CASE REPORT

Renal sympathetic denervation assisted treatment of electrical storm due to polymorphic ventricular tachycardia in a patient with cathecolaminergic polymorphic ventricular tachycardia

Katekolaminerjik polimorfik ventriküler taşikardili bir hastada polimorfik ventriküler taşikardiye bağlı elektriksel fırtınanın renal sempatik denervasyon yardımıyla tedavisi

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Summary– Electrical storm (ES) is not a rare clinical entity. Sympathetic hyperactivity may play critical role in development and continuation of ES. Some recent reports have demonstrated that catheter-based renal sympathetic denervation (RSD) may reduce sympathetic activation and have a potential role in reducing arrhythmic burden. A 46-year-old man was admitted to clinic with frequent implantable cardioverter defibrillator shocks and ES related to catecholaminergic polymorphic ventricular tachycardia (VT). Tachycardia was unresponsive to administration of beta-blockers, verapamil, and flecainide. Catheter ablation failed to suppress initial premature ventricular contractions. Based on aggravating effects of sympathetic system in clinical tachycardia, decision was made to perform RSD. After the procedure, sinus rhythm was achieved and no polymorphic VT was detected. Sustained monomorphic VT with right ventricular origin was successfully ablated via endocardial radiofrequency ablation. This new treatment modality may be a potential alternative method for patients in whom other ablative strategies have been unsuccessful.

Özet– Elektriksel fırtına (EF) nadir bir klinik durum değildir. Sempatik hiperaktivite EF'nin gelişmesinde ve devamında kritik bir rol oynayabilir. Yakın dönemdeki bazı çalışmalarda kateter temelli renal sempatik denervasyon'un (RSD) sempatik aktiviteyi azaltabileceği ve aritmik yükün engellenmesinde rolü olabileceği gösterilmiştir. Kırk altı yaşında erkek hasta katekolaminerjik polimorfik ventriküler taşikardi (VT) ile ilişkili sık yerleştirilebilir kardiyoverter defibrilatör şokları ve EF ile kliniğimize kabul edildi. Taşikardi atakları betabloker, verapamil ve flekainid tedavisine yanıtsızdı. Başlatıcı prematüre ventriküler kontraksiyonlar kateter ablasyonu ile baskılanamadı. Klinik taşikardideki semptatik sistemin arttırıcı etkilerinden dolayı RSD yapılmasına karar verildi. İşlem sonrası sinüs ritmi sağlandı ve polimorfik VT atağı görülmedi. Sağ ventrikül kaynaklı direngen monomorfik VT endokardiyal radyofrekans ablasyon ile ablate edildi. Bu yeni tedavi yöntemi diğer ablasyon stratejilerinin başarısız olduğu hastalarda olası bir alternatif olabilir.

Electrical storm (ES) is a clinical condition characterized by 3 or more ventricular arrhythmia episodes or ventricular fibrillation (VF) leading to appropriate implantable cardioverter-defibrillator (ICD) therapy in a 24-hour period.[1] Enhanced sympathetic nerve activity has been found to be associated with episodes of ES in previous studies.[2,3] As an inherited form of enhanced sympathetic nerve activity, catecholaminergic polymorphic ventricular tachycardia (CPVT) is characterized by exercise- or emotional stress-induced polymorphic ventricular tachyarrhythmia, syncope, sudden cardiac death, or ES[4,5] in young patients without structural heart disease. CPVT in great majority of patients is well controlled with

Received: June 18, 2016 *Accepted:* January 09, 2017
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appropriate medical therapy of beta-blockers at the highest tolerable dose, calcium blocker, flecainide, or even amiodarone.[6,7] Initially, cardiac sympathetic denervation surgery was studied as means to interrupt norepinephrine release in the heart at major source.[8,9] Armaganijan et al.[10] recently studied potential role of catheter-based renal sympathetic denervation (RSD) in patients with refractory ventricular arrhythmias.

Described in the present case is use of RSD in a patient with ES due to CPVT after failed endocardial ablation attempt and its potential therapeutic role.

CASE REPORT

A 44-year-old man diagnosed with CPVT was referred to clinic for polymorphic ventricular tachycardia (VT) presenting as ES. Medical history included implant of single chamber ICD (D284VRC Maximo II VR; Medtronic, Inc., Minneapolis, MN, USA) 1 year earlier due to CPVT and recurrent syncope episodes. Oral 50 mg/day atenolol had been prescribed as follow-up. Medical management, including beta-blocker (intravenous bolus of total 15 mg metoprolol succinate), flecainide acetate (150 mg), and 2 g magnesium was initiated immediately after admission. When attempts proved unsuccessful, antitachycardia pacing was performed via ICD. During hospitalization period, repeated electrocardiography (ECG) recordings revealed polymorphic and bidirectional VT episodes despite continuous per oral metoprolol succinate 200 mg/day and flecainide acetate (150 mg/day) (Figure 1). On fourth day of hospitalization, the patient suffered aborted sudden cardiac death due to incessant polymorphic VT detected on telemetry screen (Figure 2). After effective cardiopulmonary resuscitation, tachycardia was suppressed by deep sedation with endotracheal intubation. Successful extubation was performed after approximately 50 minutes.

