Atrio-Hisian block during catheter ablation targeting premature ventricular complexes originating from the left ventricle

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Introduction

Catheter ablation can be associated with inadvertent injury of the atrioventricular (AV) node or His bundle when targeting AV nodal reentrant tachycardia (AVNRT), a septal accessory pathway, para-Hisian atrial tachycardia, and para-Hisian premature ventricular complexes. We report a case of a male patient with symptomatic premature ventricular contractions (PVCs) originating from the para-His bundle in whom radiofrequency (RF) ablation to the inferoseptum of the left ventricle (LV) achieved elimination of the clinical PVCs but simultaneously resulted in transient atrio-Hisian (AH) block.

Case report

An 82-year-old male patient with a history of myocardial infarction was referred to our institution for catheter ablation of symptomatic, drug-resistant, and frequent PVCs and symptomatic and frequent paroxysms of atrial fibrillation. On 12-lead electrocardiogram, PVCs showed left bundle branch block morphology and R/S transition in lead V_2. Leads I and aVL had positive forces (R), lead II had a positive force (Rs), and leads III and aVF had negative forces (rS). Echocardiography revealed a normal ejection fraction of 72% without asynergy or hypertrophy. Holter monitoring confirmed the presence of a 23% monomorphic PVC burden.

KEY TEACHING POINTS

- Catheter ablation can be associated with inadvertent injury of the atrioventricular (AV) node or His bundle when targeting para-Hisian premature ventricular complexes (PVCs).
- It has been reported that a slow pathway in AV nodal reentrant tachycardia can be eliminated by left atrial or left ventricle (LV) ablation, and PVCs originating from the basal LV can be eliminated by right atrial ablation around the slow pathway region.
- This report showed that ablation of the basal recess of the LV septum can injure the fast pathway or compact AV node, leading to AV block.

After written informed consent was obtained, an electrophysiological study was performed under CARTO guidance (Biosense Webster, Inc, Diamond Bar, CA) 5 days after the cessation of mexiletine. The bandpass frequencies of the filtered electrograms ranged from 30 to 500 Hz (bipolar signals) and 1 to 40 Hz (unipolar signals) via a WorkMate electrophysiology system (Abbott Labs, St. Paul, MN). The pace map score was calculated by software installed in the CARTO system (PASO Module, Biosense Webster). A 6F 20-pole dual-site mapping catheter (BeeAT; Japan Lifeline Co, Ltd, Tokyo, Japan) was inserted through the internal jugular vein. A decapolar catheter (Pulscouter; Kaneka Medical Products, Osaka, Japan) was also placed in the His bundle region. An intracardiac echocardiography probe with a CARTO navigation sensor imbedded close to the phased array (SoundStar™) was positioned in the right atrium (RA) and then moved to the right ventricle (RV), tracing the endocardial surface of the RV and LV. The patient presented in sinus rhythm with frequent PVCs (Figure 1A). Although electrophysiological studies were not performed

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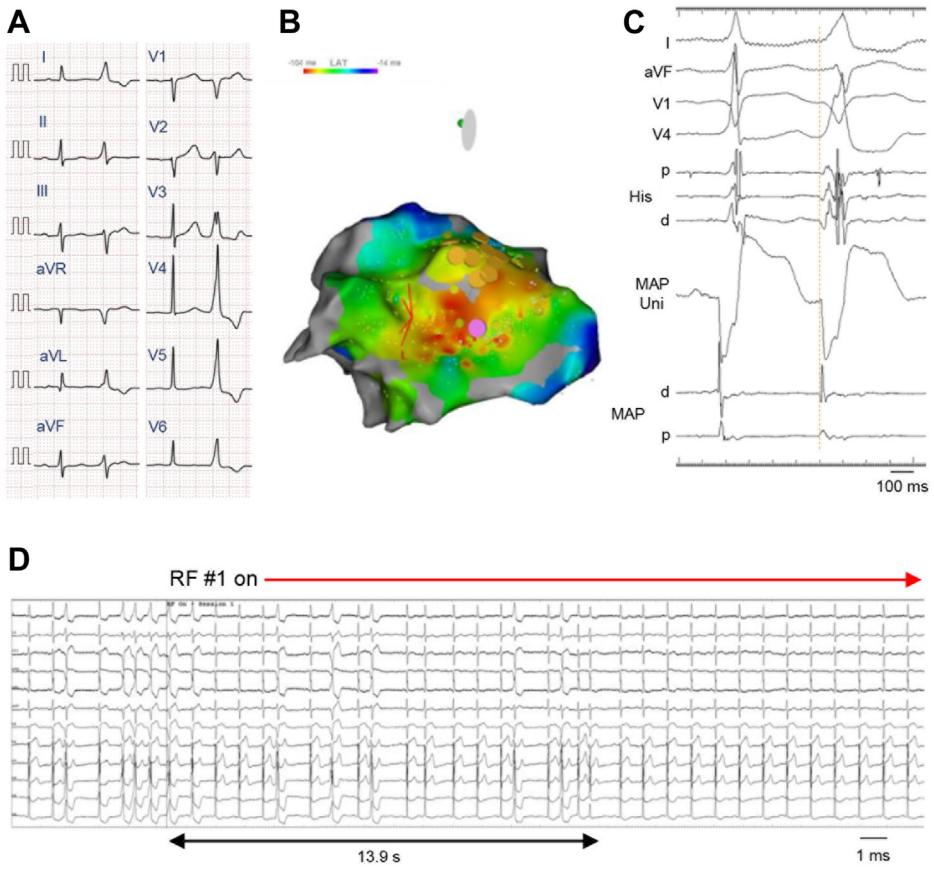

