Successful epicardial ablation for ventricular tachycardia originating from the true apex of apical aneurysm associated with hypertrophic cardiomyopathy

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Abstract

Hypertrophic cardiomyopathy (HCM) with apical aneurysm (AA) is rare, but have been reported to be associated with refractory ventricular tachycardias (VTs). Majority of such cases had a central isthmus of the reentry circuit on the border zone of AA. In this report, we describe a rare case of the successful epicardial ablation for a refractory VT originating from a true apex of the aneurysm in a HCM patient. Mid-diastolic potential during sustained VT was recorded at the isolated epicardial myocardium surround by the gross unexcitable scar in AA, and radiofrequency current application rendered VT non-inducible.

Introduction

Hypertrophic cardiomyopathy (HCM) with left ventricular aneurysm has been reported to have a higher incidence of ventricular arrhythmia compared to that without [1]. Several prior case reports and studies have demonstrated the efficacy of radiofrequency catheter ablation (RFCA) from the endocardium or epicardium[2-5]. In the majority of cases, the central isthmus of those ventricular tachycardia (VT) circuits was present at the border zone of the aneurysm and RFCA targeting this area either by endocardial or epicardial approach has been reported to eliminate VT. Here, we describe a rare case of successful epicardial ablation of VT originating from the isolated myocardium surrounded by the apical dense scar area in a patient with HCM with apical aneurysm (AA).

Case Report

A 60-year-old man with HCM with mid-ventricular obstruction (MVO) and AA was referred to our hospital because of recurrence of an electrical storm due to monomorphic VT. He was admitted to the referring hospital because of an electrical storm and worsening of heart failure. Initially, intensive heart failure treatment using mechanical circulation support and pharmacological antiarrhythmic treatment with amiodarone was undertaken. The electrical storm was inhibited and an implantable cardioverter-defibrillator was implanted. However, electrical storm of the same VT recurred in one week, and endocardial VT ablation was performed. Although VT was temporarily terminated by endocardial ablation, it recurred shortly after the procedure. Therefore, the patient was referred to our hospital.

A twelve lead electrocardiogram of the clinical VT (**Figure 1A**) showed a left bundle branch block morphology and negative concordance with a northeast axis deviation, suggesting an exit from the apico-septal area.

Contrast-enhanced computed tomography (CT) showed a thick interventricular septum and AA (**Figure 1B**).

In prior endocardial ablation, pace mapping at the apico-septal border zone of the aneurysm showed a morphology similar to clinical VT, and an activation map of the VT showed the earliest activation in the same area (**Figures 2A and 2B**). Although endocardial RFCA (Navistar ThermoCool, Biosense Webster Inc., Diamond Bar, CA, USA; maximum power of 35W) for the scar border zone located in the neck of the AA terminated the VT, VT recurred immediately.

As prior endocardial ablation failed and diastolic potential was not recorded in the area with the earliest activation during VT, we planned an epicardial ablation. The procedure was performed under deep sedation using continuous infusion of dexmedetomidine and propofol combined with non-invasive ventilation support. The epicardial approach was secured using a sub-xiphoid puncture. A voltage map and activation map during VT were created using a three-dimensional mapping system (CARTO 3, Biosense Webster Inc.). A low-voltage area was observed at the AA, and the unexcitable scar was widely confirmed in the apex and apico-lateral area of the aneurysm (**Figure 3A**). As VT was hemodynamically stable, an activation map was created and the earliest activation with slight advancement from the QRS onset was observed at the apico-septal neck of the aneurysm (**Figures 3A and 3B**). Entrainment pacing showed a modest fused QRS morphology, whereas the difference between post-pacing interval and tachycardia cycle length (PPI - TCL) was within 20 ms (**Figure 3B**). Radio frequency (RF) current delivery (Navistar ThermoCool, maximum power of 50W) changed the VT QRS morphology, but it did not terminate the VT. Further mapping within the aneurysm showed a mid-diastolic potential at the apex, which was surrounded by the unexcitable scar (**Figures 3C**). RF application terminated VT immediately, and VT became noninducible after additional RF delivery for the surrounding myocardium. During 12 months follow-up, no recurrence of VT has been documented.

Discussion

This report presents a case of a successful RFCA for a recurrent monomorphic VT originating from an AA associated with HCM. The presence of AA in HCM patients has been defined as a risk modifier for sudden cardiac death in the current guidelines [1]. On the other hand, monomorphic VT in this population is rare, and there have been limited case reports or case series[2-5]. The majority of these reports showed that the central isthmus of the VT circuit was present at the border zone (or neck) of the apical aneurysm (**Table 1**).