Abbreviations:

CPVT	*Catecholaminergic polymorphic ventricular tachycardia*
ECG	*Electrocardiography*
ES	*Electrical storm*
ICD	*Implantable cardioverter defibrillator*
LV	*Left ventricle*
PVC	*Premature ventricular contraction*
RF	*Radiofrequency*
RSD	*Renal sympathetic denervation*
RV	*Right ventricle*
VT	*Ventricular tachycardia*
VF	*Ventricular fibrillation*

After hemodynamic stabilization was achieved, the patient was taken to electrophysiology laboratory. First plan to suppress VT episodes was to ablate initial premature ventricular contractions (PVCs), which were thought to trigger polymorphic VT. Written informed consent was obtained before performing endocardial ablation procedure. Due to polymorphic nature of PVCs and hemodynamic instability of the patient, non-contact mapping catheter was used to create activation mapping of all preceding PVCs. Successive attempts at entrainment were unsuccessful because of polymorphic nature of the tachycardia, so we continued ablation using only activation mapping. The first targeted PVC was thought to originate in the left ventricle (LV). A 9F EnSite Array non-contact mapping catheter (St

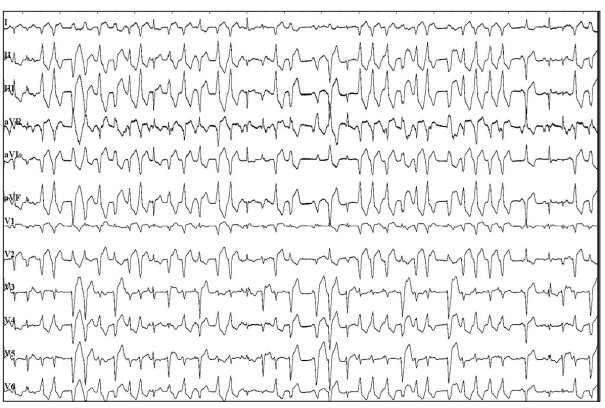

Figure 1. Twelve-lead electrocardiography recording of clinical polymorphic ventricular tachycardia.

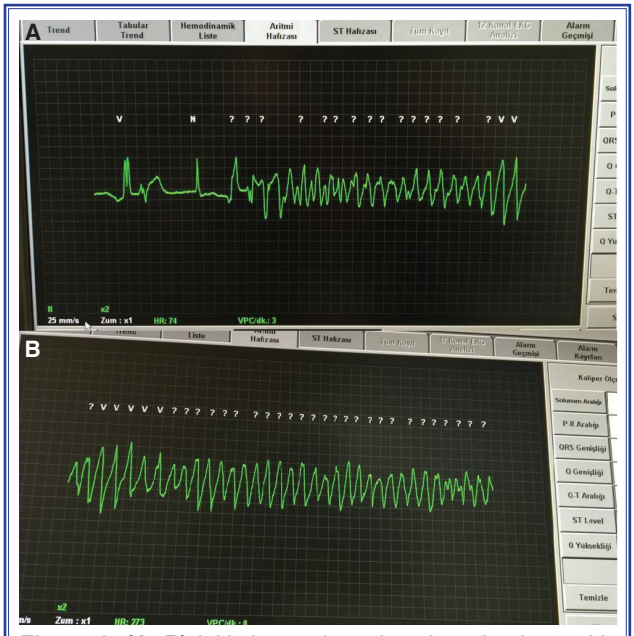

Figure 2. (A, B) Initiation and continuation of polymorphic ventricular tachycardia that caused cardiac arrest episode are seen on telemetric recordings.

Jude Medical Inc., St Paul, MN, USA) was positioned in LV using retrograde aortic approach. Activation mapping of the LV was performed with ablation catheter (Marinr; Medtronic Inc., Minneapolis, MN, USA). Earliest local ventricular point was localized at lateral wall of the LV apex (Figure 3a). After successful ablation of this site, LV septal wall and right ventricle (RV) outflow tract were successfully ablated in similar fashion (Figure 3b, c). Although successful ablation of these 3 PVCs was achieved, episodes of tachycardia continued from other sites. Therefore, decision was made to perform RSD to decrease triggering effects of sympathetic system. The patient was informed in detail and provided written consent.

