Successful chemical ablation of refractory ventricular tachycardia from the left ventricular summit using the double balloon technique with chronic total occlusion percutaneous coronary intervention techniques

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Introduction

Endocardial radiofrequency catheter ablation (RFA) for ventricular arrhythmia (VA) is an established procedure with variable success rates. Compared to antiarrhythmic drugs such as amiodarone, sotalol, and β-blockers, endocardial substrate–based RFA reportedly improves survival rates in patients with ischemic cardiomyopathy.[1] However, endocardial RFA may sometimes be unsuccessful, since 5%−15 % of lesions are located in the intramural or subepicardial region, which may require interventions outside of conventional approaches.[2] The success rate of ventricular tachycardia (VT) ablation also depends on the anatomical site. Endocardial and epicardial RFA for VAs arising from the left ventricular (LV) summit is challenging owing to anatomical barriers such as the left coronary arteries and epicardial adipose tissue.[3] Herein, we present a case of VT arising from the LV summit that was refractory to the conventional endocardial RFA and successfully treated with adjunctive chemical ablation using techniques for chronic total occlusion (CTO) percutaneous coronary intervention (PCI).

KEY TEACHING POINTS

- Endocardial and epicardial radiofrequency catheter ablation (RFA) for ventricular arrhythmias (VAs) from the left ventricular (LV) summit is challenging owing to anatomical barriers such as the left coronary arteries and epicardial adipose tissue.
- Retrograde coronary venous ethanol ablation (RCVEA) is useful for ventricular tachycardia (VT) substrates of intramural origin or substrates proximal to the coronary arteries, and especially effective for cases of failed endocardial or epicardial RFA for VT arising from the LV summit. However, RCVEA is not considered useful when there is no suitable vein with direct connection to capillaries or in the presence of collateral veins.
- RCVEA using a double balloon, combined with chronic total occlusion percutaneous coronary intervention techniques such as retrograde wiring and retrograde wire externalization, may be a novel and less invasive treatment for VT arising from the LV summit even in the absence of suitable veins. This method may be useful as an alternative strategy for refractory VAs and has the potential to replace the epicardial approach.

Case report

A 78-year-old man with a history of ischemic heart disease, reduced LV ejection fraction (LVEF), paroxysmal atrial fibrillation (PAF), VT, and pulmonary emphysema was admitted to our hospital with acute decompensated heart failure. The patient had undergone RFA for paroxysmal atrial fibrillation 3 years prior and endocardial RFA for VT 4 and 2 years prior at another hospital, which failed to eliminate the VT. Implantable cardioverter-defibrillator implantation had also been performed 4 years prior. Intravenous inotropic agents, intravenous vasodilators, and diuretics helped improve the pulmonary congestion; however, hemodynamically stable VT was still frequently observed after treatment for heart failure.

A 24-hour Holter monitoring system revealed a 30% premature ventricular contraction (PVC) burden, which was suspected to cause the observed LV dysfunction; thus, RFA was

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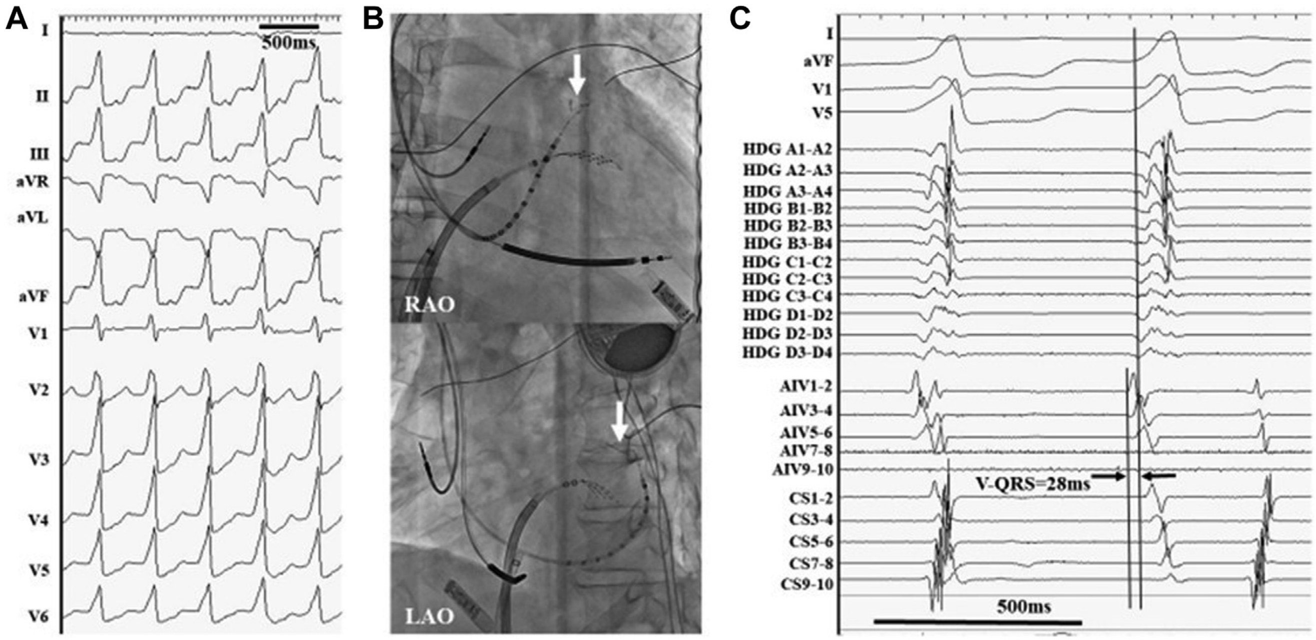

