New implications for the recurrence mechanism of an upper septal ventricular tachycardia: a case report

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Background	Verapamil-sensitive fascicular ventricular tachycardia (VT) is the most common type of idiopathic left ventricular tachycardia, and it is divided into three types. Upper septal ventricular tachycardia (US-VT) is likely in patients with prior episodes of left posterior fascicular (LPF)-VT ablation, however, little is known about the recurrence mechanism of US-VT.
Case summary	A 53-year-old man had an US-VT after two catheter ablation sessions for a common idiopathic LPF-VT. The US-VT was successfully treated by ablating the proximal site of the LPF without making any further branch or fascicular block. This successful ablation point corresponded completely with the earliest pre-systolic potential (P2) site of the LPF-VT during the 1st session of catheter ablation.
Discussion	An US-VT recurrence could occur if a critical slow conduction is not affected by the catheter ablation. This recurrence might be the result of changing the re-entrant circuit after damage to the LPF. In order to eliminate the LPF-VT and prevent an US-VT recurrence, the earliest P2 site should be investigated carefully and ablated sufficiently.
Keywords	Idiopathic left ventricular tachycardia • Verapamil-sensitive • Upper septal ventricular tachycardia • Recurrence • Case report

Learning points

- Ablation at a more distal site of the left posterior fascicular (LPF) than the earliest P2 site can eliminate the LPF-ventricular tachycardia (VT). However, there remains the possibility of an upper septal-VT recurrence.
- In patients without a recorded P1 potential, the earliest P2 recording site should be targeted and might better be ablated sufficiently.
- The His-Ventricular interval and QRS duration during VT might help identify the successful ablation site.

Introduction

Verapamil-sensitive fascicular ventricular tachycardia (VT) is the most common type of idiopathic left ventricular tachycardia (ILVT), and it is divided into three subtypes: left posterior fascicular (LPF)-VT, left anterior fascicular-VT, and upper septal fascicular (US)-VT.[1] LPF-VT, which is the most common type of ILVT, has been studied extensively, and the involvement of the LPF in the circuit of the VT has been reported.[2] However, US-VT has not been studied sufficiently because of its rare frequency. Although US-VT is likely in patients with a prior LPF-VT ablation, its mechanism has not been elucidated.[3]

We present an interesting case of a fascicular ILVT with a transformation from an LPF-VT to an US-VT, whose electrophysiological (EP) features and successful ablation site have the potential of a breakthrough in understanding the mechanism of the US-VT recurrence.

Timeline

Four months before ablation	The patient was transferred to the hospital for the initial episode of left posterior fascicular ventricular tachycardia.
First day	The patient underwent the 1st session of catheter ablation.
A month later	The same ventricular tachycardia recurred.
Four months later	The patient underwent a 2nd session of catheter ablation.
Six months later	An upper septal ventricular tachycardia was documented when the patient presented with palpitations.
A year later	The patient underwent a 3rd session of catheter ablation.
Three years later	Ventricular tachycardia never recurred after the 3rd session of catheter ablation.

Case presentation

A 53-year-old man presented with a recurrent tachycardia and was referred for catheter ablation. The patient suffered from frequent palpitations, and the tachycardia was haemodynamically stable. A 12-lead electrocardiogram (ECG) during sinus rhythm (SR) exhibited a slight right axis deviation (*Figure 1A*), and the ECG during the tachycardia exhibited a right bundle branch block (RBBB) configuration, north-west axis deviation, QRS duration of 110 ms, and cycle length (CL) of 274 ms (*Figure 1B:* VT1), and it was terminated by an infusion of low-dose verapamil. No specific abnormalities were identified on echocardiography, thallium myocardial scintigraphy, or cardiac magnetic resonance imaging. The patient had an episode of palpitations a half a year prior. The frequency of the palpitations gradually increased as time passed, and they began to occur about once a week. Taking verapamil and flecainide could not completely suppress the tachycardia. Since the patient had a symptomatic recurrent idiopathic left VT, catheter ablation therapy was recommended as a class I indication.[4]

