Left ventricular thrombus following radiofrequency ventricular tachycardia ablation

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Introduction

Radiofrequency catheter ablation (RFCA) has become a mainstay in the treatment of both atrial and ventricular tachyarrhythmias since first being developed in 1986. Over 235,000 ablation procedures are performed annually, with 7% of procedures being for ventricular tachycardia (VT) or premature ventricular contractions (PVC), a percentage that continues to grow.[1] As experience in ablation techniques has grown, associated complications have become less common, but they remain an area of focus as the field advances. Thrombus formation is one such complication commonly associated with RFCA, with an incidence of 0.6%–0.8%, a risk that grows to 2.0% for the ablation of ventricular tachyarrhythmias.[2] Despite its being a relatively rare complication, the clinical sequelae of thrombus formation can be devastating, prompting a recent resurgence in the study of postablation thrombogenesis and treatment.

KEY TEACHING POINTS

- While thromboembolic complications have traditionally been associated with ablation of atrial arrhythmias, there is emerging data to suggest they can occur with ablation of ventricular arrhythmias.
- Factors contributing to thrombus formation include denudation of the endocardium, char resulting in coagulum at the areas of ablation, steam pops, recurrent ventricular tachycardia contributing to a low-flow state, and myocardial stunning following the ablation of a ventricular arrhythmia.
- The ideal strategy regarding anticoagulation following ablation of ventricular arrhythmias remains ill defined.

Case report

A 43-year-old man presented from home to the emergency department after a syncopal episode occurring while eating lunch with his family. His past medical history is significant for hypertension, hyperlipidemia, obesity (body mass index of 36.4), and early coronary artery disease with a prior 5-vessel coronary artery bypass graft surgery at the age of 34 using a sequential left internal mammary artery to left anterior descending artery (LAD) and first diagonal, right internal mammary artery to the posterior descending artery, and sequential saphenous vein graft to obtuse marginal 3 and 4 with resultant ischemic cardiomyopathy, contributing to a scar-mediated monomorphic ventricular tachycardia cardiac arrest in 2018 requiring a single-chamber Boston Scientific implantable cardiac defibrillator (ICD) for secondary prevention. Two days prior to his presenting episode, the patient reported having a similar syncopal episode preceded by symptoms of dizziness followed by an unconscious episode lasting for an estimated 5 minutes. After regaining consciousness, the patient reported his defibrillator shocked him 2 times; however, symptoms of dizziness improved for a day, convincing him he did not need further medical attention. Unfortunately, on the day of presentation, his symptoms of dizziness associated with palpitations returned with another 30-second episode of unconsciousness, prompting him to come to the emergency department.

Upon arrival to the hospital, the patient was hemodynamically stable. Labs revealed an acute kidney injury with a creatinine of 1.4 mg/dL, up from a baseline of 0.9 mg/dL, presumed to be due to hypoperfusion related to the syncopal episode. They were otherwise unremarkable. His ICD was interrogated, revealing 2 episodes of wide complex tachycardia at a rate of 280 beats per minute treated with 2 successful defibrillations (Figure 1A). Electrophysiology was consulted and recommended a repeat transthoracic