Figure 1 **A:** Twelve-lead electrocardiographic morphology showing ventricular tachycardia (VT). **B:** A decapolar catheter was advanced to the anterior interventricular vein (*arrow*). **C:** The earliest ventricular activation during the VT preceded the QRS onset by 28 ms at the distal tip of the decapolar catheter.

performed. This procedure was performed after informed consent was obtained for all procedures, including chemical ablation using ethanol. All procedures were approved by the institutional review board.

Electroanatomical mapping was performed using a 3-dimensional mapping system (EnSite System; St. Jude Medical, Inc, St Paul, MN). VT was frequently observed at the start of the examination. Additionally, a 12-lead electrocardiogram exhibited VT with a right bundle branch block and inferior axis morphology, QS pattern in lead I, a maximum deflection index of 0.71, intrinsicoid deflection time of 102 ms, shortest RS complex of 118 ms, and pseudo-delta wave of 44 ms (Figure 1A).[4,5] A 2.7F over-the-wire (OTW)-type decapolar catheter (EPstar FIX AIV; Japan Lifeline, Tokyo, Japan) was advanced into the anterior interventricular vein (AIV) via the great cardiac vein (GCV) through the superior vena cava (Figure 1B). The earliest ventricular activation during VT preceded the QRS onset by 28 ms at the distal tip of the decapolar catheter (Figure 1C). The tip of the ablation catheter was advanced to the basal anterior segment of the left ventricle near the decapolar catheter (Figure 2A).

Activation mapping using the HD Grid™ catheter (Abbott Medical, Abbott Park, IL) demonstrated a centrifugal activation pattern, with the earliest activation close to the earliest decapolar catheter (Figure 2B). The endocardial potentials recorded close to the earliest decapolar catheter preceded the QRS onset by 34 ms and also showed QS morphology in the unipolar electrogram (Figure 2C). We then delivered radiofrequency energy to the earliest site by using an irrigation catheter with a contact force sensor (TactiCath; St. Jude Medical, Inc), which could transiently terminate the sustained VT, albeit the VT could not be completely eliminated (Figure 2D). Ice-cold saline was then infused through the infusion lumen of the decapolar catheter to decrease the frequency of PVC (Figure 2E), which suggested that the critical substrates of the VT were located close to the perfusion area.

Preprocedural computed tomography (CT) demonstrated marked gastric dilatation, which was associated with the risk of stomach injury during pericardiocentesis; therefore, epicardial ablation was not performed. Based on this, chemical ablation for the refractory VT was performed. Retrograde coronary venography of the GCV showed collateral flow to the AIV overlying the LV summit (Figure 3A and 3B). A 6F internal mammary artery (IMA) guiding catheter (GC) (Mach1; Boston Scientific Corporation, Marlborough, MA) was inserted into the coronary sinus via the right internal jugular vein. Although a Cruise guidewire (Asahi Intec, Tokyo, Japan) guided through the 6F IMA GC could not be passed through the collateral vessel from the GCV in an antegrade manner, the guidewire was retrogradely proceeded to the collateral vessel from the AIV and eventually into the IMA GC through the GCV (Figure 3C and 3D). Thereafter, a Mizuki microcatheter (Kaneka, Osaka, Japan) was advanced OTW in a retrograde fashion into the IMA GC. Subsequently, the Cruise guidewire was removed and exchanged for a 330 cm RG3 guidewire (Asahi Intec) to thread through the Y-connector of the IMA GC. The RG3 guidewire was manually engaged into an introducer needle antegradely inserted into the Y-connector of the hemostatic system (Figure 3E).