An EP study was performed. A CARTO3 three-dimensional mapping system (Biosense Webster, Diamond Bar, CA, USA) was used with intracardiac echocardiography (SoundStar catheter, Biosense Webster). A 5-Fr decapolar steerable electrode catheter with 2 mm interelectrode spacing (Abbott Laboratories, Chicago, IL, USA) was positioned on the left ventricular (LV) septum through a retrograde

transaortic approach. A 5-Fr quadripolar electrode catheter (Japan Lifeline, Tokyo, Japan) was positioned in the high right atrium region, 5-Fr decapolar electrode catheter (Japan Lifeline, Tokyo, Japan) in the coronary sinus (CS), and 5-Fr 12-pole electrode catheter (Japan Lifeline, Tokyo, Japan) at the His and right ventricular (RV) apex regions. VT1 was induced by both atrial and ventricular pacing. During VT1, a multipolar electrode catheter (MEC) placed on the LV septum showed a pre-systolic potential (P2) and LV myocardial potentials (*Figure 2A and B*). However, no diastolic potential (P1) was recorded. An excellent pace map was obtained from the mid to distal parts of the MEC on the LV septum. Radio frequency (RF) energy (with a power setting of 30–35 W and 60–120 s duration) using an irrigated catheter (SurroundFlow, Biosense Webster) was applied to the distal area of the LPF during SR, and VT1 became non-inducible. The patient was followed up with pill in the pocket therapy using verapamil after the ablation.

However, the same tachycardia still occurred at the same frequency as that after the 1st session, and a 2nd EP study was performed. A CARTO3 system was used. During the second session, RF energy (with a power of 30–35 W and 60–120 s duration) using an irrigated catheter (SurroundFlow, Biosense Webster) was delivered to the mid-LPF region during SR, and VT1 became no longer inducible. After the 2nd session, an ECG during SR revealed an LPF hemiblock, which was not evident after the 1st session (*Figure 1C*). The patient was followed up with pill in the pocket therapy using verapamil after the ablation.

