

Catheter ablation of ventricular arrhythmia originating from isolated outflow tract diverticulum

Two case reports and literature review

Xinbin Zhou, MD, Haibin Xu, MD, Zhijun Wang, MD st

Abstract

Rationale: Congenital ventricular diverticulum is a rare cardiac malformation with a prevalence of about 0.26% in unselected adult patients during other diagnostic procedures. Ventricular arrhythmia originating from outflow tract diverticulum is even rarer and its etiology, epidemiology and proper treatment still remain controversial.

Patient concerns: We present 2 cases of isolated outflow tract diverticulum incidentally revealed by cardiac angiography during catheter ablation for ventricular arrhythmia. The diverticulums in both cases were found to be the origins of the arrhythmia.

Diagnoses: The 2 patients were both diagnosed with ventricular arrhythmia originating from the outflow tract diverticulum.

Interventions: Catheter ablation was successfully performed for case 1 while a conservative observation strategy was chosen for case 2.

Outcomes: Case 1 has been asymptomatic and free of premature ventricular contractions (PVCs) and both patients have no cardiac event during observational follow-up for 12 months.

Lessons: For ventricular arrhythmia originating from outflow tract diverticulum, catheter ablation may be beneficial and choosing the mouth of the diverticulum or the outflow sites around as the ablation target may be reasonable. An observational follow-up strategy for the small and asymptomatic diverticulum may also be recommended.

Abbreviations: CVD = congenital ventricular diverticulum, EA = earliest activation, LCC = left coronary cusp, LVOT = left ventricular outflow tract, PVC = premature ventricular contraction, RVOT = right ventricular outflow tract, VT = ventricular tachycardia.

Keywords: catheter ablation, outflow tract diverticulum, ventricular arrhythmia

1. Introduction

Congenital ventricular diverticulum (CVD) is a rare cardiac malformation, which may coexist with other congenital cardiac anomalies or present alone. CVD may be associated with significant high morbidity and mortality; however, much remains unknown about its etiology and clinical manifestation, and its treatment recommendations are still undefined and controversial.

Editor: Jacek Bil.

Funding/support: This work was supported by the Zhejiang Provincial Medical Science and Technology Foundation of China (2017KY507 to XZ).

The authors have no conflicts of interest to disclose.

Department of Cardiology, First Affiliated Hospital of Zhejiang Chinese Medical University, Hangzhou, China.

* Correspondence: Zhijun Wang, Department of Cardiology, First Affiliated Hospital of Zhejiang Chinese Medical University, Hangzhou, Zhejiang 310006, China (e-mail: ayla-chen@163.com).

Medicine (2017) 96:46(e8564)

Received: 16 July 2017 / Received in final form: 11 October 2017 / Accepted: 18 October 2017

http://dx.doi.org/10.1097/MD.00000000008564

Here, we present 2 cases of isolated outflow tract diverticulum manifested by premature ventricular contractions/ventricular tachycardia (PVCs/VT) that were incidentally revealed by ventriculography during catheter ablation. The written informed consents were obtained from the 2 patients.

2. Case report

2.1. Case 1

A 46-year-old woman with frequent monomorphic PVCs and history of palpitations was referred for catheter ablation at our cardiology unit. There was no family history of any heart disease. Previous 24-hour Holter ECG revealed large burden of PVCs (34.2%), and the transthoracic echocardiogram did not show any pathological finding. Her physical examination and chest computed tomography were normal, and laboratory results were within the normal range. The 12-lead ECG suggested a typical left ventricular outflow tract (LVOT) origin morphology of PVCs (Fig. 1A).

An electrophysiological study was guided by the Ensite NavX/ Velocity mapping system (St Jude Medical, St Paul, MN). The coronary angiography was firstly performed and no coronary stenosis was found.

Through a 7F arterial short sheath in the right femoral artery, a standard irrigated-tip therapy ablation catheter (Coolpath Duo, St Jude Medical) was inserted via a retrograde transaortal approach for mapping and ablation.

Copyright © 2017 the Author(s). Published by Wolters Kluwer Health, Inc. This is an open access article distributed under the Creative Commons Attribution-NoDerivatives License 4.0, which allows for redistribution, commercial and non-commercial, as long as it is passed along unchanged and in whole, with credit to the author.

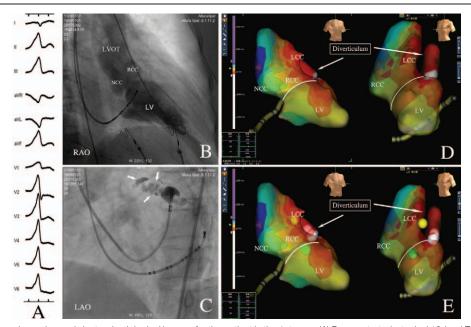


Figure 1. 12-lead ECG, angiography, and electrophysiological images for the patient in the 1st case. (A) Demonstrated a typical 12-lead ECG morphology of PVCs originated from LVOT, (B) shows the angiography result of LVOT and LV, (C) shows an isolated diverticulum in the LVOT with white arrows, (D) shows the earliest activation site at the mouth of the diverticulum (white point); and (E) exhibits the 3 ablation sites. White point for the 1st ablation, yellow point for the 2nd ablation, and green point for the 3rd ablation. ECG=electrocardiograph, LAO=left anterior oblique, LCC=left coronary cusp, LV=left ventricular, LVOT=left ventricular outflow tract, NCC=noncoronary cusp, PVC=premature ventricular contraction, RAO=right anterior oblique, RCC=right coronary cusp.

