Successful ablation of ventricular tachycardia in a patient with Chagas disease using ethanol ablation in the coronary venous system: A case report

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Introduction

Chagas disease is a parasitic zoonosis that constitutes a severe public health problem and is endemic in 21 Latin American countries.[1] It is estimated that between 6 and 8 million people are infected with *Trypanosoma cruzi* (*T cruzi*), with an additional 65 million at risk of acquiring the disease by vector-borne transmission, blood or congenital transmission, or food-borne transmission.[2,3] Chagas disease has an acute, indeterminate, and chronic phase. If untreated, the acute phase may transition to an indeterminate phase characterized by seropositivity for *T cruzi* in the absence of clinical symptoms. A total of 30%–40% of these indeterminate patients may develop organ involvement such as cardiomyopathy, megaesophagus, or megacolon.[4]

Patients with chronic Chagas disease often develop areas of scar tissue and wall thinning in the apical, basal inferior, and lateral walls of the left ventricle (LV), resulting in ventricular tachycardia (VT).[5] The VT often has epicardial circuits owing to wider epicardial scar area in comparison to the endocardium.[6,7] Because of the scar distribution, an epicardial mapping and ablation strategy is often needed. Retrograde coronary venous ethanol ablation has proven to be a feasible, safe, and effective treatment for ventricular arrhythmias arising from deep intramural regions or near coronary vessels.[8]

We describe the first case of ethanol being used in the coronary venous system to create an epicardial ablation in a patient with chronic Chagas VT.

KEYWORDS Chagas disease; Ventricular tachycardia; Ethanol ablation; Coronary veins; Balloon angioplasty
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KEY TEACHING POINTS

- Chronic Chagas disease may cause basal lateral scarring in the left ventricle, resulting in ventricular tachycardia (VT).
- Epicardial mapping and ablation is often required to treat patients with VT from chronic Chagas disease.
- Retrograde coronary venous ethanol ablation in conjunction with a double balloon technique is a viable option to treat epicardial VT in patients with chronic Chagas disease.

Case report

An 81-year-old female patient with a significant past medical history of persistent atrial fibrillation and embolic stroke was initially evaluated for declining left ventricular function (left ventricular ejection fraction dropping from 55% to 40%) and exercise intolerance in 2014. She underwent ablation of atrial fibrillation with subsequent symptomatic improvement and restoration of left ventricular ejection fraction. Two years later, sinus node dysfunction was noted, for which she underwent dual-chamber pacemaker implant. She then presented to the clinic with complaints of presyncope. Her baseline electrocardiogram revealed sinus bradycardia with a bifascicular block (right bundle branch block with a left anterior fascicular block). Device interrogation revealed monomorphic VT; therefore, further work-up was undertaken. Prior cardiac computed tomography angiography had shown no evidence of coronary artery disease. Cardiac magnetic resonance imaging revealed a preserved ejection fraction of 48% with 21% scar, most pronounced in the inferobasal LV. Cardiac positron emission tomography revealed no inflammatory process but a small-size, severe-intensity perfusion defect in the basal lateral segments with 27% scarring of the LV. A suspicion for inflammatory nonischemic cardiomyopathy (particularly sarcoidosis) was raised.

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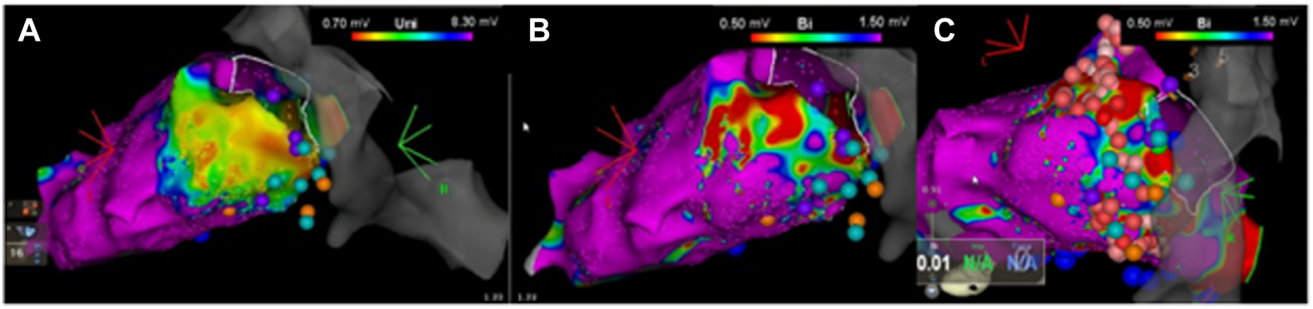

Figure 1 Electroanatomic maps (EAM) of the left ventricle. **A:** Unipolar and **B:** bipolar EAM revealing a large basal, lateral epicardial scar in comparison to the endocardium. **C:** Bipolar EAM with local abnormal ventricular activity and late potentials marked (*aqua and purple dots*), biopsy locations (*orange dots*), and endocardial ablation lesions (*red and pink dots*).