However, after the 2nd session, the patient still suffered from palpitations with the same frequency as that before the ablation and they started 1 month after the 2nd ablation session. Six months after the 2nd ablation session, a 12-lead ECG revealing a different VT (*Figure 1D:* VT2) was documented. VT2 had an RBBB configuration, right axis deviation, QRS duration of 112 ms, and CL of 286 ms, which had a quite different axis from VT1. A 3rd EP study was performed using a CARTO3 system with a SoundStar intracardiac echocardiography catheter. A 5-Fr decapolar steerable electrode catheter with 2 mm interelectrode spacing (Abbott Laboratories, Chicago, IL, USA) was positioned on the LV septum through an antegrade transseptal approach. A 5-Fr quadripolar electrode catheter (Japan Lifeline, Tokyo, Japan) was positioned in the RV apex region, 5-Fr decapolar electrode catheter (Japan Lifeline, Tokyo, Japan) in the CS, and 5-Fr hexapolar electrode catheter in the His region. In addition, an 8-Fr non-irrigated ablation catheter (Japan Lifeline, Tokyo, Japan) was positioned on the non-coronary cusp. VT2 spontaneously occurred after an isoproterenol infusion. There was a slight variation in the VT2 CL, and the preceding H-H had the same interval as the V-V (*Figure 3A*). Entrainment pacing from the RV apex exhibited constant fusion, and the post pacing interval (PPI) (337 ms) was longer than the VT2 CL (294 ms). Due to the RF energy applications in the 1st and 2nd sessions, entrainment pacing from the mid to distal LPF area did not capture the fascicle or ventricle. The His-Ventricular (H-V) interval (20 ms) during VT2 was long and was similar to that of VT1. From those EP findings and the QRS morphology, we estimated that VT2 was an US-VT, and a retrograde slowly conducting limb should possibly have been connected to the proximal site of the LPF. Due to the RF energy applications in the 1st and 2nd sessions, the LPF potentials were only detected in a very limited area of a proximal site. By
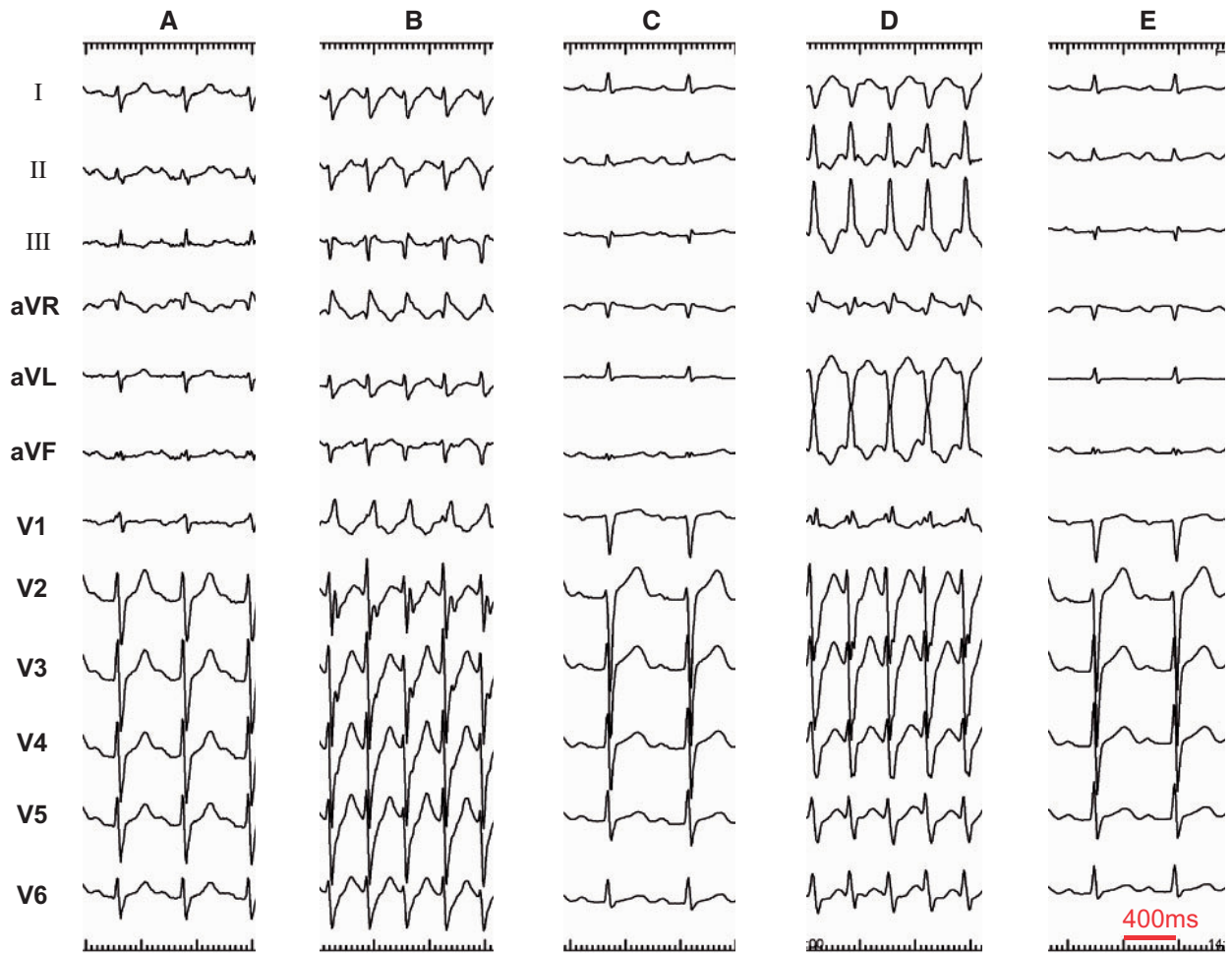
Figure 1 Twelve-lead electrocardiograms during sinus rhythm and the ventricular tachycardia. (*A*) Sinus rhythm before ablation. (*B*) VT1. (*C*) Sinus rhythm after the 2nd session. (*D*) VT2. (*E*) Sinus rhythm after the 3rd session.

placing the ablation catheter at that point, VT2 transitioned to a pleomorphic fusion beat, and 30 W of RF energy using an irrigated catheter (SurroundFlow, Biosense Webster) terminated the VT within a few seconds. The successful ablation site corresponded completely with the earliest P2 site during VT1 in the 1st session (*Figure 3B*). VT2 became no longer inducible. The 12-lead ECG and H-V interval (60 ms) did not change after the ablation (*Figure 1E*). After the ablation, the patient was followed up without any antiarrhythmic drugs, and the VT has never recurred during 21 months of follow-up.

Discussion

This case exhibited an US-VT recurrence after ablation of an LPF-VT. In this case, the successful ablation site of the US-VT corresponded completely with the earliest P2 site of the LPF-VT, therefore, these two tachycardias were thought to use the same critical slow conduction pathway as their retrograde conducting limb. It has already been reported that US-VTs are likely to occur in patients with a prior LPF-

VT ablation, however, their mechanism has not been elucidated. To the best of our knowledge, this is the first report that showed that an US-VT recurrence occurred using the same critical slow conduction pathway as the LPF-VT.

