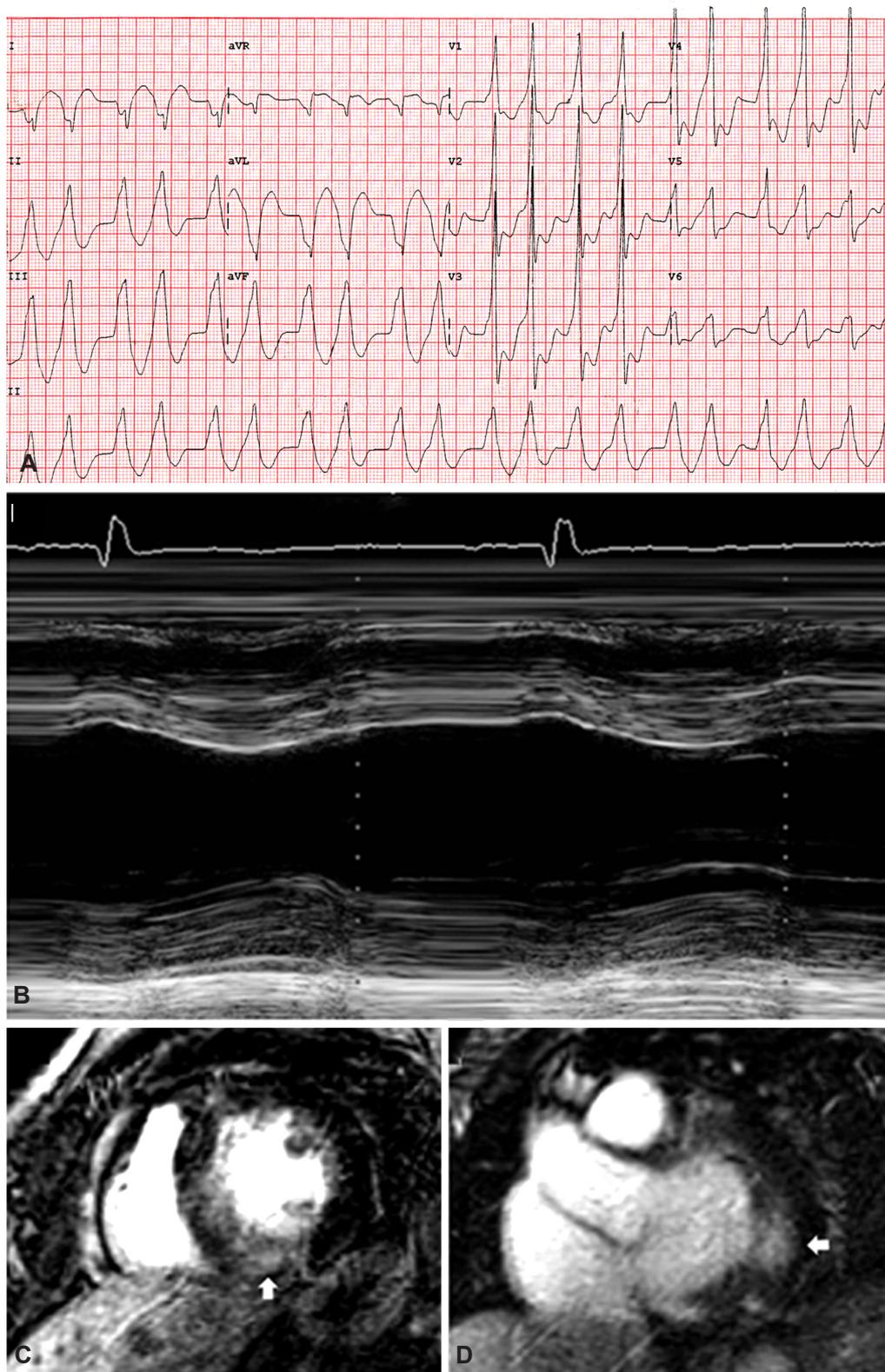
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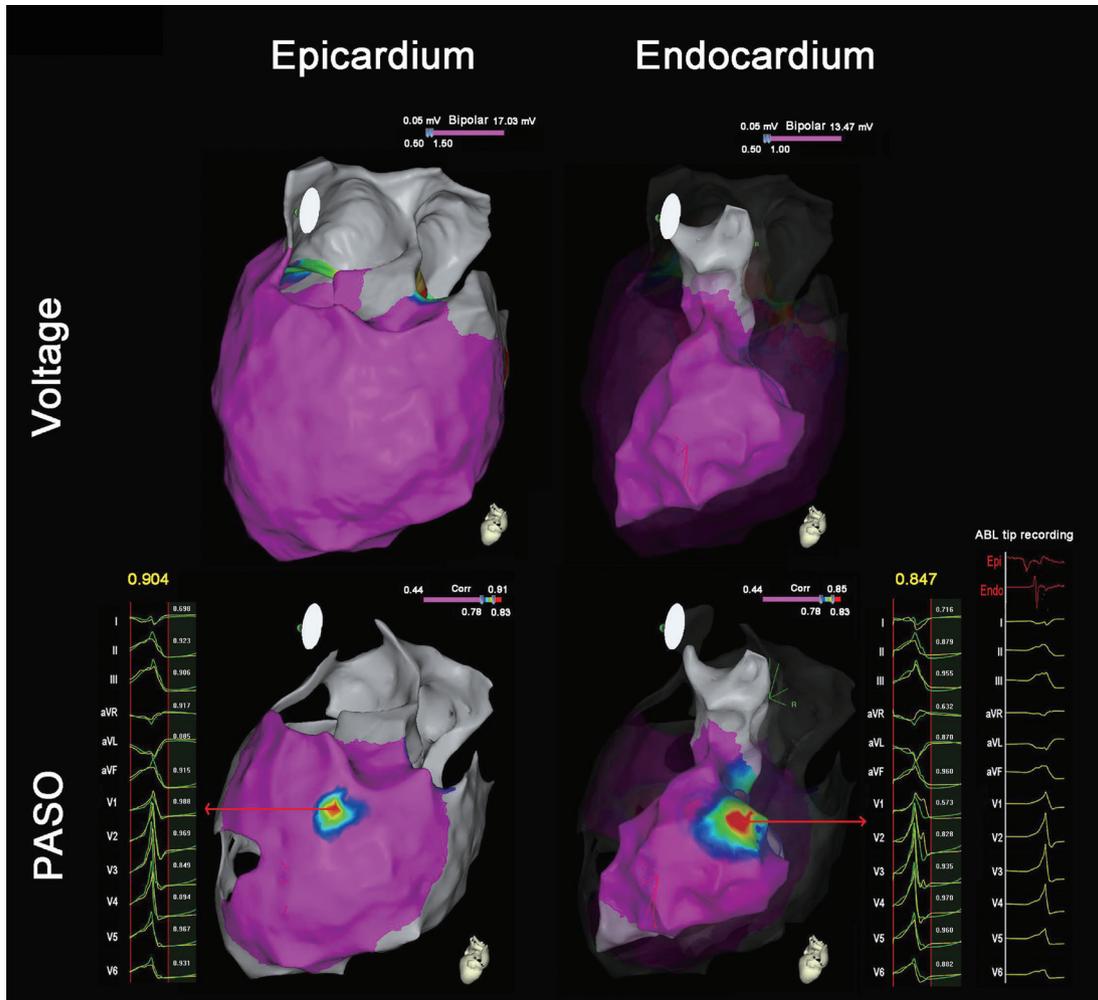
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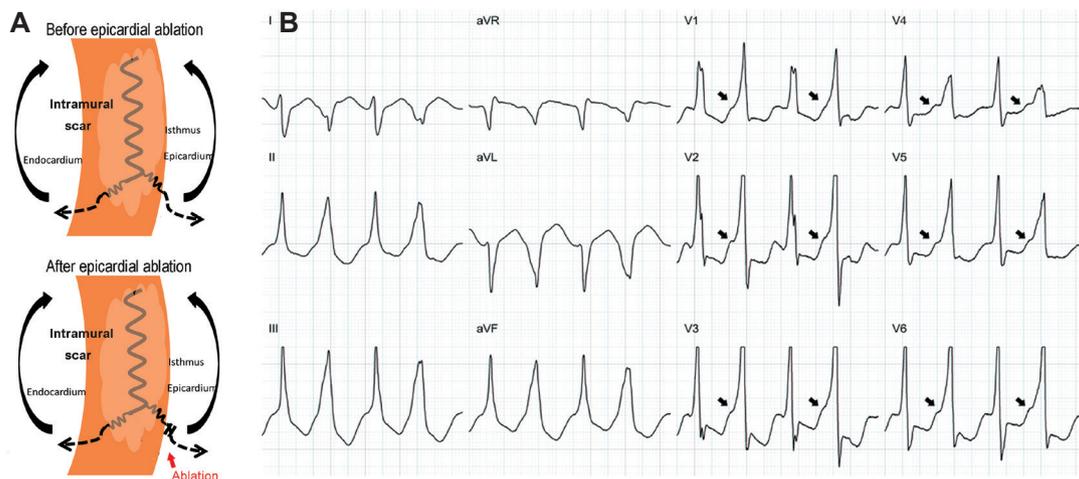
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**Fig. 1.** Electrocardiographic morphologies of ventricular arrhythmia and echocardiography in a patient with severe concentric left ventricular hypertrophy (LVH). (A) Ventricular tachycardia (VT) ECG demonstrates a right bundle branch block, positive precordial concordance, and inferior axis with the presence of a pseudo-delta wave, high maximum deflection index, q wave in lead I, suggesting the tachycardia originating from the basal lateral mitral area epicardially. Alternation of VT cycle length with variation of QRS morphologies is noted. (B) Echocardiography reveals severe concentric LVH (intraventricular septum at diastole/ posterior wall at diastole: 17/18 mm); cardiac magnetic resonance imaging shows subendocardial late gadolinium enhancement in the posterior septum, posteroinferior wall (C, arrow) and basal lateral wall (D, arrow) of the LV. LV: left ventricle, ECG: electrocardiography.