Procedure was performed in the catheterization laboratory with patient under conscious sedation. To precisely define ablation sites and to potentially decrease use of radiopaque contrast agent, EnSite Velocity 3-dimensional mapping system (St. Jude Medical, Inc., St. Paul, MN, USA) was used. Catheterization of the right femoral artery was accomplished using standard Seldinger technique with 7F short sheath. Subsequently, this was replaced with steerable long sheath (Agilis NxT, St. Jude Medical, Inc., St. Paul, MN, USA) using standard "over the wire" technique. Unfractionated heparin was administered intravenously,

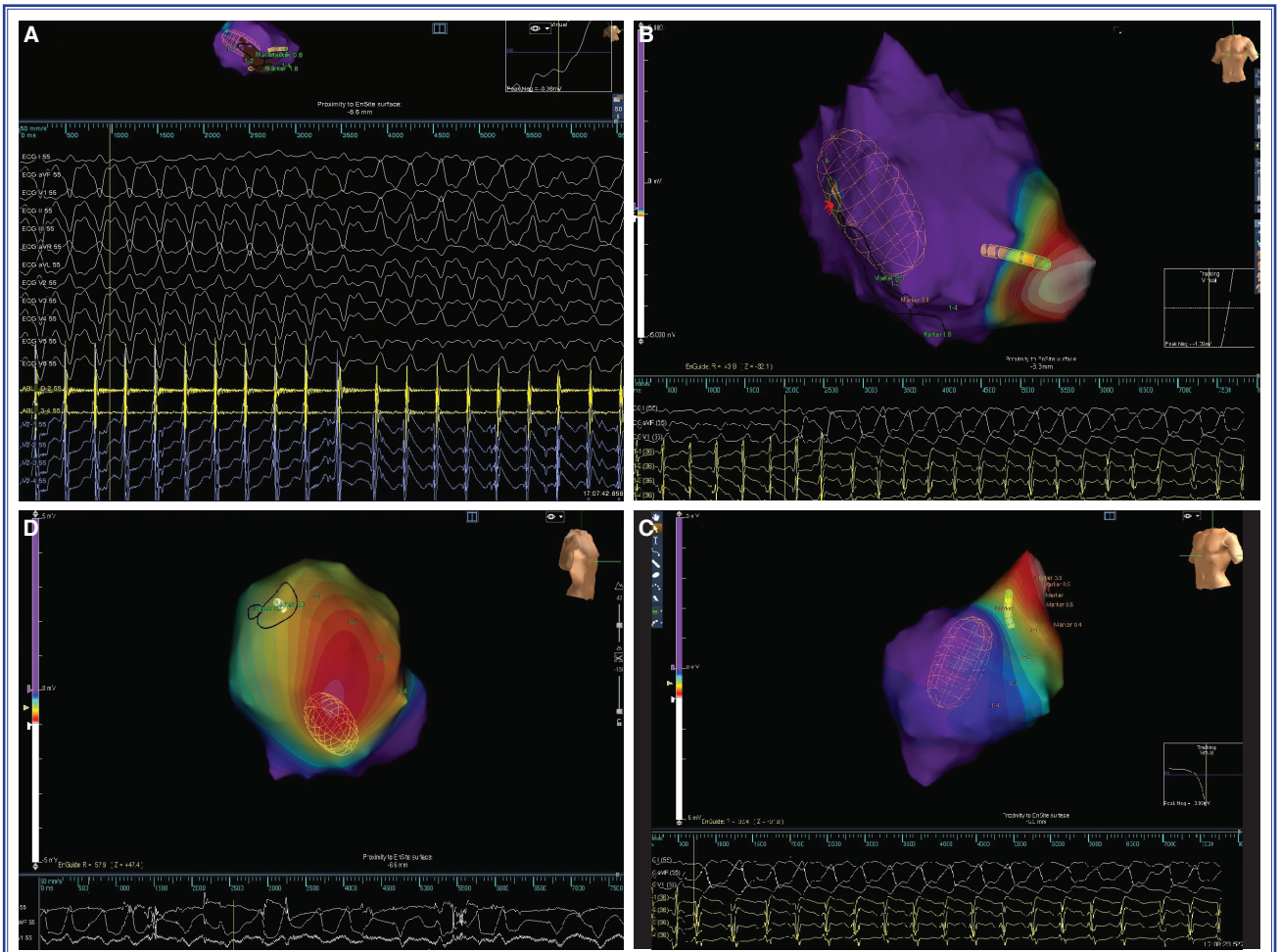

Figure 3. Non-contact mapping view of premature ventricular contractions thought to have initiated polymorphic ventricular tachycardia episodes. **(A)** Two distinct ventricular tachycardia episodes are visible in intracardiac electrograms. **(B)** The earliest activation on apical wall of the left ventricle. Yellow line indicates activation site of premature ventricular contractions considered to have initiated non-sustained ventricular tachycardia episode with right bundle branch block morphology and superior axis. **(C)** The earliest activation on posterobasal wall of the left ventricle. Yellow line indicates activation site of premature ventricular contractions considered to have initiated polymorphic ventricular tachycardia episodes. **(D)** The earliest activation on septal side of the right ventricle outflow tract. Yellow line indicates activation site of premature ventricular contractions considered to have initiated non-sustained ventricular tachycardia episode with left bundle branch block morphology and inferior axis.