Talib *et al.*[3] reported the prevalence, and surface electrocardiographic and EP characteristics of verapamil-sensitive US-VT. They reported that US-VT has several characteristic findings. First, the His potential precedes the ventricular potential during the VT and the H-V interval is always shorter during the VT than SR. Second, the baseline ECG exhibits SR with a QRS duration of <120 ms, and the presence of a Q-wave in the inferior leads and S-wave in lead I or aVL. Third, the QRS duration of the VT is slightly wider than that during SR. Fourth, the QRS morphology exhibits a right axis or north-west axis deviation and/or RBBB. VT2 in our case met all the listed conditions, and this led to the fact that VT2 was undoubtedly an US-VT. Their report also showed that six patients (50%) had a previous history of one or two radio frequency catheter ablation (RFCA) sessions for common ILVT, and that was also applicable in our case.
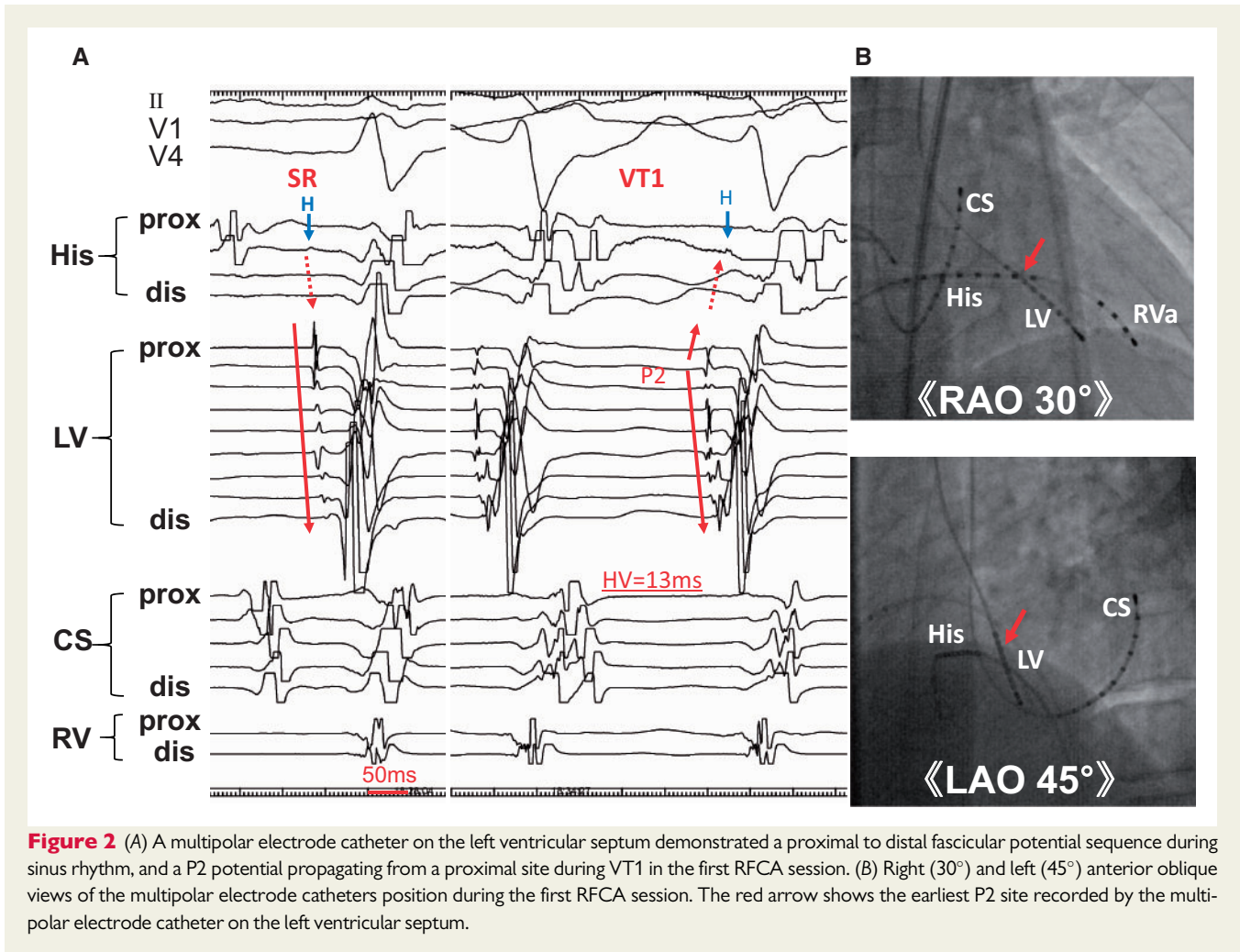
Figure 2 (A) A multipolar electrode catheter on the left ventricular septum demonstrated a proximal to distal fascicular potential sequence during sinus rhythm, and a P2 potential propagating from a proximal site during VT1 in the first RFCA session. (B) Right (30°) and left (45°) anterior oblique views of the multipolar electrode catheters position during the first RFCA session. The red arrow shows the earliest P2 site recorded by the multipolar electrode catheter on the left ventricular septum.