Figure 1 **A:** Twelve-lead electrocardiogram showing sinus rhythm and subsequent premature ventricular contraction (PVC). **B:** Electroanatomical mapping of the PVC showed the earliest activation site located at the basal mid-septum of the right ventricle (RV) and a centrifugally spreading activation pattern. The pace mapping corresponding to this site (*pink tag*) was a perfect match. Areas with His and right bundle branch potentials are tagged in yellow. **C:** The unipolar electrogram recorded at the earliest site showed an rS pattern, and the bipolar electrogram preceded QRS onset by 13 ms. **D:** The initial ablation attempt at the earliest activation site in the RV resulted in delayed suppression. MAP = mapping catheter; RF = radiofrequency.

to evaluate AV conduction properties and to induce supraventricular tachycardias, a jump-up of the AH interval was observed at baseline (Supplemental Figure 1). An activation map was created with a Pentaray catheter (Biosense Webster) (Figure 1B), which identified the earliest activation site at the basal mid-septal wall of the RV (13 ms earlier than onset of the QRS, Figure 1C). At the same site, pace mapping with a high output setting of 8 V and 2.0 ms showed an excellent pace map score of 98%. The first RF energy application performed at that site with a ThermoCool STSF catheter (Biosense Webster) suppressed the PVCs (Figure 1D) in 13 seconds, but 2 minutes later the PVCs recurred. We experienced similar responses of the PVCs to 11 subsequent RF applications around that area (average contact force 8–18 g at a power of 25–35 W). However, the effect of all ablation attempts was transient, and the ventricular activation sequence and QRS wave morphology did not change.

As the patient had been diagnosed as having symptomatic paroxysmal atrial fibrillation, we performed atrial trans-septal puncture and bilateral pulmonary vein isolation. After the confirmation of bidirectional block in the pulmonary veins with a circular decapolar catheter (Lasso; Biosense Webster), the LV was mapped by the ThermoCool STSF catheter (Figure 2A). As the burden of the PVCs was reduced after the RV ablation, we did not perform activation mapping but carefully attempted sequential unipolar ablation inside the LV anatomically. During the PVCs, the ablation catheter placed just opposite to the temporarily successful site in the RV showed an rS pattern on the distal unipolar electrode, and the bipolar electrogram showed a local potential occurring just at the same time (0 ms) as QRS onset (Figure 2B). Ablation was carefully attempted, and during the RF energy application junctional rhythm suddenly appeared, with subsequent complete AV block (Figure 2C). Just prior to the occurrence of complete AV block after the rapid junctional beats, there were P waves with a long AV interval, suggesting that the fast pathway was initially injured, followed by injury to the slow pathway, which finally resulted in complete AV block.