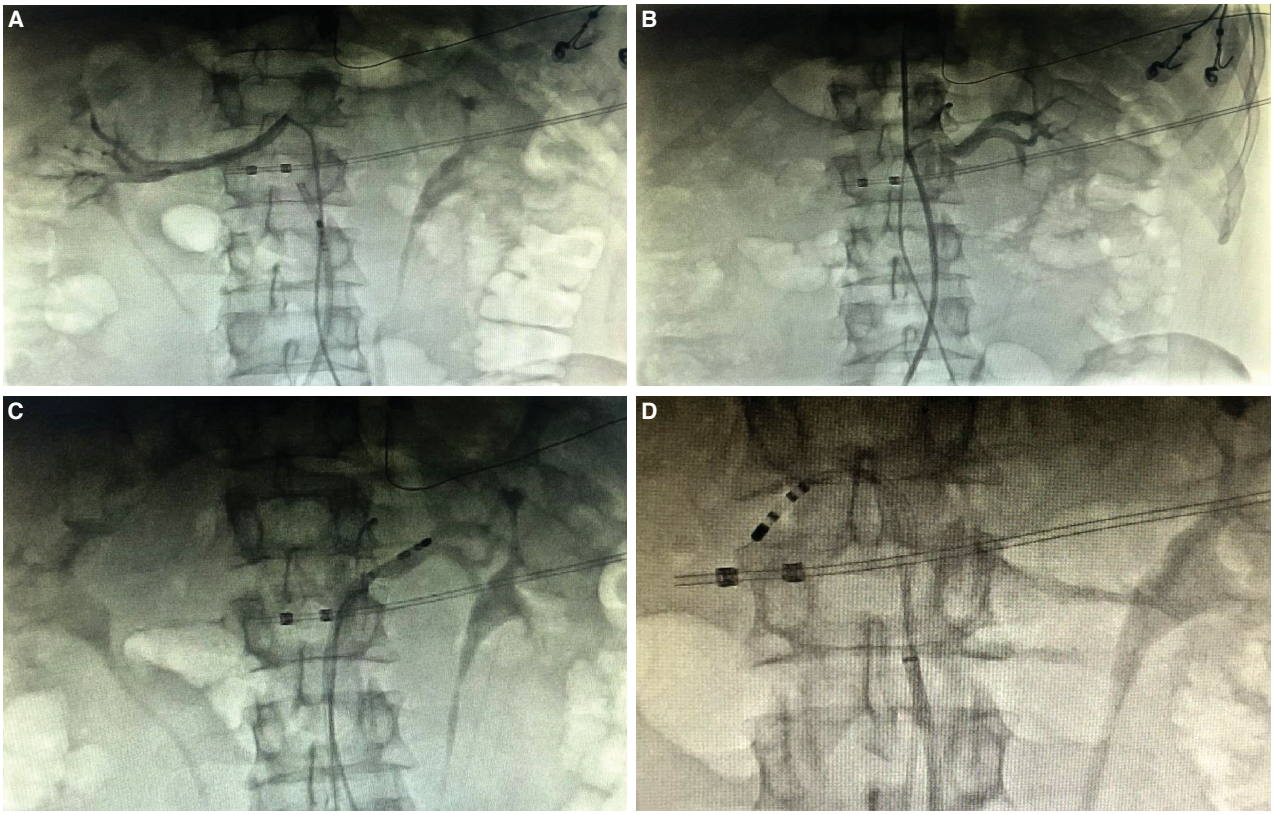

Figure 4. Fluoroscopic view of renal arteries. **(A)** The right renal artery. **(B)** The left renal artery. **(C)** Ablation catheter in the right renal artery. **(D)** Ablation catheter in the left renal artery.

targeting an activated coagulation time of between 250 and 350 seconds. Sheath was advanced to level of the renal arteries, and the ostia were located using selective renal angiography (Figure 4a, b). Introducer was then carefully deflected to the ostium of each renal artery to introduce non-irrigated Marinr ablation catheter as seen in Figure 4c, d. Total of 20 points were ablated with setting of 5 W, 45°C for 20 seconds in each renal artery (Figure 5). Impedance and temperature were continuously monitored during radiofrequency (RF) ablation. Contrast dye was injected into both renal arteries after ablation to demonstrate focal renal artery irregularities, which can be potential indicator of ablation success. Polymorphic VT episodes disappeared immediately at the end of the procedure (Figure 6). Renal function as assessed by serum creatinine and proteinuria remained unchanged from baseline. Blood pressure decreased slightly after the procedure (from 130/80 mmHg to 125/80 mmHg).