Recently, Liu *et al.*[2] reported that the macro-re-entrant loop of the LPF-VT involved the ventricular myocardium, part of the LPF, slow conduction zone, and P1 fibre (in cases in which a P1 was recorded). They showed the sequence of the MEC placed on the LPF and reported that the H-V interval during the LPF-VT correlated well with the location of the earliest P2, which was considered to be the site of the connection between P1 and P2. They also had successful ablation results when targeting the earliest P2 sites in patients without a P1 potential being recorded. In our case, no P1 was recorded, and the site of the connection between P1 and P2 was considered to be proximal, as judged by the earliest P2 site and the long H-V interval (13 ms) during the LPF-VT (*Figure 2A* and *B*). In our 1st and 2nd sessions, RF energy was applied to the distal and mid area of the LPF, and VT1 became non-inducible; however, VT2 occurred soon after the 2nd session. VT2 was successfully ablated during the 3rd session, and the successful ablation point of VT2 was in the same area as the earliest P2 site of VT1 during the first session (*Figures 2B* and *3D*). This finding indicated that there was only one connection between the slow conduction and LPF in this patient, and it was due to the development of an LPF disturbance that changed the VT morphology from VT1 to VT2.

Considering the mechanism of the LPF-VT, there were two possible mechanisms of the US-VT recurrence. One of them was that the RF energy was applied to a more distal LPF site than the connection between P1 and P2, as was shown in our case (*Figure 4A*). The other one was that there were several connections between P1 and P2, and the RF energy was applied only to the distal connection (*Figure 4B*). That could be distinguished by the sequence of the MEC placed on the LPF during the LPF-VT.

As our case showed, RFCA at a more distal site than the earliest P2 can eliminate an LPF-VT but can cause an US-VT. In patients with a P1 potential recorded, the optimal target is undoubtedly the P1 itself, or the connection site between P1 and P2, whereas in patients without a P1 potential being recorded, the optimal target should be the earliest P2 site. Further studies are needed to identify the true mechanism of the US-VT recurrence.

