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CASE REPORT
Arrhythmias / Electrophysiology

A case of successful ablation of ventricular tachycardia focus in the left ventricular summit through the left atrial appendage: a case report

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Background Although premature ventricular complexes and ventricular tachycardia (VT) from outflow tracts are easy to map and ablate, some foci create the greatest challenges for the electrophysiologist. One such example is the 'Bermuda triangle' of the heart.

Case summary In this article, we describe the rarely used but acceptable approach to the 'Bermudian' focus. We present a case of a 38-year-old male patient with sustained monomorphic VT, who underwent radiofrequency ablation of arrhythmogenic myocardium. After unsuccessful ablation through the posterior right ventricular outflow tract (RVOT), left coronary cusp (LCC), and distal coronary sinus, tachycardia was eliminated from the left atrial appendage (LAA). Complaints such as palpitations and weakness disappeared after the procedure.

Discussion Radiofrequency ablation of VT might be performed using LAA. This approach is used when the epicardial location of arrhythmia-causing tissue is suspected and ablation through the RVOT, LCC, and great cardiac vein fails.

Keywords Idiopathic ventricular tachycardia • Ventricular outflow tract • Coronary sinus • Left atrial appendage • Radiofrequency ablation • Transseptal puncture • Case report

Learning points

- Radiofrequency ablation of the ventricular tachycardia may be performed using the left atrial appendage (LAA). This approach is used when epicardial location of arrhythmia-causing tissue is suspected and ablation through the right ventricular outflow tract, left coronary cusp, and great cardiac vein fails.
- Catheter manipulations in the LAA might be feasible and future researches are needed.

Introduction

The 'Bermuda triangle' of the heart is the area of the myocardium situated between three neighbouring structures of the left ventricular (LV) summit: the posterior right ventricular outflow tract (RVOT), left coronary cusp (LCC), and distal coronary sinus (CS), i.e. origin of the great cardiac vein (GCV). This epicardial area is well known for its inaccessibility; the closer the focus is to the centre of the triangle, the more challenging it is to the operator to ablate or even to find the focus from any of the triangle's borders.[1]

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Timeline

Dates	Relevant past medical history and interventions
	Tachycardia first appeared after acute respiratory infection in 2012. Patient addressed no doctors. No genetic pre-disposition. No smoking and alcohol consumption. Since last montd, he has been experiencing episodes of palpitations much more frequently.

Dates	Summaries from initial and follow-up visits	Diagnostic testing	Interventions
04 September 2017	Primary visit to cardiologist. Paroxysmal ventricular tachycardia (VT) was diagnosed.	24 h electrocardiography (ECG) monitoring—sinus rhythm. In total, 23 412 single monomorphic premature ventricular complexes (PVCs), 14 episodes of non-sustained and three episode of sustained VT. Echocardiography—no structural pathology. General and biochemical blood tests—unremarkable.	Amiodoron (Cordaron) 600 mg a day.
14 September 2017	Follow-up visit. Patient reported about four episodes of sustained palpitations during last 10 days and numerous single 'wrong beats' of the heart. As pharmaceutical treatment was not efficient, patient was sent for radiofrequency ablation.	24 h ECG monitoring—sinus rhythm. In total, 15 351 single monomorphic PVCs.	No
15 September 2017	Visit to interventional cardiologist. Radiofrequency ablation was recommended.	Infections (Hepatitis B, C; AIDS) negative	Radiofrequency ablation of left ventricular summit.
16 September 2017	Post-operation follow-up	24 h ECG monitoring—sinus rhythm. In total, 1314 single monomorphic PVCs. No VT-episodes	Allapinin 25 mg a day.
20 October 2017	Post-operation follow-up (1 month)	24 h ECG monitoring—sinus rhythm. In total, 2298 single monomorphic PVCs. No VT-episodes	No