After the RG3 guidewire was successfully externalized, the distal tip of the retrograde microcatheter was pulled back to the AIV and another Mizuki microcatheter was advanced over the RG3 guidewire to the collateral vessel in an antegrade manner. The RG3 guidewire and retrograde

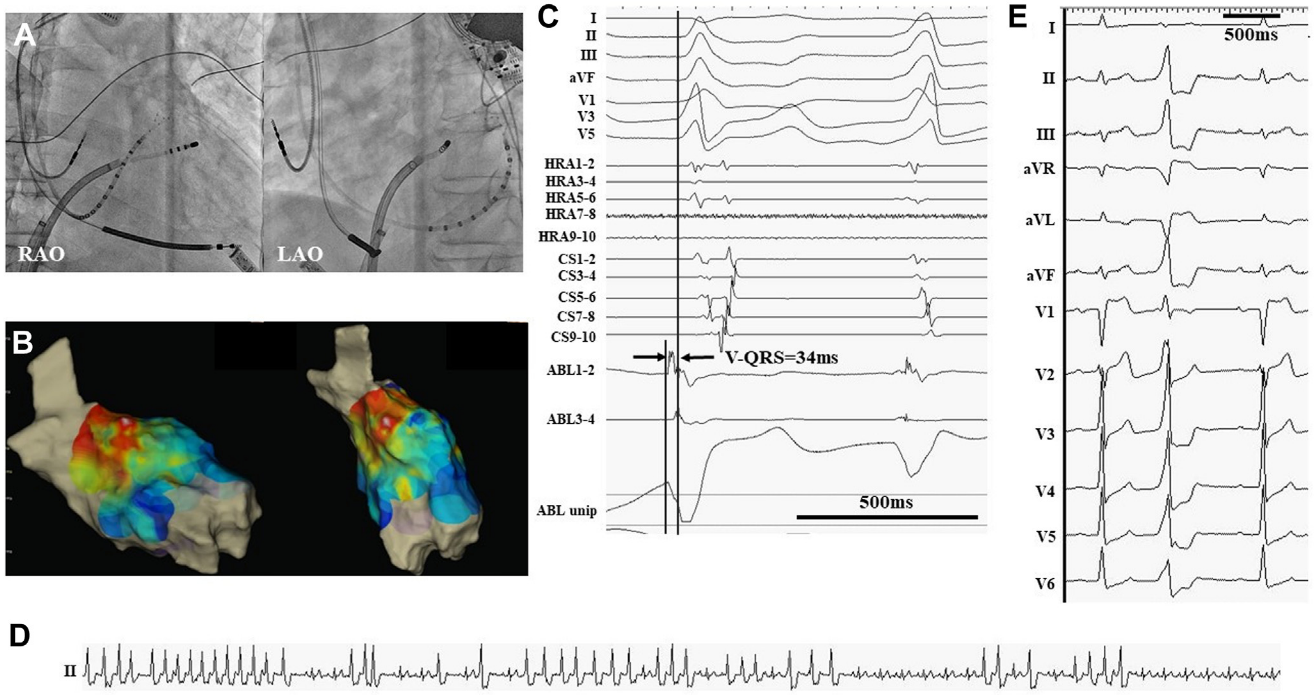

Figure 2 **A:** The tip of the ablation catheter was advanced to the basal anterior segment of the left ventricle near the decapolar catheter. **B:** Activation mapping demonstrated the centrifugal activation pattern with the earliest activation close to the earliest decapolar catheter. **C:** The endocardial potentials recorded close to the earliest decapolar catheter preceded QRS onset by 34 ms, which also showed the QS morphology in a unipolar electrogram. **D:** The radiofrequency energy was delivered to the earliest site using an irrigation catheter, which could transiently terminate the sustained ventricular tachycardia, but not eliminate it completely. **E:** Ice-cold saline infused through the infusion lumen of the decapolar catheter decreased the frequency of premature ventricular contractions.