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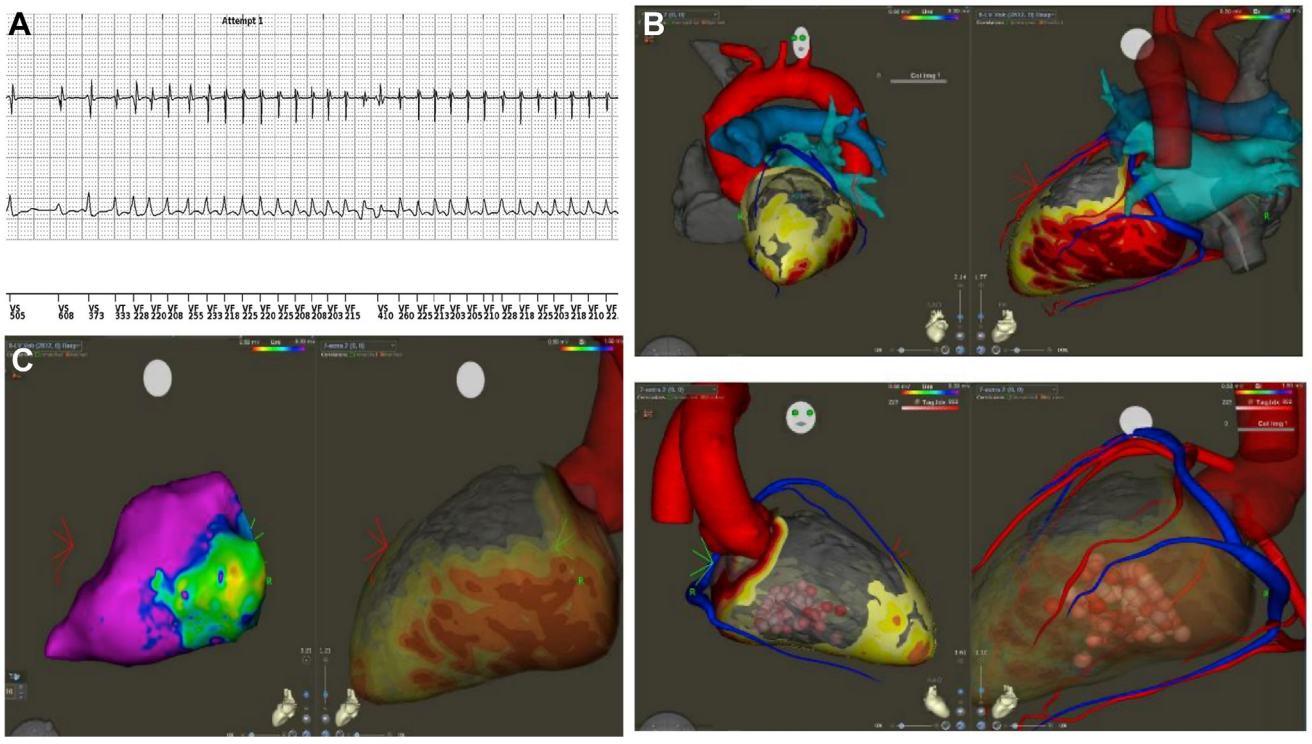

Figure 1 **A:** Single-chamber device electrogram demonstrating ventricular tachycardia (VT) in the ventricular fibrillation zone. **B:** inHEART coronary computed tomography angiography used for VT ablation planning. **C:** Left ventricle (LV) unipolar voltage map demonstrating scar in the inferior LV.

echocardiogram, ischemic workup, and cardiac magnetic resonance imaging (MRI). Transthoracic echocardiogram showed an ejection fraction (EF) of 51% with normal right ventricular systolic function. As the patient had a recent left heart catheterization in 2018, revealing all but the left internal mammary artery–to–LAD grafts being patent, the decision was made to pursue a rest-stress Tc-99m SPECT, which ultimately demonstrated a moderate-size distal LAD scar as well as a large right coronary artery/left circumflex territory scar with minimal peri-infarct ischemia. Given these findings reassuring that ischemia was unlikely to be driving the presenting rhythm, electrophysiology recommended to proceed with VT ablation.