Fortunately, AV conduction quickly recovered spontaneously, and the clinical PVCs had completely disappeared during this application. Although the HV interval was unchanged, the AH interval was prolonged by 140–280 ms (Figure 3A). Comparison of fluoroscopic views between the RV and LV ablation sites showed that the ablation site in the LV was located inferiorly to the His bundle region

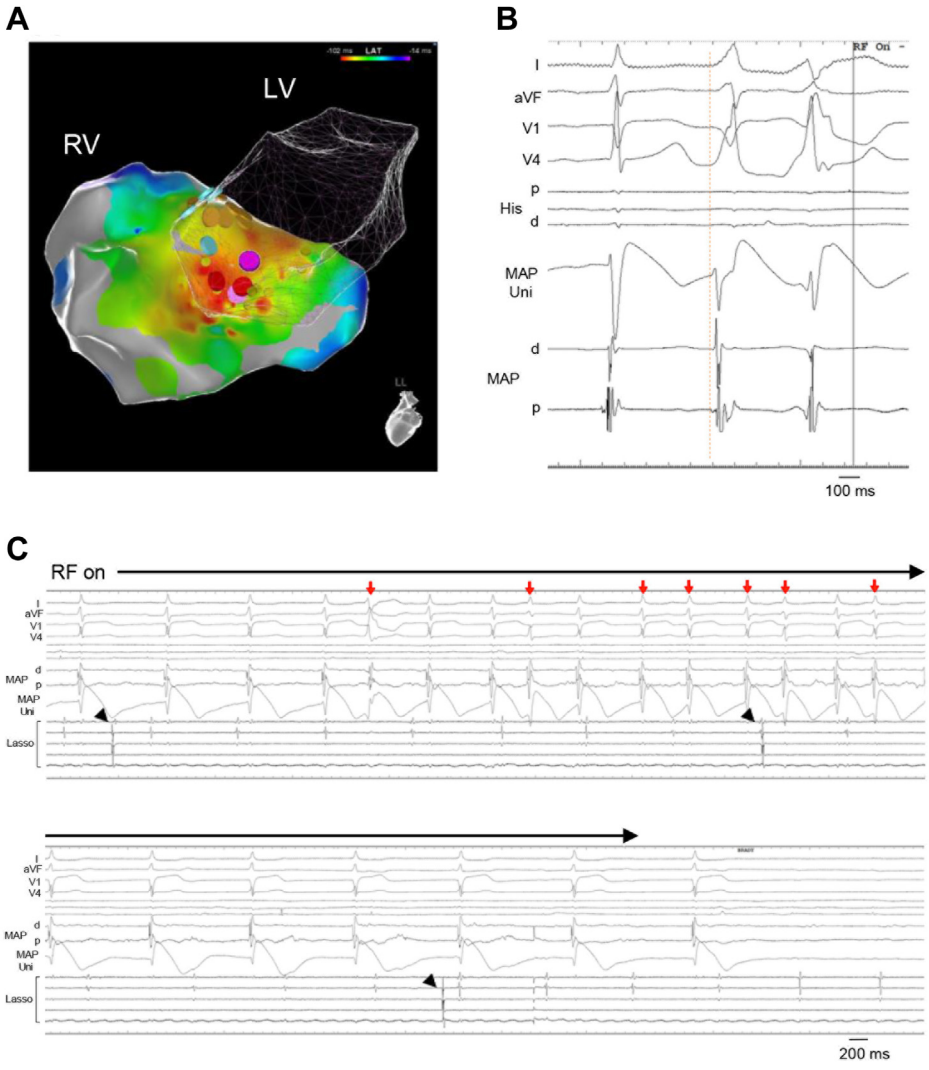

Figure 2 **A:** Right ventricle (RV) endocardial activation map and mesh overlay of the left ventricle (LV) endocardial map. The basal inferoseptum of the LV was targeted for ablation (*pink tag*), which resulted in the elimination of the premature ventricular contraction and caused a transient complete atrioventricular (AV) block. His potentials are tagged in yellow and left bundle branch potentials are in sky blue. **B:** The electrograms at the successful ablation site inside the LV are shown. The unipolar electrogram showed an rS pattern, and the bipolar electrogram was timed approximately with onset of the QRS. Unfortunately, the decapolar catheter that had been placed in the His area was displaced and pulled out into the inferior vena cava, as it interfered with Pentaray mapping inside the RV. **C:** The electrograms recorded during ablation inside the LV show junctional rhythms accompanied by occasional ventriculoatrial block (*red arrows*) and subsequent complete AV block. The Lasso catheter placed inside the left superior pulmonary vein (LSPV) recorded far-field atrial activation. Black arrowheads indicate dissociated activities inside the LSPV. CS = coronary sinus; d = distal; MAP = mapping catheter; p = proximal; RF = radiofrequency.