microcatheter were removed, and a Cruise guidewire was advanced through the antegrade microcatheter from the GCV to the collateral vessel. Another Cruise guidewire was inserted through an 8.5F sheath (LAMP45; St. Jude Medical, Inc) from the femoral vein and advanced to the collateral vessel via the AIV (Figure 3F). Two 2.0 × 8.0-mm OTW balloons (Emerge OTW; Boston Scientific Corporation) were inserted into the collateral vessel via the IMA GC and an 8.5F sheath (Figure 3G). The proximal and distal portions of the collateral vessel were occluded using 2 2.0 × 8.0-mm OTW balloons inflated to 4 atm. Thereafter, 1.0 mL of contrast medium was injected into the proximal balloon lumen. After confirmation of contrast stagnation in the occluded area, 2.0 mL of 98% ethanol was injected over 360 seconds (Figure 3H). Each balloon was moved 8 mm 3 times, both distally and proximally. Ethanol was injected at each site, and target PVCs were eliminated (Figure 3I).

After the procedure, the monomorphic PVCs were resolved, no VT was initiated under intravenous isoproterenol infusion, and creatine kinase was not elevated on the following day. The patient was free from VT episodes for approximately 10 months during the follow-up period after ablation, and LVEF improvement was confirmed.

Discussion

As the substrates of VT are located in the intramural and epicardial ventricular myocardium as well as the endocardial ventricular myocardium, VT is sometimes difficult to eliminate using RFA from the endocardial side, requiring an epicardial approach.[6] A previous study on epicardial RFA for VT revealed the complete elimination of all inducible VT in 71.6% of the patients after RFA and no procedure-related deaths.[7] However, epicardial RFA is unsuitable for patients with a high body mass index; past cardiac surgery[8]; the presence of a phrenic nerve, coronary artery, or epicardial fat at the ventricular target[9]; and VAs arising from the LV summit.[10] In the present case, endocardial RFA could not eliminate monomorphic PVCs originating from the LV summit, and marked gastric dilatation was confirmed on the pre-procedural CT; therefore, we chose chemical ablation instead of epicardial RFA.

Compared with transarterial coronary ethanol ablation, retrograde coronary venous ethanol ablation (RCVEA) is a safer approach because diluted and detoxified ethanol is administered via antegrade blood flow and does not cause off-target myocardial injury owing to ethanol leakage. However, an occlusive balloon is necessary since the antegrade blood flow washes out the retrogradely infused ethanol.[11] This approach should be useful for VT substrates of intramural origin or substrates proximal to the coronary arteries. Additionally, this approach is especially effective and safe for cases of failed endocardial or epicardial RFA for VT arising from the LV summit.[11]

RCVEA with a double balloon technique allows selective ethanol injection into the intramural branches between the