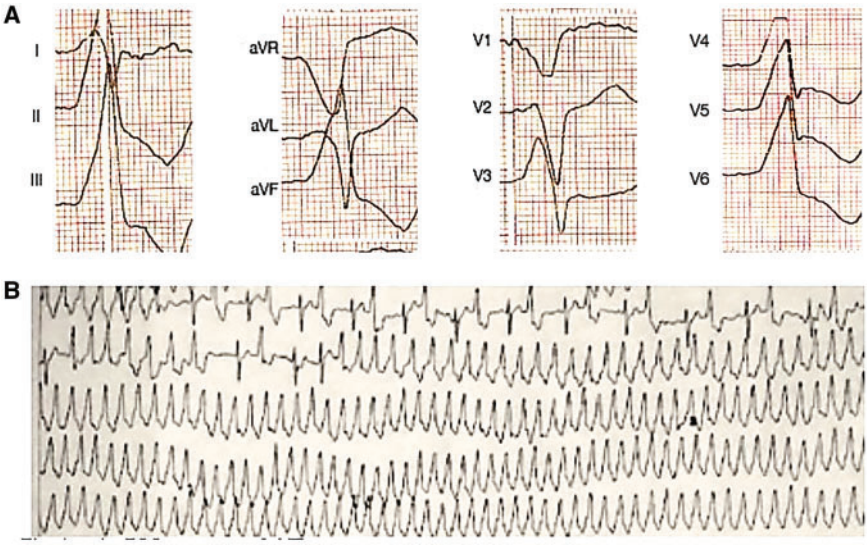

Figure 1 (*A*) Electrocardiography pattern of ventricular tachycardia. (*B*) Ventricular tachycardia episode during 24 h electrocardiography monitoring.

Case presentation

A 38-year-old Asian male patient presented to our hospital with complaints of frequent palpitations and weakness. His past medical history was significant for an acute respiratory infection in 2012, which supposedly had initiated the tachycardia. The patient's tachycardia had increased since 1 month before admission to our hospital for which his cardiologist had prescribed amiodarone 600 mg/day before 10 days. The patient was haemodynamically stable, and his heart auscultation revealed frequent premature beats. Surface electrocardiography (ECG) revealed that both premature ventricular complexes (PVCs) and ventricular tachycardia (VT) had similar left bundle branch block (LBBB) morphology and inferior axis with R/S transition in V_3 (*Figure 1A* and *B*). However, transthoracic echocardiography and complete standard blood tests were unremarkable.

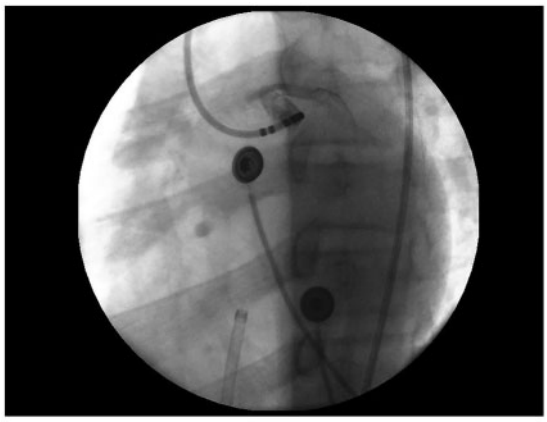

Figure 2 Catheter is in left sinus of Valsalva, contrast inflated through catheter port.

After finding no contraindication for the procedure, we performed the electrophysiological study of the heart. During activational mapping, we determined the earliest activation time (d*T* = 32 ms) in the posterior RVOT. After failure of ablation (45 W, 17 mL/min) in this area (irrigated EZ Steer™ Bi-Directional Ablation Catheter, Biosense Webster), we assumed that the focus of arrhythmia was located in the left side of the heart and consistently checked coronary cusps to find the earliest activation site. Activation mapping performed to identify the earliest activation site showed a d*T* of 20 ms in the right coronary cusp and a d*T* of 32 ms in the LCC. Because ablation procedure in the left sinus of Valsalva (45 W, 17 mL/min) was unsuccessful (*Figure 2*), we used the CS approach. The earliest activation time in the GCV was 32 ms. To avoid coronary artery injury, we performed selective angiography of the left coronary artery (LCA). The catheter tip was located in the projection of the proximal left anterior descending artery 5–6 mm away from the vessel's margin. Ablation through the GCV (20 W, 17 mL/min, 60 s) did not eliminate the arrhythmia.

A focal reaction of the underlying tissue (accelerated ventricular rhythm with clinical PVC morphology) was observed during the unsuccessful catheter ablation through the distal CS and LCC. The earliest activation time was 30–31 ms in all three structures (RVOT, LCC, and CS), and accelerated ventricular rhythm was found during

