Recovery of cardiomyopathy induced by ventricular premature beats of paraHisian origin after successful radiofrequency catheter ablation

Parahis çıkışlı ventriküler erken atımlar ile tetiklenen kardiyomyopatinin başarılı radyofrekans ablasyon sonrası düzelmesi

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Summary- A 51-year-old male patient presented with frequent and symptomatic premature ventricular complexes (PVCs) that had induced cardiomyopathy. Radiofrequency (RF) catheter ablation of the origin of the PVCs was planned and the patient underwent electrophysiological study. During activation mapping, the earliest ventricular activation was identified at the His bundle region. Small His bundle electrogram recording by the distal ablation electrode during sinus rhythm revealed that the earliest ventricular activation during PVC preceded the QRS onset by 58 milliseconds. The PVCs were successfully eliminated without occurrence of atrioventricular block using incremental application of RF energy to the para-Hisian region. At the conclusion of a 5-month follow-up period, the patient remained asymptomatic and transthoracic echocardiography demonstrated an improvement in left ventricular ejection fraction.

Catheter ablation is preferred in the treatment of patients with premature ventricular complex (PVC) who remain symptomatic and resistant to medical therapy.^[1] In particular, the catheter ablation method is widely used to successfully treat PVCs originating from the right ventricular (RV) outflow tract.^[2] Frequent PVCs may cause ventricular dyssynchrony and increased oxygen demand, thereby resulting in PVCinduced cardiomyopathy with left ventricular (LV) dysfunction.^[3] Several studies have demonstrated the beneficial effects of PVC radiofrequency (RF) catheter ablation therapy in improving the LV function **Özet**– Elli bir yaşında erkek hastada sık ve semptomlu ventriküler erken atımlara (VEA) bağlı VEA ile tetiklenen kardiyomiyopati saptandı. Ventriküler erken atım odaklı radyofrekans (RF) kateter ablasyonu planlanarak hastaya elektrofizyolojik çalışma yapıldı. Aktivasyon haritalaması sırasında, en erken ventriküler aktivite his alanında saptandı. Sinüs ritmi sırasında küçük His elektrogramı Ablasyon distal de (ABL-d) saptanmakta ve VEA sırasında en erken ventriküler aktivite QRS başlangıcından 58 msn önce görülmekte idi. Parahisyan bölgeye yapılan giderek çoğalan RF enerji uygulaması ile atriyoventriküler blok olmadan VEA başarı ile sonlandırıldı. Beş aylık takip sürecinde, hasta semptomsuz seyretti ve transtorasik ekokardiyografi ile sol ventrikül ejeksiyon fraksiyonunda artma gösterildi.

in cases of PVC-induced cardiomyopathy.^[4,5] PVCs originating from the para-Hisian region are rare and may be the cause of idiopathic ventricular arrhythmia. Catheter ablation therapy applied to this region has a high-risk of prompting atrioventricular (AV) block.^[6,7]

Abbreviations:

- AV Atrioventricular
- ECG Electrocardiography
- LV Left ventricular
- PVC Premature ventricular complex
- RF Radiofrequency
- RV Right ventricular
- VT Ventricular tachycardia

Presently described is a case of PVC originating from the para-Hisian region causing PVC-induced



cardiomyopathy, which was successfully treated with RF catheter ablation.

CASE REPORT

A 51-year-old male presented at the cardiology outpatient clinic with the complaints of palpitations, dyspnea, and dizziness. A 12-lead electrocardiography (ECG) on admission revealed monomorphic PVCs. Additionally, PVC had a left bundle-branch block configuration, monophasic R wave in leads I and aVL, as well as a precordial transition between leads V2-V3 (Fig. 1). His symptomatic PVCs had been refractory to Class I antiarrhythmic agents for about 1 year. LV ejection fraction was 45% on echocardiographic examination based on the modified Simpson method, the LV was dilated, and there was global hypokinesia. The results of 24-hour ambulatory ECG monitoring revealed bigeminal and trigeminal PVCs in a monomorphic pattern (31,046 beats; 29.7% of total beats). No episode of ventricular tachycardia (VT) was observed. Coronary angiography was performed to evaluate the presence of coronary ischemia and showed no significant stenosis in the coronary arteries.

Informed consent was obtained from the patient and a cardiac electrophysiology study was performed in a fasting non-sedated state. Antiarrhythmic agents had been discontinued for at least 5 half-lives prior to the study. Baseline ECG revealed PVCs with the same configuration observed in the clinical PVC. For mapping and pacing, multiple-electrode catheters were introduced and positioned in the high right atrium, RV apex, and the His bundle region through the

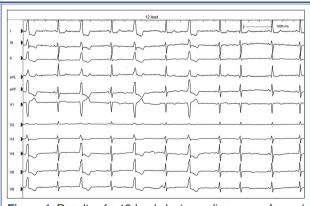


Figure 1. Results of a 12-lead electrocardiogram performed on admission. Bigeminal premature ventricular complexes with a left bundle-branch block configuration and normal axis morphology. femoral vein under fluoroscopic guidance. Although the patient had bigeminal and trigeminal PVCs, programmed electrical stimulation and incremental burst pacing to the RV apex and the RV outflow tract did not induce sustained VT. The 12-lead ECG and intracardiac electrograms were monitored continuously.

Detailed activation and pace mapping, and RF ablation of the site of PVC origin were performed using a 7-F conventional ablation catheter with a deflectable tip and a 4-mm distal electrode (Fig. 2). During activation mapping, the earliest ventricular activation was identified in the His bundle region. A small His bundle electrogram recording by the distal ablation electrode during sinus rhythm revealed that the earliest ventricular activation during PVC preceded QRS onset by 58 milliseconds (Fig. 3). The QRS morphology of

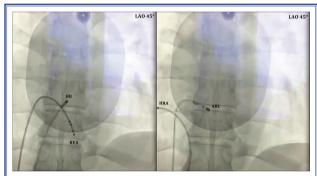


Figure 2. Fluoroscopic images and catheter position at site of successful ablation.

