


Chemical ablation of ventricular tachycardia arising from the left ventricular summit

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Introduction

A 38-year-old male patient underwent endocardial catheter ablation (EDCA) of ventricular tachycardia (VT) using irrigated radiofrequency energy (RF). Although EDCA could terminate VT, it recurred. Retrograde ethanol infusion into cardiac vein, through which venous blood drained from focus of VT, terminated VT without any adverse events and rendered it noninducible.

Catheter ablation of ventricular tachycardia (VT) arising from the left ventricular summit (LVS), which is associated with an intramural origin, close proximity to the coronary vessels, and inaccessibility to an epicardial approach, is challenging [1, 2]. Transarterial coronary ethanol infusions have been reported as a last resort to treat VT [3–5]; however, this procedure has various risks. To overcome that limitation, a retrograde coronary venous ethanol ablation (RCVEA) has been described as an alternative to the arterial approach [6, 7]. In this report, we present a patient with a sustained VT complicated by dilated cardiomyopathy (DCM), who could be treated with an RCVEA.

Key Clinical Message

Ventricular tachycardia (VT) arising from the left ventricular summit is very tough to treat endocardially and epicardially due to the distance from the VT origin and close proximity to the coronary arteries, respectively. Ethanol infusions into coronary veins feeding VT origins were able to safely abolish this type of VT.

Keywords

Ablation, clinical electrophysiology, ventricular tachycardia.

Case Report

A 38-year-old man underwent catheter ablation of a drug refractory VT (Fig. 1A), which was associated with DCM. This patient had already undergone an implantation of an implantable cardioverter–defibrillator (ICD). Amiodarone at 200 mg had failed to suppress this VT, and a combination therapy of 80 mg of dl-sotalol and 300 mg of mexiletine was prescribed to prevent VT. However, the patient experienced multiple appropriate treatments by the ICD despite optimal pharmacological treatment. Before the ablation procedure, coronary angiography was performed to confirm the location of the coronary arteries around the ablation site. At first, he underwent an endocardial catheter ablation using irrigated radiofrequency current energy (RF) at the site where the diastolic ventricular electrogram recorded from the tip of the ablation catheter preceded that of the onset of the QRS complex by 88 msec (Fig. 1B). We performed entrainment mapping from an endocardial site with a cycle length 20 msec shorter than that of the VT cycle length,

and the postpacing interval was almost equal to that of the VT. In addition, the pace-mapping score using the CARTO system (Diamond Bar, CA) demonstrated a high mapping score of 12/12 and high PaSo score of 0.96 (Fig. 1C). Irrigated RF current energy at a power of 35 W for 2 min was delivered around that site seven times during the VT and resulted in the termination of the VT within 12 sec after the first application of RF energy. Even with intravenous isoproterenol, an aggressive stimulation protocol with triple ventricular premature extrastimulation failed to induce the VT, which was easily provoked before the ablation procedure. Two days after the first ablation session, this VT spontaneously recurred during hospitalization. Due to the close proximity to the coronary artery, which inhibited an epicardial approach, we were determined to perform a chemical ablation to treat this VT. After obtaining informed consent, we inserted long guiding sheaths (outer sheath; GCS aim SL 59 cm, inner sheath; GCS direct SL II 50 cm, Abbott) into the coronary sinus via the right internal jugular vein as deep as possible. Coronary venograms were performed using an occlusion balloon wedge pressure catheter (Harmac

Medical Product Inc., CA). We tried to insert a 2 Fr multipolar electrode catheter for mapping and pacing; however, we failed to position the tip of that catheter at an epicardial site opposite the best endocardial site as guided by the earliest activation or best pace-mapping. Then, we introduced a 1.5 mm microinfusion catheter used for coronary angioplasty (Ryujiin, TERUMO, Tokyo) into a small coronary venous branch in an “over-the-wire” fashion through a guiding wire that was selectively cannulated into the target coronary venous branch. We inflated the balloon of the infusion catheter to 4 to 6 atmospheres aiming to occlude the selected vein and injected contrast medium through the central lumen to investigate whether this branch ran toward the target site. Coronary sinus venography was performed, and the fluoroscopic image demonstrated a spatial relationship between the endocardial transient successful site and culprit venous branch (Fig. 2A,B). The distance between those two sites was 14 mm measured from the fluoroscopic image. Coronary angiography was also performed to investigate the spatial relationship between the culprit venous branch and closest coronary artery (Fig. 2C,D). We infused saline