Although polymorphic VT was no longer seen during hospitalization period, monomorphic sustained VT with right bundle branch block and superior axis was detected on 12-lead ECG recording. Based on this result, patient underwent an additional endocardial RF ablation. Electrophysiological study was performed to identify the mechanism of tachycardia. Deflectable decapolar catheter was placed in the coronary sinus via the left femoral vein under

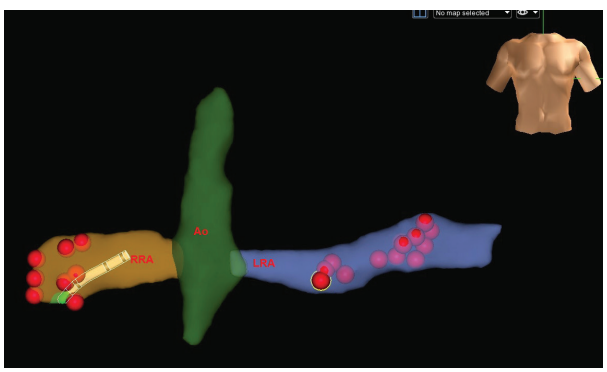

Figure 5. EnSite Velocity (St. Jude Medical, Inc., St. Paul, MN, USA) 3-dimensional electroanatomical mapping system view of renal arteries and aorta. Red spheres indicate radiofrequency application sites. Ao: Aorta; LRA: Left renal artery; RRA: Right renal artery.

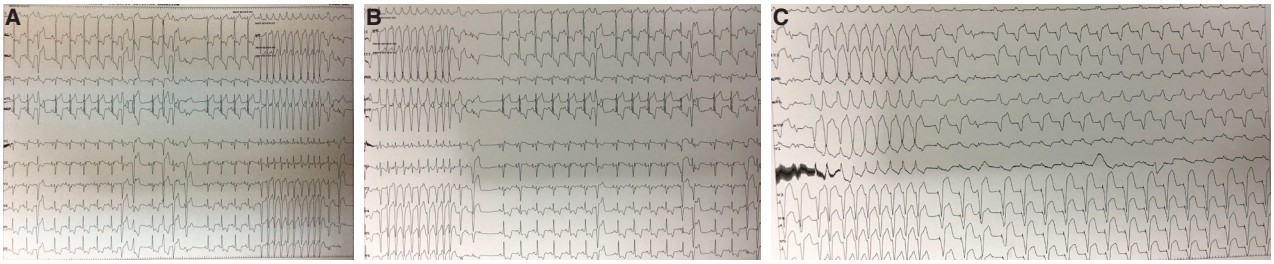

Figure 6. Twelve-lead electrocardiography recording during renal sympathetic denervation. **(A, B)** Conversion of bidirectional ventricular tachycardia (VT) to monomorphic VT **(C)** followed by paced rhythm.

fluoroscopic guidance. Quadripolar catheters were introduced through the right femoral vein and positioned in the His bundle region and the RV apex. During the procedure, VT was induced easily with either mechanical, ventricular, or coronary sinus stimulation, and rapid coronary sinus pacing temporarily suppressed VT, which returned after cessation of coronary sinus pacing (Figure 7). Spontaneous PVCs were also detected, which were identical to clinical ventricular tachycardia (Figure 8). Endocardial activation mapping of clinical VT performed using 3-dimensional electroanatomical mapping system (EnSite NavX; St Jude Medical, Inc., St Paul, MN, USA) confirmed centrifugal propagation of activation. Site of earliest activation was located in the apical septum of both ventricles (Figure 9). On the basis of all these electrophysiological findings, reentry was excluded as responsible mechanism. Distance mea-