Prior to the procedure, a coronary computed tomography angiography was performed for the purposes of creating an inHEART map to aid in substrate localization (Figure 1B). Imaging demonstrated disease within the basal inferior and inferolateral wall, which was confirmed with unipolar voltage mapping (Figure 1C). Of note, no intraluminal cardiac thrombus was seen on this imaging study (Figure 2A and 2B). The following day, the patient underwent VT ablation. Using a Baylis trans-septal needle, electrocautery was applied to gain access to the left atrium. A heparin bolus and infusion were given to maintain an activated coagulation time (ACT) between 350 and 400 seconds. Two ventricular extrastimuli were delivered from the right ventricle (RV) apex at a cycle length of 600 ms, with rapid sustained monomorphic ventricular tachycardia being induced at 600/250/190 ms. The morphology was a right bundle branch with a superiorly directed axis, consistent with the patient's clinical VT and known inferior left ventricular scar midway between the base and apex (Figure 2C). As it was not hemodynamically tolerated, the VT was pace-terminated. Local abnormal ventricular activity as well as unipolar voltage mapping were used for the VT ablation strategy to obtain scar homogeneity. Radiofrequency energy (RF) was applied at 40 W with a target contact force of 10–20 grams for between 30 and 60 seconds for each ablation lesion using a ThermoCool STSF (Biosense Webster, Irvine, CA) RF ablation irrigated with normal saline at 15 mL/min. After extensive ablation of the basal inferior wall using an irrigated catheter, repeat stimulation was performed with delivery of up to 2 ventricular extrastimuli from the RV apex, similar to induction without recurrence in the presumptive clinical VT. Therapeutic activated coagulation times were maintained throughout the entirety of the procedure, lasting 3 hours and 52 minutes. Total RF time was calculated to be 26 minutes and 39 seconds, with no steam pops noted.

The day following the procedure, a cardiac MRI with gadolinium that had been delayed owing to scheduling conflicts was performed. Despite artifact from sternotomy wires as well as his ICD, limited images demonstrated subendocardial-to-transmural delayed myocardial enhancement along the inferior basilar to mid myocardium. Additionally, there was a curvilinear nonenhancing thrombus extending a length of 4.0 $\times$ 3.5 cm with a thickness of 0.6 cm not present on the computed tomography performed prior to the procedure (Figure 3A and 3B). Following this discovery, the patient was started on apixaban 5 mg twice daily for 1 month.

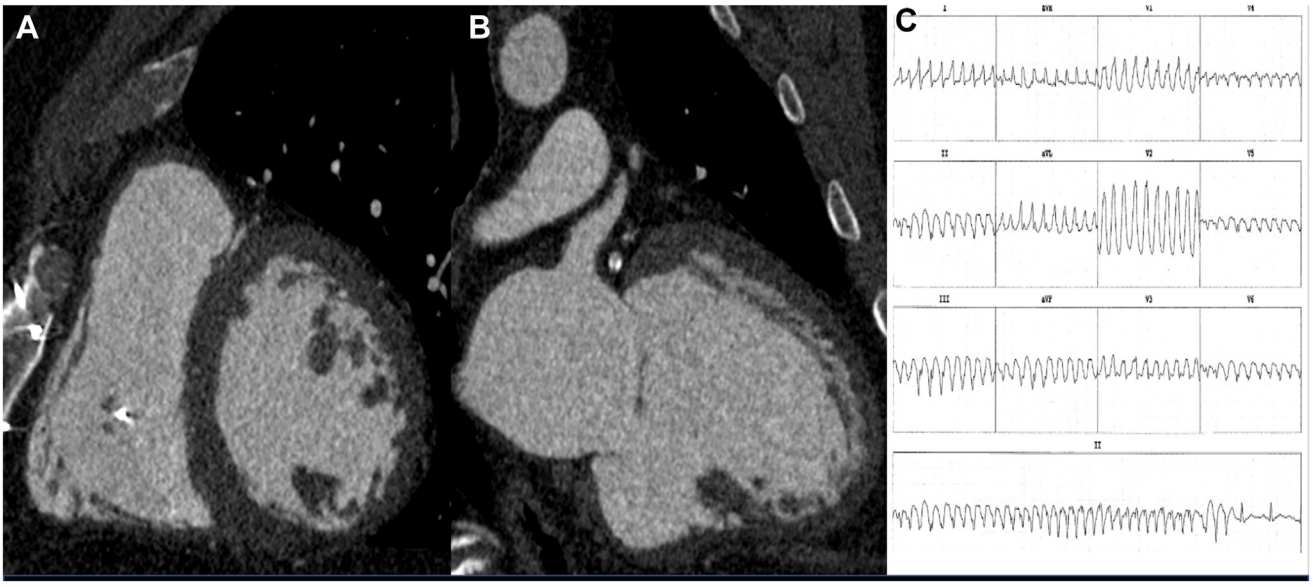

Figure 2 Contrast-enhanced gated cardiac computed tomography angiography obtained 2 days prior to ventricular tachycardia (VT) ablation. **A:** Midventricular short-axis orientation. **B:** Two-chamber orientation. **C:** VT induced during VT ablation procedure.