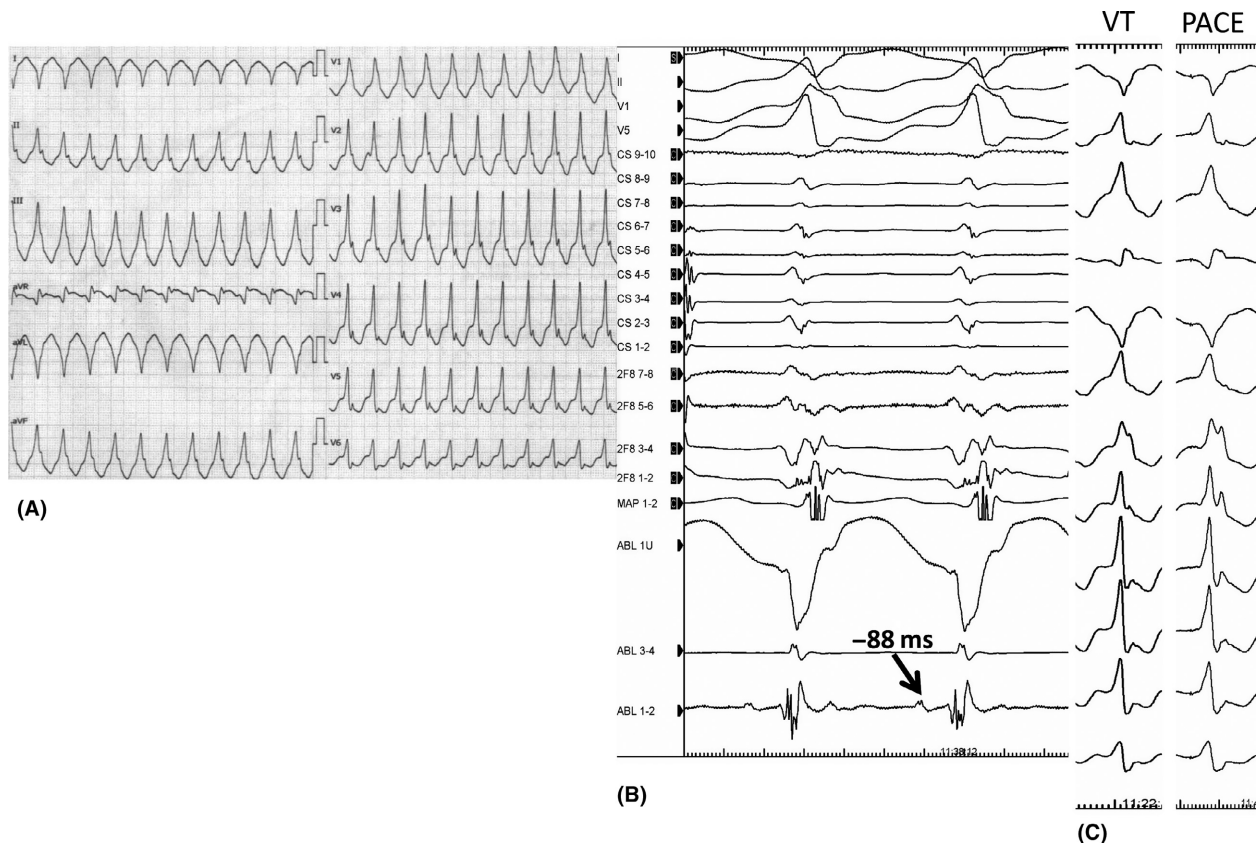


Figure 1. (A) Twelve-lead electrocardiographic morphology of the ventricular tachycardia. (B) Highlight of the twelve-lead electrocardiography of the ventricular tachycardia. (C) Endocardial pace-mapping demonstrates almost a perfect pace-map.