**Fig. 2.** The voltage mapping (upper panel) and (Pace Mapping Software, PASO; Biosense Webster, Inc., Diamond Bar, CA, USA) mapping (lower panel) demonstrated the best pacemapping site at the epicardial basal lateral mitral annulus with globally normal voltage. The local electrograms of the irrigated tip catheter (Thermocool, Biosense Webster Inc., Diamond Bar, CA, USA) at the epicardium but not at the endocardium preceded the ventricular ectopy by 33 msec.



**Fig. 3.** Changing morphology during ventricular tachycardia. (A) Illustration demonstrates change in the ventricular tachycardia (VT) exit site from the epicardium to the endocardium and epicardial alternans after epicardial ablation. (B) Epicardial ablation results in variation of VT ECG morphology and cycle length. Presence of pseudo-delta waves (arrow) in the even beats but not in the odd beats suggest the VT exit to the epicardium and endocardium alternatively. ECG: electrocardiography.

treatment with antiarrhythmic drugs and general anesthesia. Therefore, the patient underwent an electrophysiological study (EPS).

Transthoracic echocardiography revealed severe concentric LVH (Fig. 1B; interventricular septum/posterior wall thickness: 17/18 mm) without noticeable wall motion abnormalities. The LV ejection fraction was 57%. Cardiac magnetic resonance imaging (CMR) demonstrated subendocardial late gadolinium enhancement in the basolateral wall, posterior septum, and posteroinferior wall of the LV (Fig. 1C and D).

### Electrophysiologic study and radiofrequency catheter ablation

After obtaining informed consent, the EPS was performed in a fasting state under moderate sedation. A quadripolar catheter (St Jude Inc., St Paul, MN, USA) was positioned in the right ventricle. Hemodynamically unstable VT developed either spontaneously or was induced by programmed ventricular stimulation from the right ventricular apex with 3 extrastimuli. Long return cycle after manifest entrainment from the RVA suggested the possible mechanism of reentry tachycardia (Supplementary Figs. 1 and 2 in the online-only Data Supplement). VT was terminated via DC cardioversion after unsuccessful overdrive pacing. Epicardial access was obtained via subxiphoid puncture. Three-dimensional LV endocardial and epicardial anatomies were created using Carto 3 (Biosense Webster, Inc., Diamond Bar, CA, USA) with a 3.5 mm irrigated tip catheter (Thermocool, Biosense Webster, Inc., Diamond Bar, CA, USA). LV endocardial bipolar (>1.5 mV) and unipolar voltage mapping (>8.27 mV),<sup>9</sup> and epicardial bipolar voltage mapping (>1.0 mV) revealed no remarkable scar {mapping density was 1.33 points/cm<sup>2</sup> (338 points) and 1.47 points/cm<sup>2</sup> (809 points) in LV endo- and epicardium, respectively} (Fig. 2). There were no identifiable late or fractionated electrograms endocardially and epicardially. Given the hemodynamically unstable nature, we targeted the potential exit site at the endocardial and epicardial LV by pacemapping {pacemapping software (PASO) of Carto 3 (Biosense Webster Inc., Diamond Bar, CA, USA)} and activation map of repetitive monomorphic premature ventricular complexes/nonsustained VT. The optimal pace mapping site,  $r=0.904$ , of the epicardium was identified at the basal lateral mitral annulus, which approximated the earliest activation site of the VT. The potential of the basal lateral annulus preceded the onset of QRS by 33 msec. On the other hand, the best pace mapping site ( $r=0.847$ , lower than epicardium) of the endocardium was identified at the corresponding sites of the highest correlation epicardium, whereas the activation maps showed that the local activation time was same as the onset of QRS. However, after application of ablation to the earliest epicardial