ABL: Ablation catheter; HB: His bundle; HRA: High right atrium; RVA: Right ventricular apex.

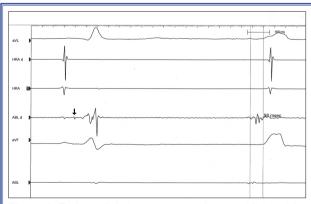


Figure 3. Endocardial electrograms at the para-Hisian ablation site exhibiting the earliest ventricular activation during the premature ventricular complex (PVC). His bundle electrogram was recorded from the distal electrode of the ablation catheter during sinus rhythm (black arrow) and the earliest ventricular activation observed during PVC preceded QRS onset by 58 milliseconds. the PVCs obtained with pace mapping of this potential ablation site in which early endocardial activation was observed was similar to clinical PVCs seen on 12-lead ECG. Pace mapping from the ablation catheter perfectly matched (12 of 12 leads) the configuration of the clinical PVCs. Subsequently, RF energy was delivered to sites close to the His bundle region using a temperature control system set at 55°C and a maximum power of 30 W, and RF catheter ablation was carefully performed in the His bundle region. RF ablation was not performed in the areas with the largest His bundle potential in order to avoid complete AV block. RF ablation technique used was to apply incremental quantities of RF energy, beginning at 15 W, for 5 to 10 seconds to the area at least 5 mm from the spots with the largest His bundle potential. No prolonged AV interval, abnormal increase in impedance, or consecutive junctional ectopic beat was observed during RF ablation. RF energy was gradually increased (target temperature of 55°C). At 30 W of RF energy, the PVCs suddenly disappeared in the first 5 seconds. RF ablation at 30 W of RF energy was performed 3 times, each lasting 10 seconds. No PVCs were observed during the waiting period of 30 minutes after each application of RF ablation. Following the procedure, a 12-lead ECG showed no PR prolongation, AV complete block, or bundle-branch block. At the conclusion of a 5-month follow-up period, the patient remained asymptomatic, and a 24-hour ambulatory Holter recording was performed. The results of the monitoring indicated a PVC burden of 0.2% without any antiarrhythmic agent. Transthoracic echocardiography also revealed an improvement in LV ejection fraction (55% according to modified Simpson method).

DISCUSSION

PVC-induced cardiomyopathy provoked by frequent and symptomatic PVCs originating in the para-Hisian region was successfully treated with RF catheter ablation.

Previous studies have demonstrated a relationship between frequent PVCs and the development of cardiomyopathy. Factors such as a high PVC burden, symptom duration, epicardial origin of focus, and QRS duration have been shown to be involved in the development of PVC-induced cardiomyopathy.^[8,9] It is, therefore, critical to detect the origin of PVCs in patients with PVC-induced cardiomyopathy for treatment with RF catheter ablation.

Yamauchi et al.^[10] reported that VT/PVCs originating in the area close to the His bundle had distinctive characteristics on a 12-lead ECG. In that case, para-Hisian VT/PVCs had a monophasic tall R wave in lead I, a relatively small R wave in lead III compared with lead II, an R wave in lead aVL, a relatively narrow QRS duration in the inferior leads, a QS pattern in lead V1, an early precordial transitional zone in leads V2-V3, and a relatively tall R wave in leads V5, V6. Similarly, in our case, the PVC morphology was consistent with the para-Hisian focus observed in ECG recordings upon admission. Ashikaga et al.^[7] reported successful RF catheter ablation of PVCs with para-Hisian origin and an earliest ventricular activation preceding QRS onset by 33 milliseconds using incremental RF energy. We also performed successful RF ablation of para-Hisian PVCs where a small His bundle electrogram recorded during sinus rhythm revealed that the earliest ventricular activation during PVC preceded the QRS onset by 58 milliseconds. We believe that using incremental quantities of RF energy, performing RF ablation at least 5 mm from the largest His bundle potential, and delivering RF energy to the distal His area covered with a central fibrous body reduced the risk of AV block.

Following effective RF catheter ablation of PVCs in patients with PVC-induced cardiomyopathy, the determinants of recovery of LV functions and the time course are not well established. Deyell et al.[11] reported that QRS duration of PVC was an independent predictor of the recovery of LV function. Similarly, Yokokawa et al.^[12] showed that recovery of LV function might be prolonged following successful ablation of PVC with prolonged QRS duration and epicardial origin, and that this period might be a mean of 12±9 months (range: 5-45 months). In the present case, after a 5-month follow-up period, transthoracic echocardiography revealed early recovery of LV function. We suggest that the relatively narrow PVC-QRS duration in the 12-lead ECG and PVCs with early ventricular activation originating from the endocardial surface might have contributed early recovery of LV function in this patient.

Conclusion

In conclusion, we performed a successful RF catheter ablation of PVCs originating in the para-Hisian region in a patient with PVC-induced cardiomyopathy under the guidance of activation mapping. The use of incremental quantities of RF energy and positioning the ablating catheter far from the largest His bundle potential with detailed mapping reduced the risk of AV block. Based on our experience, we find that RF catheter ablation treatment to the origin of a high PVC burden can be used in PVC-induced cardiomyopathy, which is a reversible cause of LV dysfunction.

Informed consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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Keywords: Para-Hisian; premature ventricular complex; radiofrequency ablation.

Anahtar sözcükler: Parahisyan; erken ventriküler atım; radyofrekans ablasyon.