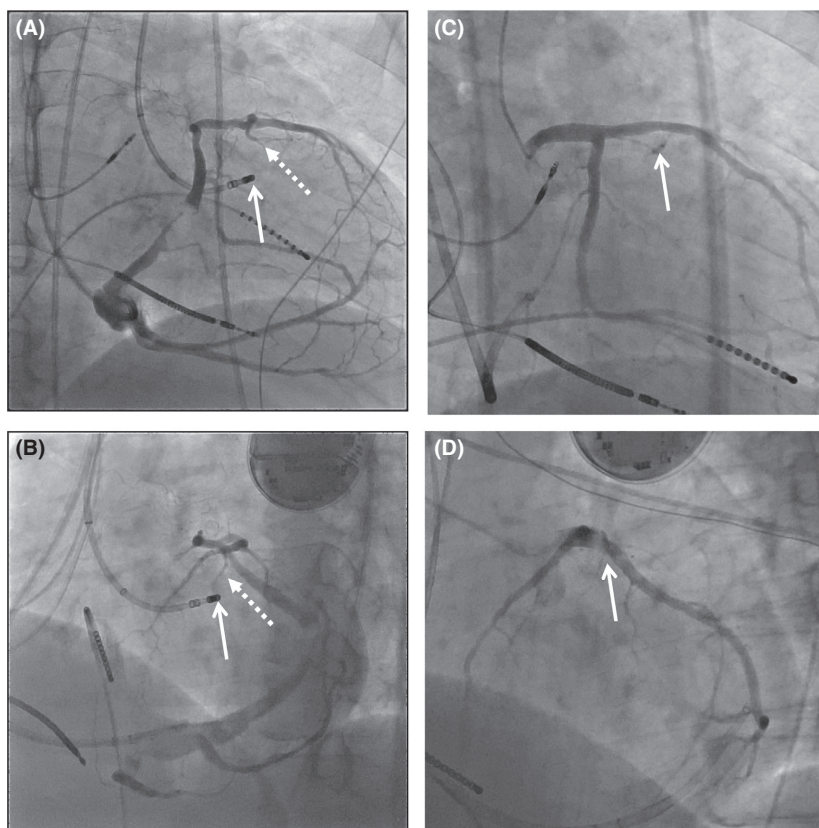


Figure 2. Fluoroscopic image of the endocardial ablation (white arrow) catheter, and coronary sinus venography showing the culprit vein (white broken arrow) (Panel A = right anterior oblique projection of 30 degree [RAO], Panel B = left anterior oblique projection of 60 degree [LAO]). Fluoroscopic image of the left coronary artery and location of the infusion catheter (white arrow). (Panel C = RAO view, Panel D = LAO view).

through the central lumen of the infusion catheter, which resulted in a transient slowing of the VT rate (Fig. 3A). Then, we also infused 3 mg of diluted procainamide, which resulted in a significant slowing of the VT rate (Fig. 3B). We considered that the coronary venous branch into which the infusion catheter was selectively inserted was the appropriate vessel for administering ethanol for the elimination of the arrhythmogenic focus of this VT. We infused 1 cc of 98% ethanol over 90 sec and repeated the ethanol infusion three times into the same branch. The VT suddenly terminated within 5 sec after the onset of the ethanol infusion (Fig. 3C). We tried to induce the VT with an aggressive stimulation protocol including triple ventricular premature contractions (VPCs) under intravenous isoproterenol; however, the VT could not be provoked. We performed voltage mapping using a CARTO system, and a significant low-voltage area could be constructed (Fig. 4). Ten days after the ablation procedure, we performed programmed ventricular stimulation for an induction test of this VT using the ICD. No sustained VT could be induced even with triple VPCs under intravenous isoproterenol. Although the ICD had

delivered appropriate therapies to treat VT several times per month prior to the chemical ablation, the patient has been free from any VT episodes for approximately half a year.

Discussion

Many VTs are derived from deep intramural origins that are difficult to target with contact-based instrumentation [8]. An intramural site of origin could be defined as a site of the earliest activation without a matching pace-map. In addition, when an intramural site of origin is located close to an adjacent anatomic area of the great cardiac vein, distinguishing an intramural site of origin from an epicardial or endocardial site of origin may be difficult [9]. In the present study, the VT was transiently eliminated with an endocardial approach despite very early activation being recorded from the tip of the ablation catheter and a very high pace-map score.