site (25–30 Watts; targeting for an impedance drop of 10 Ohms), VT was still inducible with an alternation of 2 QRS morphologies (Fig. 3). The absence of pseudo-delta wave of the odd beats suggested endocardial exit of tachycardia at the corresponding endocardial basal lateral mitral annulus, where matching pacemapping morphology was identified. Initial application of radiofrequency energy up to 30 Watts to the identified endocardial exit sites had no effect on the ectopic beats until the energy was increased up to 50 Watts. Post ablation, pharmacological provocation was performed under intravenous infusion of isoprenaline 4 µg/min to achieve at least 20% heart rate increment. No further ventricular arrhythmia was seen despite repeated induction. Patient underwent implantable cardioverter-defibrillator implantation immediately afterwards, and remained clinically uneventful at 2 months of follow-up.

### Discussion

This case demonstrated successful catheter ablation of ventricular tachyarrhythmia in a patient with hypertensive LVH presenting with an electrical storm. In this case, the absence of identifiable myocardial scars in endocardial and epicardial voltage mapping and requirement of endocardial and epicardial ablation suggested that the presence of an intramural scar was responsible for the maintenance of VT. Besides, manifest entrainment during ventricular pacing suggests the possible mechanism of scar-related reentry. In spite of the earlier activation on local epicardial electrograms preceding the onset of ventricular arrhythmias, the development of VT alternans after modification of the epicardial exit also explained the fact about intramural arrhythmogenic substrates. Catheter ablation is an effective alternative strategy in management of drug-refractory ventricular arrhythmias in patients with structural heart disease. However, the thickened myocardium in LVH could reduce energy penetration during ablation, and combined epicardial and endocardial ablation is frequently required in order to achieve a successful outcome.

LVH, which is frequently identified in hypertensive patients,<sup>10</sup> is associated with development of ventricular arrhythmias,<sup>3)11–13</sup> which can increase the risk of SCD.<sup>7</sup> Our case not only echoed the above statement, but it also demonstrated a rare clinical manifestation of electrical VT storm in patients with hypertension. Dukkupati et al.<sup>14</sup> demonstrated that monomorphic VTs in patients with hypertrophic cardiomyopathy (HCM) frequently originated from the LV apex, with the presence of scar either endocardially or epicardially. On the contrary, in our case, VT originated from the basal lateral mitral annulus without detection of significant scars either endocardially and epicardially. This could reflect the distinct features and substrate characteristics of hypertensive LVH. However, further investigations

are warranted to determine whether the ventricular arrhythmias and electrophysiological properties in all patients with hypertensive LVH display a uniform pattern.

Furthermore, Hutchinson et al.<sup>9)</sup> demonstrated that in patients with non-ischemic cardiomyopathy, the endocardial unipolar voltage mapping in the absence of endocardial bipolar abnormalities could be a useful tool in identifying the presence of arrhythmogenic substrates transmurally or epicardially. In discordance with their previous finding, there was no remarkably abnormal unipolar voltage mapping endocardially within the intramural scar identified by CMR. It is therefore postulated that the increased LV wall thickness could decrease the predictive value for detecting arrhythmogenic substrates transmurally or epicardially in hypertensive patients. However, a further study is needed to elucidate the appropriate strategies to define a transmural scar with regard to differences in the extent of HCM.

In conclusion, our case demonstrated successful ablation of ventricular tachyarrhythmias in a patient with significant hypertensive LVH, and highlighted the feasibility of applying a simultaneous epicardial and endocardial.

### Supplementary Materials

The online-only Data Supplement is available with this article at <http://dx.doi.org/10.4070/kcj.2015.45.6.526>.

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