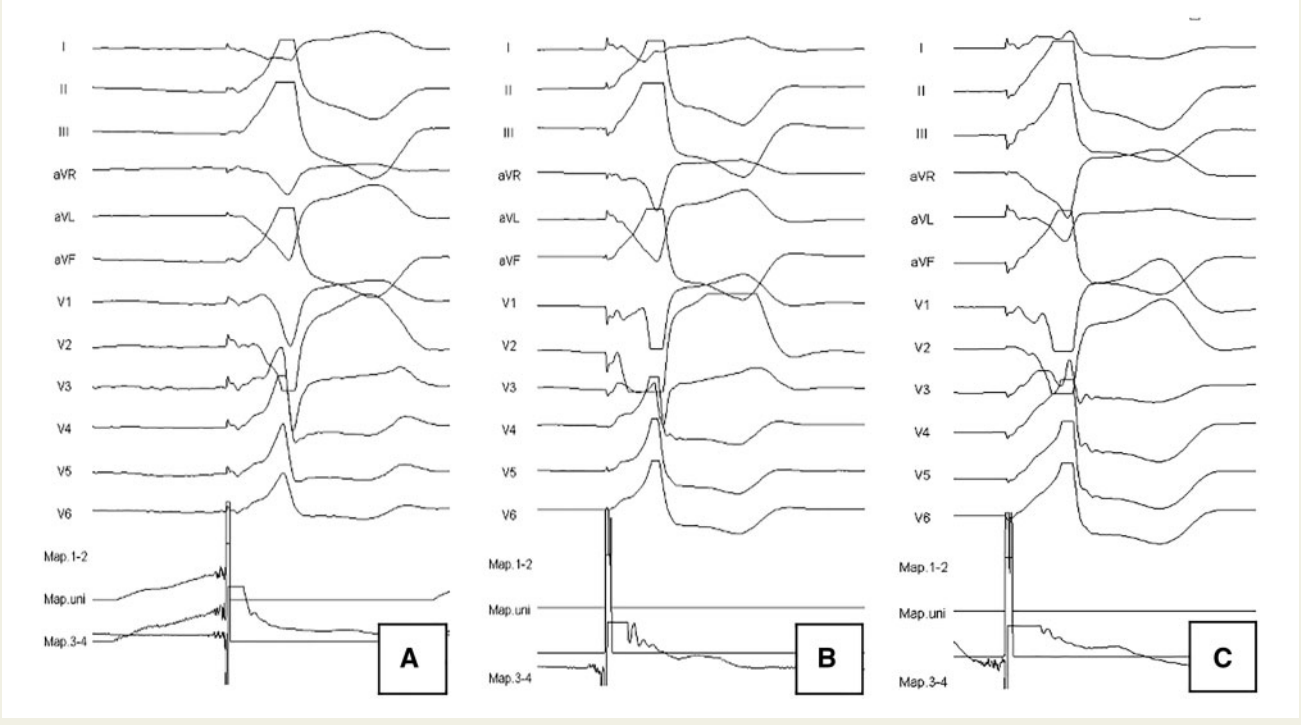

Figure 3 Pace mapping: (*A*) from great cardiac vein, (*B*) from left coronary cusp, and (*C*) from right ventricular outflow tract.

CS and LCC ablation. Because we could not reach total match of the QRS morphology during pace mapping through the LCC, RVOT, and GCV (*Figure 3*), we supposed that the arrhythmia focus was located epicardially, somewhere between these structures. Hence, we attempted to map and ablate the arrhythmogenic myocardium through the left atrial appendage (LAA). After transseptal puncture, we approached the LAA and simultaneously contrasted it with the LCA to determine the dimensional relationship between these two structures (*Figure 4*). The LAA appeared to have windsock morphology. The ablation electrode was then inserted into the LAA and directed towards the left ventricular summit (LVS). We found the earliest activation site, and the measured d*T* at this point was 32 ms. We could not achieve pace mapping from the LAA even with high stimulation parameters (25 V, 3 ms). Before ablation, selective left coronary angiography was performed and the tip of the catheter was found to be 4–5 mm away from LCA bifurcation in two projections (*Figure 5*). Radiofrequency (RF) energy was increased slowly to 40 W for 40 s and lesions were made in two neighbouring points of the myocardium in the posterior apical portion of the LAA by using an irrigated tip catheter with 17 mL/min saline inflation. After the ablation, we could not initiate VT performing standard pacing protocol. In 3 days follow-up on 24 h ECG (Holter) monitoring 1314 single monomorphic PVCs were documented. QRS morphology of newly detected PVCs was different from that of pre-ablation clinical PVCs. The patient was discharged with a prescription of allapinin 25 mg/day. At the 1 month follow-up, 2298 single monomorphic PVCs were detected. The patient had no VT episodes after the ablation.

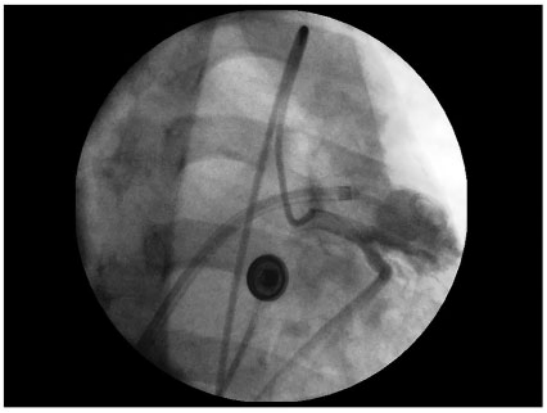

Figure 4 Selective angiography of the left coronary artery and left atrial appendage.