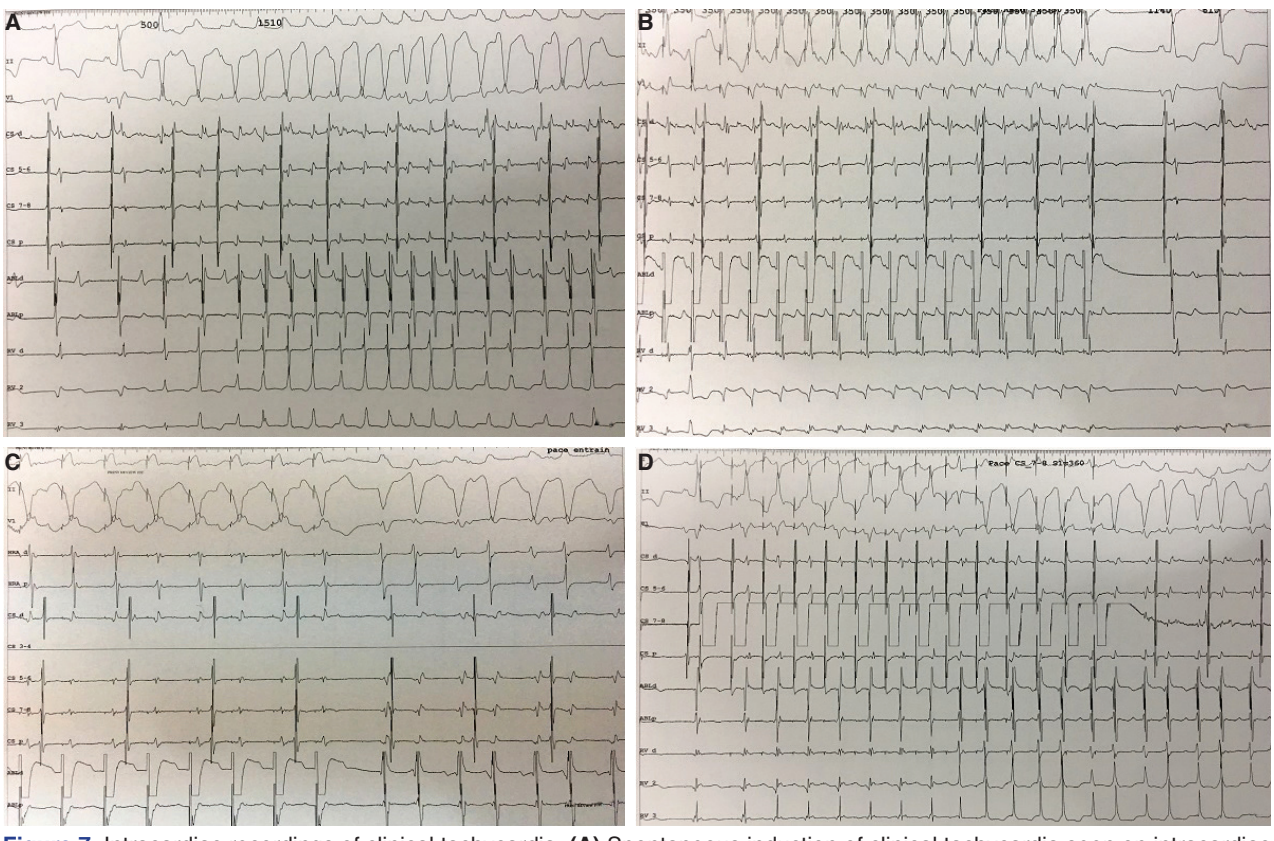

Figure 7. Intracardiac recordings of clinical tachycardia. **(A)** Spontaneous induction of clinical tachycardia seen on intracardiac recording. **(B, C)** Induction of clinical ventricular tachycardia by programmed ventricular or atrial stimulation **(D)** was not constant (B: positive, C: negative). Please note variable cycle length of tachycardia. All these electrophysiological findings may indicate that nature of clinical tachycardia is not reentry.

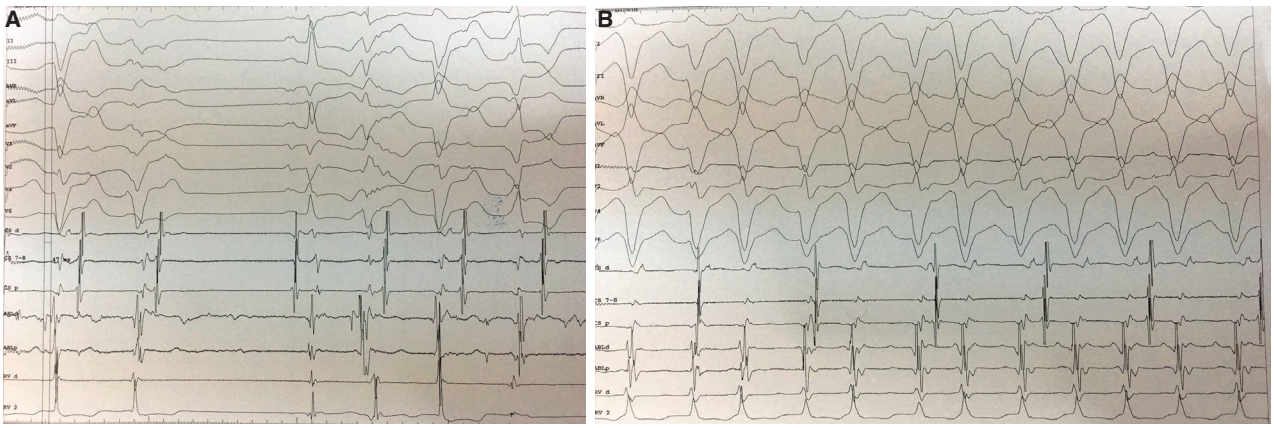

Figure 8. Intracardiac recordings of premature ventricular contraction. **(A)** Premature ventricular contraction seen on intracardiac recording. Ventricular potential recorded by distal ablation catheter (ABLD) preceded onset of surface QRS complex by 28 milliseconds in successful ablation site. **(B)** Please note that morphology of premature contraction **(A)** is exactly the same as clinical ventricular tachycardia.

sured with electroanatomical mapping between location of the earliest activation sites in the right and left ventricles was 17 mm. RF energy delivered via Marinr at earliest LV site (preceding onset of surface QRS by 28 milliseconds) did not affect VT, whereas RF energy delivered at earliest RV site (preceding onset of surface QRS by 47 milliseconds) resulted in permanent VT termination (maximum power of 50 W). Programmed RV stimulation, with and without isoproterenol, failed to induce tachycardia. Nine months after procedure, the patient was free of any arrhythmia. ICD telemetry revealed no ICD shocks and no polymorphic VT with only per oral metoprolol succinate 200 mg/day.