VT originating from the LV summit remains challenging because of the combination of an intramural origin,

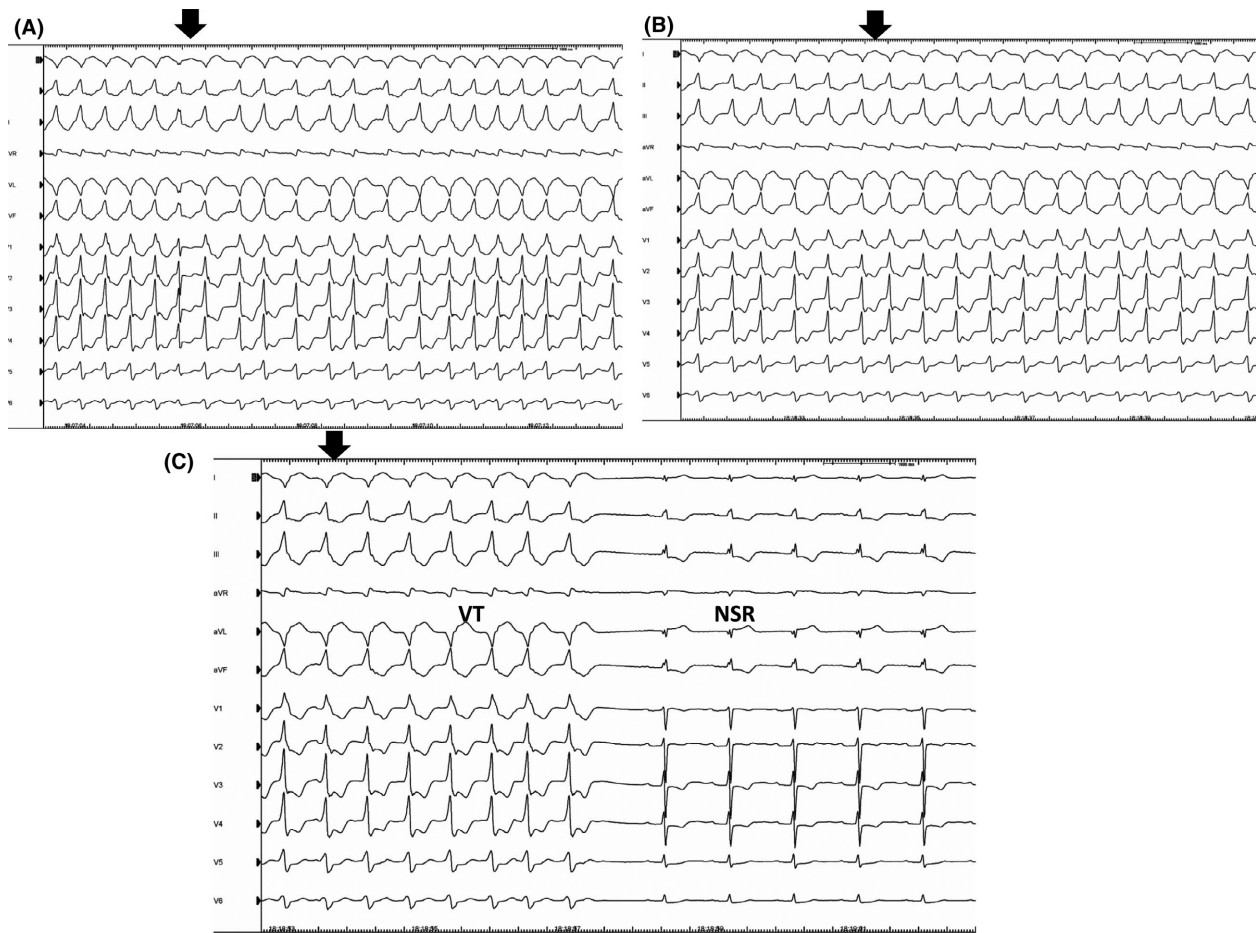


Figure 3. (A) Saline was injected (black arrow) in a bolus fashion through the central lumen of the infusion catheter during sustained VT and resulted in a transient prolongation of the VT cycle length. (B) Diluted mexiletine (5 mg) was injected (black arrow) over 10 sec during sustained VT and resulted in a significant slowing of the VT rate. (C) Ethanol (98%) was slowly infused (black arrow) during the sustained VT, and the VT terminated and normal sinus rhythm resumed.

inaccessibility to an epicardial approach, and the close proximity to coronary arteries [2, 10]. In the present case, we chose chemical ablation instead of epicardial ablation after the failure of the endocardial ablation due to the risk of damaging the coronary artery.