Discussion

In general, VT originating from the RVOT has a LBBB morphology, inferior axis, and, according to numerous investigations, is often terminated by an adenosine injection.[2] The alternative treatment options are RF ablation and conventional therapy with antiarrhythmic drugs. Evidence, current guidelines,[3] and our own clinical experience show that RF ablation can be a first-line treatment for such kind of patients because of its safety, efficacy, and availability.

Currently, there are numerous approaches to ablate ventricular arrhythmias from the Bermuda triangle, or LVS. Those are ablation through anteroseptal and posteroseptal RVOT, LCC,[4] and LV myocardium right beneath the LCC[5]; ablation through distal CS, GCV,

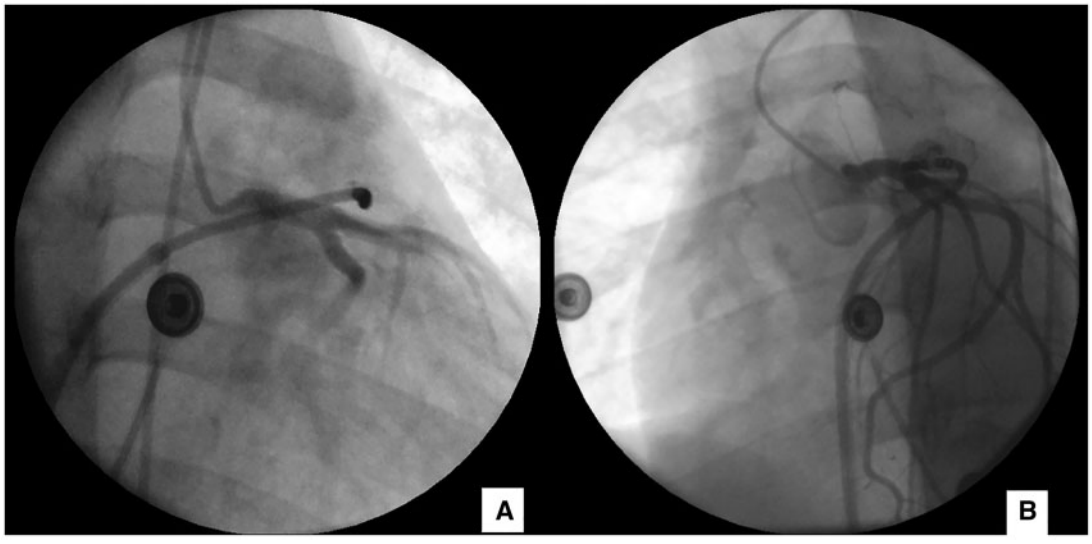

Figure 5 Relations between ablation catheter and left coronary artery. (*A*) RAO 30° and (*B*) LAO 30°. Note that in *B* simultaneous contrasting of left atrial appendage is performed through ablation catheter.

anterior interventricular vein including septal perforator vein[6]; percutaneous epicardial ablation.[7] Although there are many anatomical structures in the area of LVS that we have electrophysiological access to, there is a small area of the myocardium adjacent to all of them that is inaccessible. When ablation from commonly approached structures (i.e. RVOT, LCC, and distal CS) fails, approaching the arrhythmogenic focus through the LAA may be useful.

Radiofrequency ablation from the LAA is considered technically complex and is mostly avoided because of the putative risk of perforation, due to the very thin atrial wall. Therefore, it is not surprising that only one case report on this approach has been published in the previous 15 years.[8] In our case, windsock type of appendage and flexibility of the myocardium allowed us to place the tip of the ablation electrode right onto the target area, thereby increasing the efficacy of the procedure. However, the operator must be careful while dealing with elderly patients and other types of LAA to avoid injuries. In such cases, contact force catheters may be of high value. Although using the three-dimensional imaging system would make the procedure easier and safer, high skilled, and experienced electrophysiologist may perform the procedure conventionally. Moreover, selective coronary angiogram must be performed to assess the proximity of the LCA to the proposed ablation site.

Conclusion

Our case is a good example of using the LAA approach to destruct LVOT epicardial arrhythmogenic focus. The LAA approach is used when the epicardial location of VT-causing tissue is suspected and ablation through the RVOT, LCC, and GCV fails. Future research on the feasibility of catheter manipulations in the LAA is warranted. Selective angiogram must be performed to ensure the safety of coronary arteries before RF application.

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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