DISCUSSION

ES is a clinical condition characterized by 3 or more arrhythmia episodes of VT or VF leading to appropriate ICD therapies in a single 24-hour period.[1] Al-

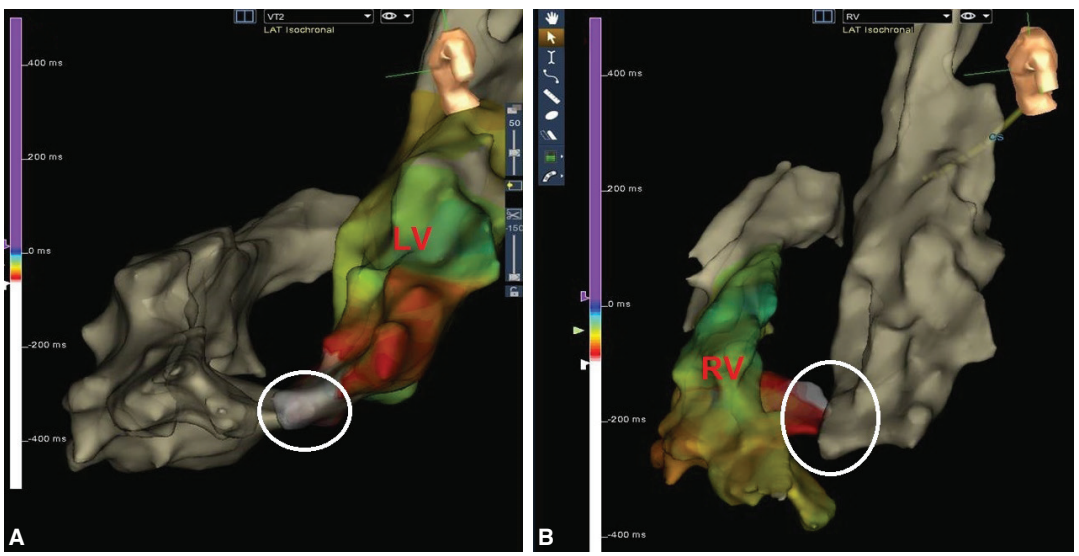

Figure 9. EnSite Velocity (St. Jude Medical, Inc., St. Paul, MN, USA) activation mapping view of the left and right ventricles. **(A)** Earliest activation site at apicoseptal wall of the left ventricle. **(B)** Earliest activation site at apicoseptal wall of the right ventricle. White spheres indicate earliest activation kissing sites in ventricles. Please note centrifugal propagation of activation wave. There is more lateral angulation on the right panel. LV: Left ventricle; RV: Right ventricle.

though advanced structural heart disease accompanies ES in the great majority of patients, rarely, it may be seen in patients with entirely normal heart structure, as in the present case.[11] CPVT is an inherited arrhythmia syndrome characterized by polymorphic VT induced by adrenergic stress.[5] To define clinical outcome, ECG characteristics, and optimal treatment of CPVT, Sumitomo et al.[6] evaluated 29 CPVT cases. Morphology of ventricular arrhythmia was polymorphic in 62%, polymorphic and bidirectional in 21%, bidirectional in 10%, and polymorphic with VF in 7%. Catheter ablation of CPVT onset focus was used in 2 cases but proved unsuccessful. Recently, Kaneshiro et al.[12] presented result of triggering ablation of CPVT. Initial PVCs were induced with epinephrine infusion. Following ablation of 2 distinct PVCs, neither PVCs nor VF were inducible, even with infusion of epinephrine of up to 1.2 μg/kg per minute (>10 times higher dose than provocation). Mapping and catheter ablation of the PVCs triggering polymorphic VT was also performed in our case. RV outflow tract and LV outflow tract under the aortic valve of the left coronary cusp were considered to be origins of PVCs. However, repeated ablation attempts failed due to occurrence of new origins.

Sympathetic denervation may be potential alternative treatment modality for CPVT. In 2008, Wilde et al.[13] reported long-term results of surgical left cardiac sympathetic denervation in 3 young adults with CPVT. Procedure was effective in all patients. They speculated that left cardiac sympathetic denervation may be viable solution for patients with CPVT who are not fully protected by beta-blockers. In a preliminary study, Atallah et al.[14] investigated potential use of left cardiac sympathetic denervation by means of video-assisted thoracoscopic surgery in 4 patients with CPVT as less invasive alternative. Symptomatic patients with available follow-up experienced marked improvement in the first month after sympathectomy.