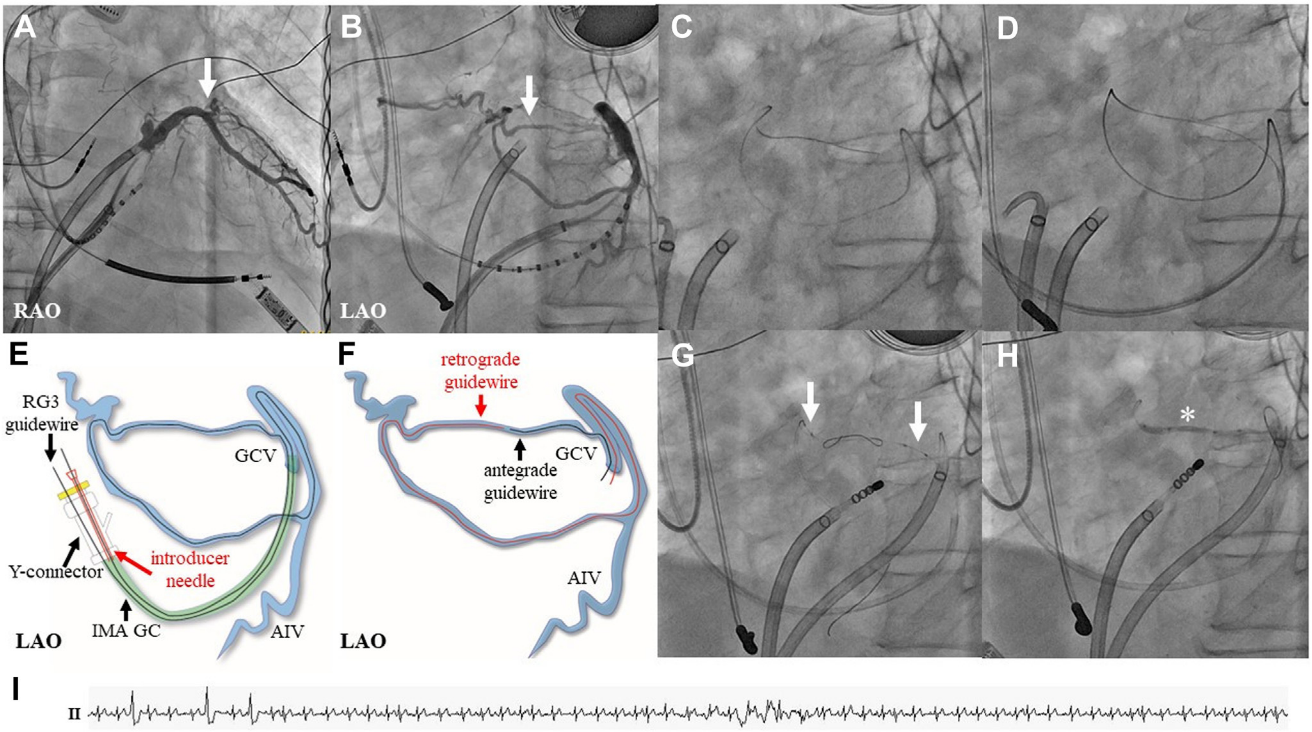

Figure 3 **A, B:** Retrograde coronary venogram of the great cardiac vein (GCV) depicted collateral flow to the anterior interventricular vein (AIV) overlying the left ventricular summit (*arrow*). **C, D:** The guidewire could proceed to the collateral vessel from the AIV and eventually into the guiding catheter (GC). A microcatheter was passed retrogradely over the guidewire and finally advanced to the GC. **E:** Schematic of the approach. The guidewire was removed and exchanged for a 330 cm RG3 guidewire to thread through the Y-connector of the internal mammary artery (IMA) GC. The RG3 guidewire was manually engaged into an introducer needle antegradely inserted into the Y-connector of the hemostatic system. **F:** Schematic of the approach. Another guidewire was inserted through an 8.5F sheath from the femoral vein and advanced to the collateral vessel via the AIV. **G:** Two over-the-wire (OTW) balloons were inserted into the collateral vessel via the GC and 8.5F sheath (*arrow*). **H:** The proximal and distal portions of the collateral vessel were occluded using OTW balloons inflated at 4 atm. Contrast medium (1.0 mL) injected into the proximal balloon demonstrated contrast stagnation in the occluded area. Subsequently, 2.0 mL of 98% ethanol was injected over 360 seconds (*asterisk*). Three ethanol injections at different balloon positions from distal to proximal were performed. **I:** After the third chemical ablation, the target premature ventricular contractions were eliminated.

balloons by occluding the coronary vein with 2 balloons, since there is no other suitable vein. This also allows for collateral vessel occlusion with a second balloon to avoid ethanol leakage in the presence of collateral veins.[12] Shortening the distance between the ethanol-infused balloon and the flow-blocking balloon was more effective in allowing ethanol to reach the myocardium, and simultaneously moving the 2 balloons enabled extensive ethanol infusion. In this case, no appropriate veins were directly connected to the capillaries, and the guidewire manipulation proceeded via the collateral into the GCV, requiring advanced skill; we thus eliminated the VT with no procedural or postprocedural complications and confirmed improvement in LVEF.