Discussion

Although the phenomenon has been documented in case studies since the early 2000s, the etiology of thrombus formation and subsequent cerebral emboli has been an area of debate. It is believed that denudation of the endocardium, char resulting in coagulum at the areas of ablation, steam pops, recurrent VT contributing to a low-flow state, and myocardial stunning can increase the risk of thromboembolic events following VT ablation.[3] Additionally, other factors, including presence of VT attributed to structural causes as opposed to idiopathic VT, have been associated with higher rates of complications, including postprocedure thromboembolic events. In 2 multicenter studies involving patients with prior myocardial infarctions undergoing VT ablation, the incidence of procedural complications was found to be as high as 10%.[4] Specifically, ablation of atrial tachyarrhythmias has been associated with development of cerebral emboli in the periprocedural period, a process believed to be due to risks associated with cardioversion and ablation lesions in the low-flow left atrium. The incidence of asymptomatic cerebral emboli detected on postprocedural MRI has been estimated to be as high as 38% of those undergoing RFCA in the left atrium.[5] More recently, it has been established that a similar phenomenon can be seen with ablations of ventricular arrhythmias. Some suggest characteristics differentiating VT ablation in patients with structural heart disease from those without structural heart disease can explain the occurrence of such complications; however, asymptomatic cerebral emboli can be seen following VT ablation in a structurally normal heart.

A small study out of UCSF in 2017 by Whitman and colleagues[1] sought to evaluate the incidence of cerebral emboli using MRI in the week prior to the procedure and an MRI in the 24 hours following the procedure. One hundred and nineteen consecutive patients undergoing either premature ventricular contraction or VT ablation were screened, with 18 patients ultimately being enrolled. Of those enrolled, 12 had ablation in the left ventricle (LV) and 6 had ablation in the RV. Baseline characteristics between both groups were similar; however, 16 new cerebral emboli were discovered in 7 patients of the LV ablation group, with no events noted in the RV ablation group. Additionally, in 70% of the patients with new emboli, a retrograde approach through the aorta was performed. Interestingly, similar studies in patients with aortic stenosis have shown 22% incidence of cerebral emboli while crossing the aortic valve during cardiac catheterization for nonelectrophysiology valvular procedures.[6] These events were posited to be related to the calcific valve disease; however, in this study none of the patients had aortic valvular disease and, in fact, were free of other risk factors for the development of stroke, including impaired systolic function and chronic kidney disease. While all of those with new emboli were asymptomatic, the findings are concerning, as emboli occurred in 58% of those undergoing LV ablation, a significantly higher percentage despite fewer ablation lesions and a higher flow system.

More recently, research has been conducted evaluating the role of postprocedure prophylactic anticoagulation. The STROKE-VT (Safety and Efficacy of Direct Oral Anticoagulant Versus Aspirin for Reduction of Risk of Cerebrovascular Events in Patients Undergoing Ventricular Tachycardia Ablation) multicenter study randomized in a 1:1 fashion 246 patients undergoing RFCA in the left ventricle via either trans-septal or retrograde aortic access to receive direct oral anticoagulant or aspirin (ASA) therapy for a 30-day follow-up period. The primary outcome was transient ischemic attack, stroke, or asymptomatic cerebral event assessed using a brain MRI 24 hours and 30 days following the procedure. In