The potential of RSD to suppress VT in humans has been explored thus far only in case reports and small series.[15–18] As first human experience, Ukena et al.[15] presented 2 patients with chronic heart failure suffering from therapy-resistant ES undergoing catheter-based RSD. One patient with hypertrophic cardiomyopathy had recurrent monomorphic VT despite extensive antiarrhythmic therapy and repeated endocardial and epicardial electrophysiological ablation attempts to destroy arrhythmogenic intramural focus in the LV. The second patient, who had dilated nonischemic cardiomyopathy, suffered from recurrent episodes of polymorphic VT and VF. Shortly thereafter, Hoffmann et al.[16] reported case of successful catheter-based RSD in the setting of acute myocardial infarction.

In a prospective nonrandomized study, Armaganijan et al.[10] reported results of catheter-based RSD to reduce arrhythmic burden in patients with ventricular arrhythmias. Total of 10 patients with refractory ventricular arrhythmias underwent RSD. Median number of VT/VF episodes/antitachycardia pacing/shocks 6 months before RSD was significantly reduced at 6-month follow-up. They concluded that RSD was associated with reduced arrhythmic burden with no procedure-related complications in patients with ICDs and refractory ventricular arrhythmias.

Increased sympathetic tone reduces ventricular effective refractory period, increases automaticity, and reduces threshold for ventricular arrhythmias.[3] Negative results observed in the Symplicity HTN-3 study (A Controlled Trial of Renal Denervation for Resistant Hypertension) raises question regarding RSD.[19] A potential explanation of negative results may be related to patient selection criteria and ablation technique. The trial included large number of participating centers. Therefore, small number of cases/center and high percentage of nonexpert operators of technique may have affected results. Furthermore, potential effect of RSD may be proportional to number of RF applications in the renal arteries. Unlike the Symplicity HTN-3 study, in which mean number of RF applications was 3.9, in the Armaganijan study and in our case, average of 6.3 and 5.2 applications were delivered to 8 and 12 lesions in the right and left renal arteries, respectively. Imnadze et al.[20] revealed that distal third of the renal arteries is more innervated than more proximal segments. However, in previous studies, proximal segments had been selected as main ablation target. Microanatomy of human renal vessels has high variability. Furthermore, accessory renal arteries and vessels that bifurcated early may influence outcome negatively. Therefore, after thorough description of microanatomy of renal nerve, we may have to change our ablation approach in the near future. The other major dilemma related to RSD is lack of procedural endpoint during intervention. Accepted

RF ablation method for RSD is defined in previous studies. Catheter is advanced 4 to 5 mm and rotated after each delivery, thus producing spiral configuration of ablations. As a potential limitation of our approach, ablation points were closer than suggested in the relevant literature. Although our main aim was to achieve more complete denervation by increasing number of ablation points, this technique cannot be suggested for all cases and may be questionable.

In our case, first detected arrhythmia was polymorphic in nature. Main reason endocardial ablation attempts failed was related to multiple origins of PVC, which is typical in CPVT. Due to close relationship between increased sympathetic activity and VT, RSD was considered reasonable therapeutic option for the patient, and effectively suppressed polymorphic VT episode. However, it should be kept in mind that second clinical tachycardia, which was monomorphic, may have same origin as first. Namely, monomorphic VT may have presented with polymorphic episodes due to sympathetic discharge. In CPVT, polymorphic VT is more common than monomorphic VT and may lead to VF. Although infrequent, there is small case series of CPVT combined with sustained monomorphic VT.[21] Due to small sample size, however, true nature of monomorphic VT was not clearly defined. On the basis of electrophysiological findings, triggered activity was considered main responsible mechanism for second arrhythmia. These arrhythmias may share common arrhythmogenic substrate with CPVT, but further studies are necessary to establish relationship. In our case, polymorphic VT episodes due to sympathetic overactivity were successfully terminated with RSD. Monomorphic VT, which may be the primary responsible substrate for clinical arrhythmia, was subsequently treated with endocardial ablation. Neither monomorphic nor polymorphic VTs were seen on ICD recordings at conclusion of 9 months of follow-up.

Conclusion

At this time, RSD is not accepted as a standard or preferred technique in treatment of polymorphic ventricular arrhythmias. However, some portion of ventricular arrhythmias does not respond to endocardial or epicardial ablation. This is especially true for ventricular arrhythmias related to sympathetic hyperactivity.

Conflict-of-interest: None declared.

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Keywords: Ablation; arrhythmia; sympathetic system; ventricular tachycardia.

Anahtar sözcükler: Ablasyon; aritmi; sempatik sistem; ventriküler taşikardi.