The clinical PVCs sustained during mapping. The 3-dimensional (3-D) geometry of the LVOT was reconstructed and the earliest activation (EA) site was located under the left coronary cusp (LCC), which was prior to onset of QRS 24ms and the unipolar endocardial electrogram showed a QS morphology (Fig. 1D).

Radiofrequency ablation was then targeted at the EA site, and during further contact activation mapping, the ablation catheter suddenly jumped out of the reconstructed geometry model, but was still within the cardiac structure. Ventricular angiography was then additionally performed by a pigtail-shaped angiography catheter to investigate the unusual phenomenon; however, no obvious malformation at the LVOT was detected (Fig. 1B).

An angiography using the irrigated-tip ablation catheter was applied around the potential abnormal structure and an isolated diverticulum was found with great perfusion pressure. The "string-of-beads" like structure was at the anterior wall of LVOT, and its distal end was blind with a narrow connection to the LVOT cavity. The largest internal longitudinal is 35 mm and transverse diameter is 12 mm (Fig. 1C).

But we failed in inserting the catheter into the structure again after soft attempts. Radiofrequency energy was delivered at the EA site at the mouth of the diverticulum (Fig. 1E). Power ranged from 15 to 40 W with a maximum temperature limited to 55 °C, and PVCs were terminated immediately. However, the patient felt great pain and the impedance fell down quickly and unusually during the short period of ablation. Considering the potential risk, the ablation at this site was aborted.

The 2nd attempt of ablation was delivered in the middle part LCC, PVCs disappeared 5 seconds after ablation but her heart rate became much slower and the patients still felt great pain during ablation.

Thus after further mapping, a 3rd attempt of ablation was delivered in the bottom of LCC, which is just over against the

diverticulum. Although the local potential of this site is not more prior than the 1st point, the ablation catheter was much more steady. Power was delivered at 35 W with a maximum temperature limited to 43 °C, which resulted in the immediate termination of PVCs 8 seconds later and the total ablation time was 180 seconds. No PVC could be induced after an intravenous administration of isoproterenol with an observational period of 30 minutes (Fig. 1E).

After the ablation procedure, anther more focused transthoracic echocardiogram was performed and Color Doppler demonstrated suspicious abnormal blood flow signal at the level of aortic root, but still no obvious abnormal structure. Due to the high risk as the diverticulum was close to the aorta and epicardium, the patient refused to take further surgical intervention.

2.2. Case 2

A 53-year-old man suffered from chest pain and palpitation was admitted to our hospital with frequent PVCs and short runs of nonsustained VT recorded by 24-hour Holter ECG. Previous treatment with metoprolol tartrate failed to release the symptoms, so he referred to our hospital for catheter ablation. He also had a history of lacunar infarction, treated with aspirin and atorvastatin.

Like the 1st case, his physical examination and transthoracic echocardiography did not reveal any abnormalities, and both troponin-I and myocardial creatine kinase value levels were normal.

As the 12-lead ECG predicted a right ventricular outflow tract (RVOT) origin of PVCs/VT (Fig. 2A), electrophysiological study was performed through transvenous pathway guided by the Ensite mapping system (St Jude Medical). During the contact activation mapping, an abnormal diverticulum was found at the anterior free wall of RVOT. And the EA site was located inside

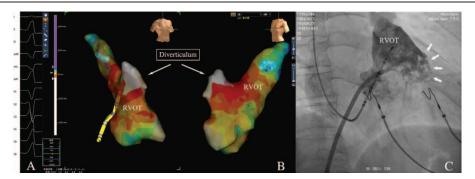


Figure 2. 12-lead ECG, electrophysiological, and angiography images for the patient in the 2nd case. (A) Demonstrated the 12-lead ECG morphology of PVCs which may originate from RVOT, (B) shows the earliest activation site at the diverticulum, and (white zone) (C) shows the diverticulum by angiography with white arrows. ECG=electrocardiograph, PVC=premature ventricular contraction, RVOT=right ventricular outflow tract.

this diverticulum and was prior to onset of QRS by 33 ms with a small r wave in the initiation part of the unipolar endocardial electrogram (Fig. 2B). An RVOT angiography was consequently performed and confirmed the isolated cauliflower-like diverticulum with unclear boundaries in the RVOT anterior free-wall. The largest transverse diameter of the diverticulum was about 3 mm (Fig. 2C).