A retrograde venous infusion of ethanol has been described as an alternative to an arterial approach. An experimental study demonstrated promising results for circumventing coronary arterial damage and myocardial infarctions [11]. Coronary venous mapping and ethanol ablation seem uniquely suited for VTs arising from the LV summit. In addition, the off-target myocardial damage created by ethanol backflow or leak is not a concern during RCVEA because the occlusive balloon, slow infusion of the ethanol, and retrograde direction of the ethanol infusion prevent such injurious effects from the RCVEA. Yokokawa et al. demonstrated the feasibility of venous

mapping and predicted that ablation using the septal perforator veins of the coronary sinus could be promising for VTs arising from intramural sites of origin [9]. The long-term outcomes of this procedure are still not well known; however, repeated ethanol injections are able to reduce the recurrence rate [7]. In the present case, we performed a total of four ethanol injections for a total ethanol dose of 4 cc. That dose of ethanol might be sufficient to destroy the entire myocardial arrhythmogenic tissue.

Conclusion

A retrograde ethanol infusion into the coronary vein is safe, feasible, and successful in eliminating VTs arising from the LV summit. Chemical mapping is also helpful to identify the culprit vein associated with the VT origin. Chemical ablation through the coronary veins could be

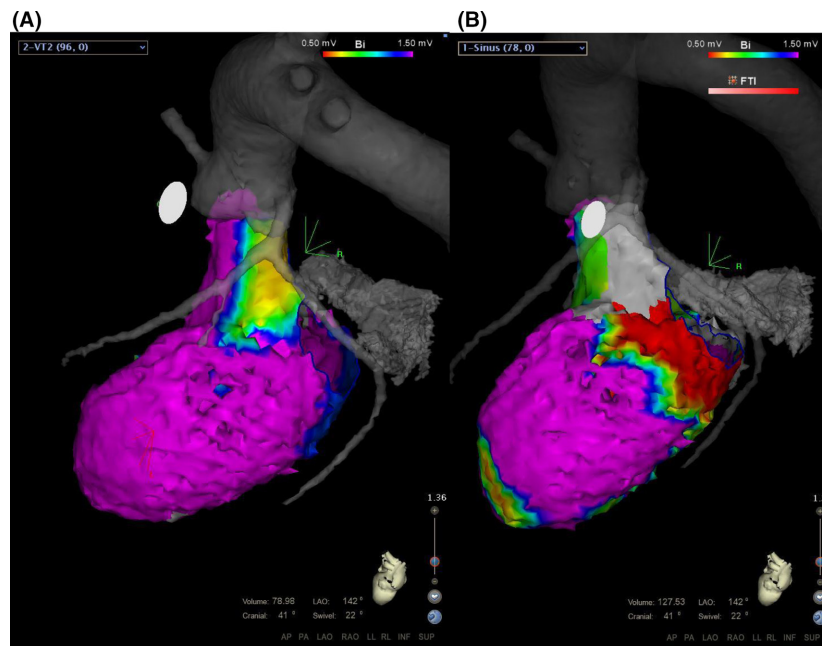


Figure 4. Electroanatomical mapping using a CARTO system. A significant low-voltage area (Panel B) compared to the control (Panel A) was constructed after the ethanol infusion.

regarded as a last resort treatment when a conventional approach of ablation fails.

Authorship

KO: Corresponding author. RN: Electrophysiologist performing present ablation case together. YY: Electrophysiologist manipulating 3D mapping system during ablation procedure. TK: Electrophysiologist helping to make figure of 3D mapping. TS: Electrophysiologist helping to make electrophysiological traces. KH: Department head.

Conflict of Interest

None declared.

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