Conclusion

In order to eliminate the LPF-VT and prevent an US-VT recurrence, the earliest P2 site might better be targeted and ablated sufficiently.
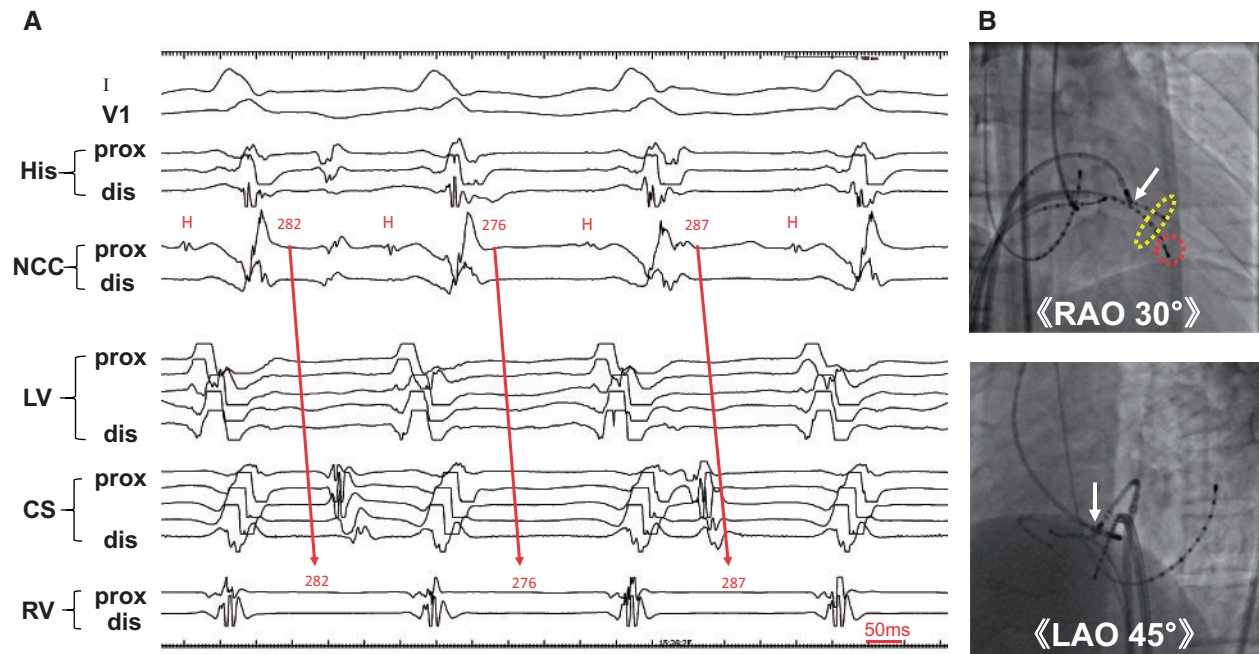
Figure 3 (A) Intracardiac recording of VT2. The preceding H-H correlated well with the V-V interval. No left posterior fascicular potentials were detected on the multipolar electrode catheter placed on the left ventricular septum. (B) Right (30°) and left (45°) anterior oblique views of the successful ablation site during the 3rd RFCA session. The red and yellow circles show the ablation site of the 1st and 2nd sessions, respectively.
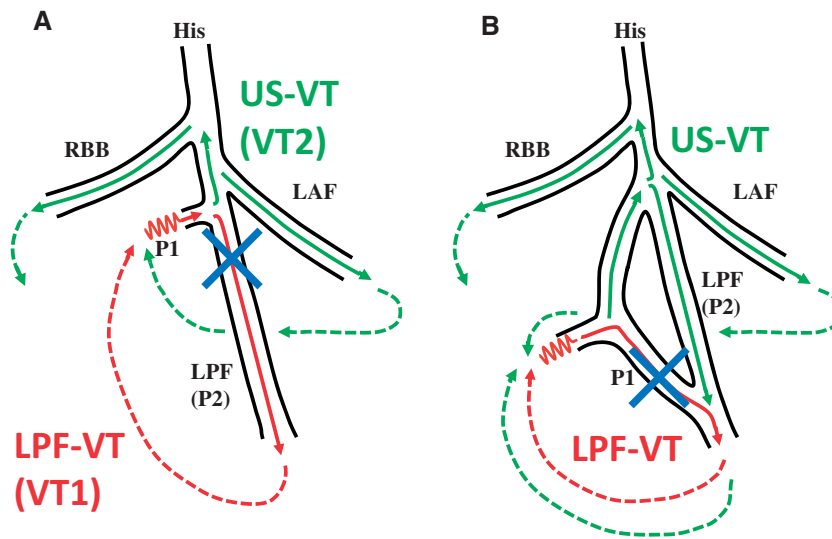

Figure 4 (A) Schematic representation of the re-entrant circuits of VT1 and VT2. P1 connects to the proximal part of the left posterior fascicular. The cross-mark shows the ablation site in the 2nd session. (B) Schematic representation of the possible mechanism of the upper septal-ventricular tachycardia recurrence in the case of having several connections between P1 and P2. Radio frequency energy was applied only to the distal connection.

Lead author biography

Junji Yamaguchi, graduated from Tokyo Medical and Dental University School of Medicine in 2012. Junior Resident in Internal Medicine, Toshiba General Hospital from 2012 to 2013. Junior Resident in Internal Medicine, Tokyo Medical and Dental University Hospital from 2013 to 2014. Senior Resident in Cardiology, Japanese Red Cross Musashino Hospital in 2014. EP doctor, Japanese Red Cross Musashino Hospital in 2017.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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