However, the patient and his family members gave up further ablation procedure or surgical intervention after cautious consideration of the potential benefits and risks. He then received follow-up treatment with propafenone and metoprolol tartrate for rhythm control.

3. Discussion

As a rare cardiac malformation, the 1st report of CVD even dates back to 1816^[1] and until now several cases with different characteristics have been reported, whereas ventricular arrhythmia originating from outflow tract diverticulum has been rarely reported, and its cause, epidemiology and proper treatment still remain controversial.^[2]

According to the cases reported, most CVD cases were asymptomatic and were usually found coincidentally during other diagnostic procedures such as cardiac catheterization, during which it is reported to be about 0.26% in unselected adult patients.^[3]

And most of the CVD cases were isolated diverticulum (69.1%), without any other cardiac malformations.^[4] The association between its location and patient's outcome has not been completely exhibited and most patients stay free of any complications throughout their lifetime.^[5]

However, CVDs could lead to serious complications such as systemic embolization, heart failure, fatal ventricular arrhythmia, diverticular rupture, or even sudden death.^[6,7] Therefore, it is really important to fully evaluate the potential cardiac risks when a CVD was diagnosed.

The subaortic area (48.0%), cardiac apex (24.0%), and leftventricular posterior wall (20.0%) were the most common locations of CVD.^[4] And according to the structural characteristics of the wall, CVD can be classified into 2 types: muscular and fibrous.^[8] Most muscular type is found in the cardiac apex (73.9%) while most fibrous type is located at the subaortic area (82.4%).^[4]

In the present 2 cases, 2 arrhythmogenic isolated outflow tract diverticulums were incidentally found by angiography during catheter ablation for PVCs/VT. The diverticulums might be fibrous type which may be responsible for ventricular arrhythmia for the 2 patients. However, the exact structures were uncertain as surgical procedure and histopathology examination were not performed.

The electrophysiological study demonstrated that the EA sites of both patients located in the CVD and the PVCs were ablated successfully after 3 attempts in the 1st case, whereas the 2nd patient gave up ablation due to the high risk. It may indicate that CVDs in the ventricular outflow tract may tend to induce ventricular arrthytmias and in which case, the EA site may located inside the CVD. However, when choosing the proper ablation target, some difficulties still exist concerning its risks and lack of experience. As exhibited in the 1st case, choosing the mouth of the CVD or the outflow sites around as the ablation target maybe reasonable.

Ventricular arrhytmias may be common complications of CVD, and catheter ablation may be beneficial under governable risks. However, until now the treatment of CVD is still undefined due to its rarity, especially for the patients with ventricular arrhytmias. For the small asymptomatic diverticulum, conservative observation was recommended and for the diverticulum with high risks, cardiac surgery might be the 1st-choice but its exact indications remain controversial.

Therefore, after cautious consideration, we chose observational follow-up for the 2 patients, which has been selected in many reported cases, and also has been supported by the evidence from a series of 16 patients with diverticulum.^[9] The 1st patient has been asymptomatic and free of PVCs and both patients have no cardiac event during observational follow-up for 12 months.

Acknowledgments

The authors thank Zhejiang Provincial Medical Science and Technology Foundation of China (2017KY507 to XZ) for the support.

References

- Romagnoli A, Ricci A, Morosetti D, et al. Congenital left ventricular diverticulum: multimodality imaging evaluation and literature review. J Saudi Heart Assoc 2015;27:61–7.
- [2] Ohlow MA, Lauer B, Lotze U, et al. Long-term prognosis of adult patients with isolated congenital left ventricular aneurysm or diverticulum and abnormal electrocardiogram patterns. Circ J 2012;76:2465–70.
- [3] Yagi S, Akaike M, Fujimura M, et al. Congenital ventricular aneurysm as an unexpected complication of monomorphic premature ventricular contractions. Intern Med 2010;49:907–12.

- [4] Li Q, Qu H, Wang H, et al. Ventricular diverticulum: a review of the literature. J Card Surg 2013;28:133–8.
- [5] Cianciulli TF, Del Carmen Gonzalez Colaso P, Saccheri MC, et al. Left ventricular diverticulum, a rare echocardiographic finding: two adult patients and review of the literature. Cardiol J 2009;16:76–81.
- [6] Marijon E, Ou P, Fermont L, et al. Diagnosis and outcome in congenital ventricular diverticulum and aneurysm. J Thorac Cardiovasc Surg 2006;131:433–7.
- [7] Ozaki N, Sato M, Inoue K, et al. Congenital left ventricular diverticulum in an adult patient. Asian Cardiovasc Thorac Ann 2017;25:388–90.
- [8] Dostalova G, Palecek T, Kuchynka P, et al. A congenital diverticulum of the left ventricular apex manifested by stroke and recurrent ventricular tachycardia. Cardiovasc Pathol 2017;28:3–6.
- [9] Mayer K, Candinas R, Radounislis C, et al. [Congenital left ventricular aneurysms and diverticula: clinical findings, diagnosis and course]. Schweiz Med Wochenschr 1999;129:1249–56. [Article in German].