A recent case series by Igararashi et al. [5], which included the largest patient number to date, concluded that 8 of 15 patients had the estimated epicardial exit of VT and only one of these 8 patients needed ablation within AA, although RF delivery from the endocardial side succeeded in abolishing the VT. To the best of our knowledge, our case is the first report describing successful epicardial ablation for VT with the central isthmus of the reentry circuit in the true apex of an AA associated with HCM. Although we could not completely exclude the possibility of eliminating the VT by endocardial ablation using more precise mapping within the AA, our case highlights that the limited myocardium surrounded by unexcitable scarring within an AA may serve as the central isthmus of the VT circuit in this scarce patient population.

Conclusion

The true apex of an apical aneurysm associated with hypertrophic cardiomyopathy may become the central isthmus of ventricular tachycardia.

Table1 Previous reports of ventricular tachycardia associated with hypertrophic cardiomyopathy and apical aneurysm

Authors	Year
Massimo et al[2]	1997
Bordignon et al[3]	2013

Authors	Year
Shimahara et al[4]	2015
Igarashi et al[5]	2018
MVO = mid-ventricular obstruction, AA = apical aneurysm	MVO = mid-ventricular obstruction, AA = apical aneurysm

References

1. Authors/Task Force m, Elliott PM, Anastasakis A, et al. 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy: the Task Force for the Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC). *Eur Heart J.* 2014;35(39):2733-2779.

2. Mantica M, Della Bella P, Arena V. Hypertrophic cardiomyopathy with apical aneurysm: a case of catheter and surgical therapy of sustained monomorphic ventricular tachycardia. *Heart.* 1997;77(5):481-483.

3. Bordignon S, Chun KR, Schmidt B. Epicardial ablation of monomorphic ventricular tachycardia storm in hypertrophic cardiomyopathy.*Europace.* 2013;15(3):346.

4. Shimahara Y, Kobayashi J, Fujita T, Sato S. Transapical myectomy and surgical cryoablation for refractory ventricular tachycardia due to hypertrophic cardiomyopathy with apical aneurysm. *Eur J Cardiothorac Surg.* 2015;48(2):334-335.

5. Igarashi M, Nogami A, Kurosaki K, et al. Radiofrequency Catheter Ablation of Ventricular Tachycardia in Patients With Hypertrophic Cardiomyopathy and Apical Aneurysm. *JACC Clin Electrophysiol.*2018;4(3):339-350.

Figure legends

Figure 1. (A) A twelve-lead electrocardiogram during sinus rhythm (left panel), showing left bundle branch block and VT (right panel). (B) Computed tomography images demonstrating ventricular hypertrophy at the mid ventricular portion and an aneurysmal apex (red arrows).

Figure 2. Three-dimensional endocardial bipolar voltage (BiV) maps (A) and local activation time (LAT) maps (B) in right anterior oblique (RAO) and left anterior oblique (LAO) views. (A) A dense scar area ($<$ 0.5 mV) was identified in the apical aneurysm (AA), and the border zone area (0.5-1.5 mV) was found in the neck of the AA. (B) The LAT map shows a centrifugal activation from the antero-septal neck of the AA. Red and pink points indicate the ablated area.

Figure 3. (A) Three-dimensional epicardial bipolar voltage (BiV, left panel) and local activation time (LAT, right panel) maps from the antero-posterior view. A green point indicates the antero-septal neck of the apical aneurysm (AA) and a blue point the true apex of AA, intracardiac recordings in which areas are shown in figures 3B and 3C, respectively. Unexcitable scars (gray points) were widely confirmed in the apex and apico-lateral area of the AA. (B) Intracardiac recordings during sustained VT (left panel) and entrainment pacing (right panel) at the neck of the AA (green point in Figure 3A). (Left panel) Local activation slightly (30 ms) proceeded with QRS onset during VT. (Right panel), whereas the difference between post-pacing interval and tachycardia cycle length (PPI-TCL) was within 20 ms, and the paced QRS morphology showed a modest fused morphology. (C) Electrograms and fluoroscopic images of the left anterior oblique (LAO) and right anterior oblique (RAO) views at the true apex of the AA (blue point in Figure 3A). During sustained VT, isolated mid-diastolic potential was recorded, and VT was terminated immediately by radiofrequency (RF) current delivery.