(Figure 3B). Intracardiac echocardiography was retrospectively analyzed, and it revealed that the right atrial septum was located opposite to the ablation site (Figure 3C).

The patient has remained free from PVCs, without any antiarrhythmic drugs, for 6 months. Fortunately, implantation of a pacemaker has not been required, although first-degree AV block (PR interval of 220 ms) has persisted.

Discussion

To the best of our knowledge, this is the first report of ablation from the inferoseptal recess of the LV base resulting in transient AV block and subsequent persistent prolongation of the AH interval, suggesting proximity of the ablation site to the fast pathway or compact AV node.

In general, the possible mechanisms of AV block during catheter ablation for ventricular arrhythmias are due to damage to the His bundle itself or to further impairment to the branch or the fascicle in patients with known conduction disturbances.[1] To this end, the identification of any pre-existing conduction abnormalities as well as the anatomic area of the His-Purkinje system with a 3D electroanatomical mapping system is imperative. However, contrary to this putative evidence, our case showed supra-Hisian injury (fast pathway) caused by ablation to the LV basal inferoseptal region.

From the anatomical aspect, the tricuspid valve is inferiorly displaced compared to the mitral valve. The slow pathway region from the coronary sinus ostium to the tricuspid valve overlaps with the LV tissue, which forms

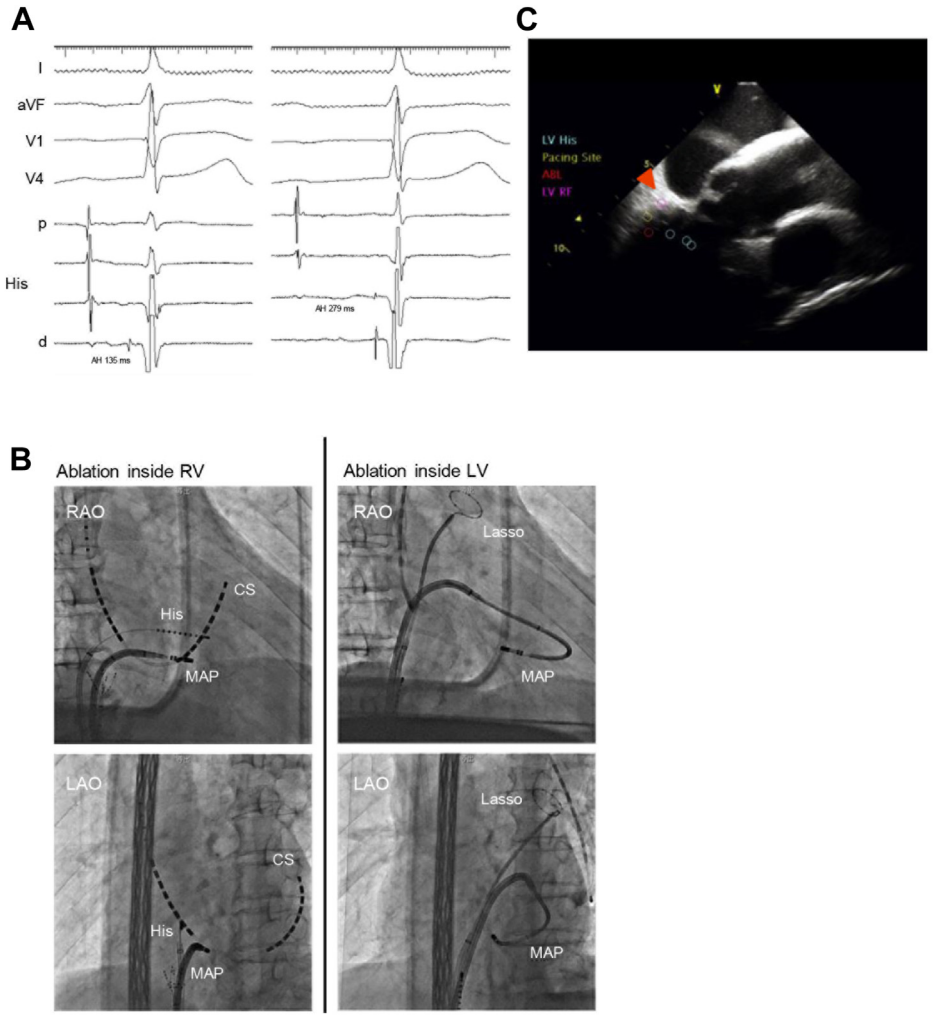

Figure 3 **A:** The electrograms before and after the ablation procedure. The AH interval was remarkably prolonged from 135 ms to 279 ms. **B:** The fluoroscopic views during ablation. In the 2 panels on the left, the mapping catheter was located at the site where the premature ventricular contraction was temporarily suppressed by the ablation; in the 2 panels on the right, it was placed at the site of complete suppression and at which transient atrioventricular (AV) block occurred. **C:** Intracardiac echocardiographic images delineated the proximity of the right atrium and the basal inferoseptal wall of the left ventricle (LV). The pink tag highlighted by the red arrowhead indicates the site where the radiofrequency application caused transient AV block and subsequent persistent prolongation of the atrial-His interval. Ao = aorta; CS = coronary sinus; HV = His-ventricular; LA = left atrium; LAO = left anterior oblique; MAP = mapping catheter; RA = right atrium; RAO = right anterior oblique.

the "AV septum." Besides being a substrate of supraventricular arrhythmia, this site also has a ventricular arrhythmogenic role. Briceño and colleagues[2] reported that PVCs arising from the slow pathway region could be successfully treated by right atrial ablation to this region in 11 of 12 (92%) patients. We did not ablate the RA in the present patient because the activation time from the RA was not earlier than that from the RV (13 ms pre-QRS) and LV endocardium (0 ms pre-QRS). Santangeli and colleagues[3] also reported PVCs that were eliminated by RF (n = 3) or cryoablation (n = 2) in the RA where a small atrial potential could be recorded, and the activation time from within the RA was significantly earlier than that from the LV endocardium in their case series of 5 patients (mean 32 ± 7 ms pre-QRS for RA vs 16 ± 5 ms pre-QRS for LV). It is well known that in slow pathway ablation for AVNRT, approximately 1% of patients require ablation of the inferoseptal aspect of the *left atrium*.