Retrograde wiring and retrograde wire externalization are frequently performed in CTO-PCI. Wire externalization refers to advancing the retrograde guidewire into and through the antegrade GC to serve as an antegrade route for balloons and stents.[13] In this case, the guidewire could not pass through the collateral vessel from the GCV via antegrade wire manipulation. However, retrograde wire manipulation enabled it to proceed to the collateral vessel from the AIV, and the OTW balloon was antegradely advanced after retrograde wire externalization. If the guidewire cannot be retrogradely advanced into the IMA GC, retrograde guidewire trapping with the snare device of an 8.5F sheath can be considered.[14]

One limitation of this study is that we did not perform pace mapping to confirm the culprit lesion. The output of pacing affects the amount of myocardium captured by pacing stimuli, and increasing the output may reduce the accuracy associated with the paced and captured locations. In this case, the substrates of the VT were suspected to be located in the intramural and epicardial ventricular myocardium as well as the endocardial LV, which might have led to lower pace mapping correlation compared with endocardial substrates; therefore, we did not perform pace mapping. RCVEA using a double balloon combined with CTO-PCI techniques may be a novel, less-invasive alternative treatment for VT arising from the LV summit when RCVEA is deemed difficult to perform owing to the absence of suitable veins. This method may be useful as an alternative strategy for refractory VAs and has the potential to replace the epicardial approach.

Conclusion

VT arising from the LV summit is refractory to conventional endocardial RFA, and the substrates of such VT are suspected to be located in the intramural and epicardial LV as well as the endocardial LV. RCVEA with a double balloon technique combined with CTO-PCI techniques has the potential to replace the epicardial approach as treatment for refractory VAs.

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References

1. Arenal Á, Ávila P, Jiménez-Candil J, et al. Substrate ablation vs antiarrhythmic drug therapy for symptomatic ventricular tachycardia. J Am Coll Cardiol 2022; 79:1441–1453.
2. Sacher F, Sobieszczyk P, Tedrow U, et al. Transcoronary ethanol ventricular tachycardia ablation in the modern electrophysiology era. Heart Rhythm 2008;5:62–68.
3. Yamada T, Doppalapudi H, Litovsky SH, McElderry HT, Kay GN. Challenging radiofrequency catheter ablation of idiopathic ventricular arrhythmias originating from the left ventricular summit near the left main coronary artery. Circ Arrhythm Electrophysiol 2016;9:e004202.
4. Daniels DV, Lu YY, Morton JB, et al. Idiopathic epicardial left ventricular tachycardia originating remote from the sinus of valsalva: electrophysiological characteristics, catheter ablation, and identification from the 12-lead electrocardiogram. Circulation 2006;113:1659–1666.
5. Berruezo A, Mont L, Nava S, et al. Electrocardiographic recognition of the epicardial origin of ventricular tachycardias. Circulation 2004; 109:1842–1847.
6. Yamada T, Maddox WR, McElderry HT, et al. Radiofrequency catheter ablation of idiopathic ventricular arrhythmias originating from intramural foci in the left ventricular outflow tract. Circ Arrhythm Electrophysiol 2015; 8:344–352.
7. Bella PD, Brugada J, Zeppenfeld K, et al. Epicardial ablation for ventricular tachycardia. Circ Arrhythm Electrophysiol 2011;4:653–659.
8. Tarantino N, Della Rocca DG, Faggioni M, et al. Epicardial ablation complications. Card Electrophysiol Clin 2020;12:409–418.
9. Edward JA, Nguyen DT. Patient selection for epicardial ablation—Part II: The epicardial approach and current challenges associated with epicardial ablation. J Innov Cardiac Rhythm Manage 2019;10:3906–3912.
10. Santangeli P, Marchlinski FE, Zado ES, et al. Percutaneous epicardial ablation of ventricular arrhythmias arising from the left ventricular summit. Circ Arrhythm Electrophysiol 2015;8:337–343.
11. Kreidieh B, Rodríguez-Mañero M, Schurmann P, et al. Retrograde coronary venous ethanol infusion for ablation of refractory ventricular tachycardia. Circ Arrhythm Electrophysiol 2016;9:e004352.
12. Da-Wariboko A, Lador A, Tavares L, et al. Double-balloon technique for retrograde venous ethanol ablation of ventricular arrhythmias in the absence of suitable intramural veins. Heart Rhythm 2020;17:2126–2134.
13. Mashayekhi K, Valuckiene Z, Neuser H, et al. Wire externalisation techniques for retrograde percutaneous coronary interventions of chronic total occlusions. EuroIntervention 2017;13:e1489–e1490.
14. Fang HY, Lee WC, Fang CY, Wu CJ. Application of a snare technique in retrograde chronic total occlusion percutaneous coronary intervention – a step by step practical approach and an observational study. Medicine (Baltimore) 2016;95:41.