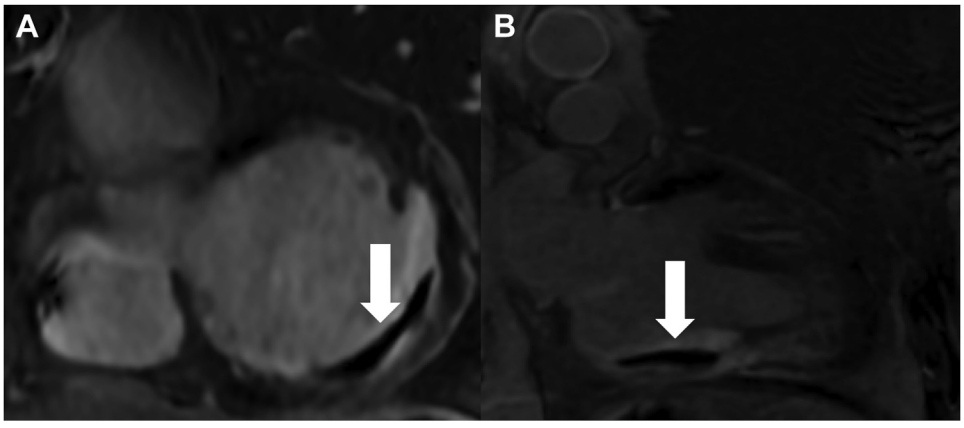

Figure 3 Delayed postcontrast cardiac magnetic resonance images obtained 24 hours following ventricular tachycardia ablation demonstrates curvilinear nonenhancing thrombus (*white arrow*) along inferolateral left ventricular wall in the region of the recent ablation. **A:** Short-axis orientation. **B:** Two-chamber orientation.

the group receiving ASA, the primary outcome was significantly higher, with transient ischemic attacks occurring in 18% vs 4.9%, strokes occurring in 6.5% vs 0%, and asymptomatic cerebral event occurring in 23% vs 12% at 24 hours postprocedure and 18% vs 6.5% at day 30.[3] In a multivariate regression analysis ASA use was found to be the strongest predictor for postprocedure cerebrovascular event, with other significant risk factors including EF, retrograde aortic access, and total ablation time. Interestingly, the benefits seen in the direct oral anticoagulant group came without an increase in mortality, bleeding, or vascular access complications.

While several studies have demonstrated evidence of thromboembolic events following VT ablation, debate persists regarding the long-term clinical sequelae of such events. This is particularly true in the population undergoing intracardiac surgery. One study published in *Stroke* sought to evaluate the relationship between new ischemic lesions following open heart surgery and their implication on cognitive function. The study evaluated 40 patients undergoing intracardiac surgery by performing neurological, neuropsychological, and MRI brain exams 24 hours prior to the surgery, with repeat MRI brain and neurological exam 5 days after the procedure and a neuropsychological exam 6 weeks after the procedure. They found that out of 37 patients ultimately undergoing postoperative MRI, 16 had new ischemic lesions consistent with embolic phenomenon. Cognitive decline, measured using the Reliable Change Index, was present in all of those with new ischemic events, as well as 35% of those without evidence of new events.[7]

Conclusion

Our case highlights what has become an emerging focus in the realm of RFCA of left-sided VT. While the proposed mechanism of thrombus formation consists of a variety of risk factors, including EF, retrograde aortic approach, ablation time, and use of nonirrigated catheters, recent data suggest a potential role for the use of postprocedure prophylactic anticoagulation as a potential step used to mitigate the inherent risk of thromboembolic events. At this point, the long-term consequences of asymptomatic cerebral emboli remain unknown. Population studies suggest that asymptomatic cerebral infarcts confer a higher risk of symptomatic infarcts later in life but recommend treatment aimed at primary prevention given the limited data.[7] Given this uncertainty, research is still necessary to elucidate the clinical neurocognitive impact of such thromboembolic phenomena in addition to an appropriate duration of prophylactic anticoagulation. Additionally, these findings suggest cognitive function testing be included as a long-term functional outcome measure for future electrophysiology studies.

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