[4] Of interest, 1 case report noted that approaching the AV septum from the basal septal part of the *LV* is necessary for the elimination of AVNRT.[5] That case indicated to us that even if mapping catheters do not record any atrial potentials in the LV, RF energy can modify and injure the slow pathway.

Taken together with these 3 reports,[2–4] it is reasonable that ablation of the inferoseptal aspect of the basal LV can transmurally injure the slow pathway region in the RA. Thus, the most important and rare point of the present case is that the fast pathway or compact AV node was simultaneously compromised, and PR (AH) prolongation persisted over time owing to LV ablation. Li and colleagues[6] reported that 7 patients with ventricular arrhythmias originating from this inferoseptal recess of the LV base were successfully treated by endocardial ablation from the LV with no complications.

The fluoroscopic views in the present study apparently show that the ablation site in the LV was located inferiorly but not superiorly or close to a common site of the His bundle, and therefore this may be unusual as a site of a fast pathway or compact AV node from the anatomical point of view. One of the possible reasons is that this patient was a very elderly man with ischemic heart disease. A short distance from the His recording site to the ostium of the coronary sinus (small Koch's triangle) is often encountered in elderly patients. Momose and colleagues[7] reported that a small Koch's triangle correlated with an elongated ascending aorta, larger LV dimensions, and aortic unfolding. In the present patient, the dilatation and elongation of the ascending aorta were significant, which could have shifted the His bundle recording site downward.

Finally, we need to describe this case from the technical point of view. During PVC ablation, no atrial potentials except the far-field potentials inside the isolated PVs were recorded. A decapolar catheter for His bundle mapping was displaced into the inferior vena cava, and a BeeAT catheter was not placed in the CS because the sheath in the internal jugular vein was used for RV mapping via a superior approach. This was one reason for the delayed recognition of the junctional rhythm and ventriculoatrial block during ablation. We assumed that bipolar ablation would be another therapeutic option, but it is not formally approved in our country and may have carried a risk for greater damage to the fast pathway and compact AV node.

Although previous studies have confirmed the significant anatomical relation between the slow pathway region in the RA and the inferoseptal aspect in the basal LV, electrophysiologists should also be concerned about the risk of injury to the fast pathway or compact AV node by LV inferoseptal ablation because anatomical variation of the fast and slow pathways can be present, especially in elderly patients with structural heart disease.

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2023.05.005.

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