A VT ablation and endomyocardial biopsy were planned, and the patient was brought to the electrophysiology lab and placed under general anesthesia. Heparin infusion was administered for an activated clotting time of >300, and transseptal puncture was performed. A steerable sheath (Baylis Versacross, Montreal, QC, Canada) was advanced into the LV. Through this, a multipolar catheter (PentaRay; Biosense Webster, Diamond Bar, CA) was advanced to create a 3-dimensional electroanatomic map of the LV. Both unipolar and bipolar voltages confirmed significant scarring in the inferobasal and lateral walls (Figure 1a and 1b). A Bipal 7 bioptome 104 cm (Cordis Corp, Miami Lakes, FL) was advanced into the inferobasal aspect of the LV and multiple biopsies were taken, aiming at the low-voltage regions (Figure 1c). VT with a tachycardia cycle length of 216 ms was induced that appeared to originate from the basal lateral wall. The VT was not tolerated and so an endocardial scar homogenization approach was used. Local abnormal ventricular activity (LAVA) and multiple late potentials were seen while mapping the scar, providing a target for endocardial ablation (Figure 1c). Owing to the location of the basal lateral LV substrate and difficulty with catheter manipulation in this region, there was concern the endocardial lesions may not be transmural. After elimination of all LAVA in the endocardial scar—which was made unexcitable—VT remained inducible. A subxiphoid epicardial ablation was considered, since

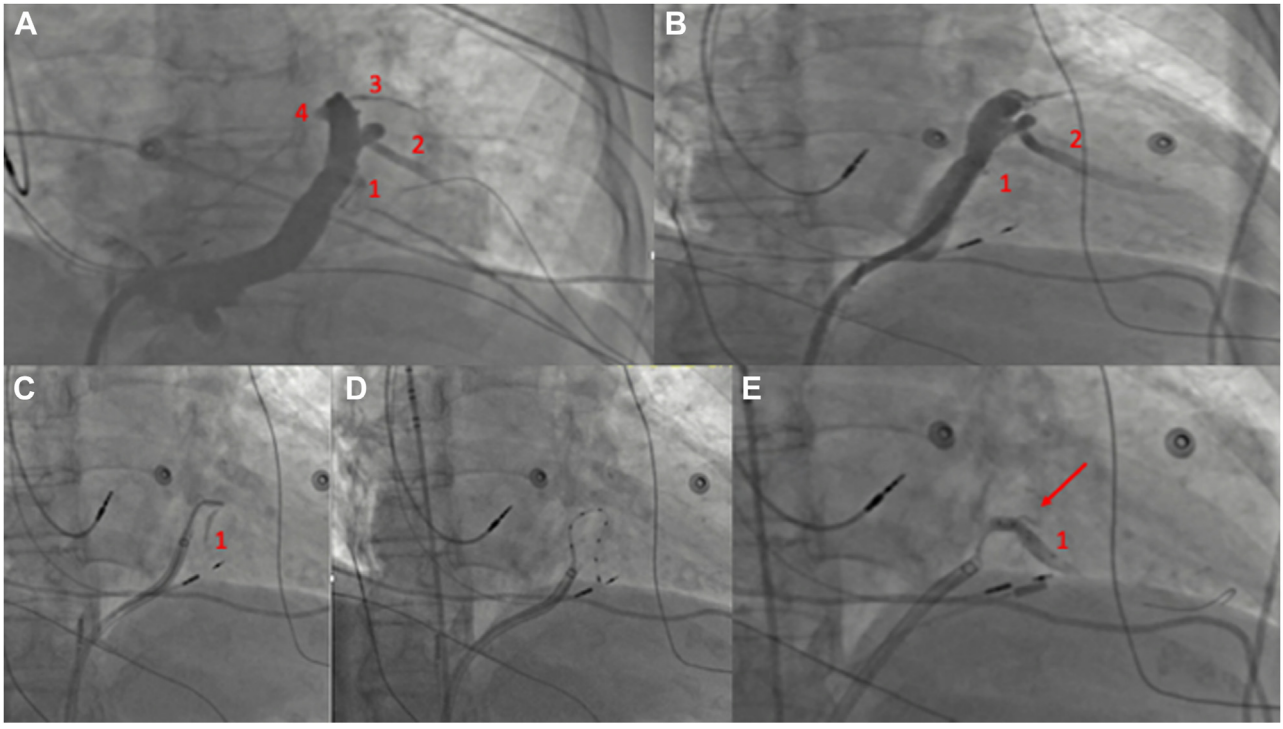

Figure 2 Fluoroscopic images of the venous anatomy and ethanol ablation. **A:** Left anterior oblique (LAO) fluoroscopic view during initial coronary sinus venogram. Veins are numbered as follows: 1, first posterior lateral branch; 2, second posterior lateral branch; 3, marginal branch; 4, left ventricular annular branch. **B:** Right anterior oblique (RAO) fluoroscopic view revealing os of the first posterior lateral venous branch and the second posterior lateral venous branch. **C:** RAO view revealing venogram of the first posterior lateral vein using a JR4 guide. **D:** Fluoroscopic image of EPstar (Baylis Medical) in the first posterior lateral vein branch. **E:** RAO view revealing the double balloon technique exposing a collateral coronary venous branch (*red arrow*) overlying the endocardial, basal lateral, left ventricular scar.

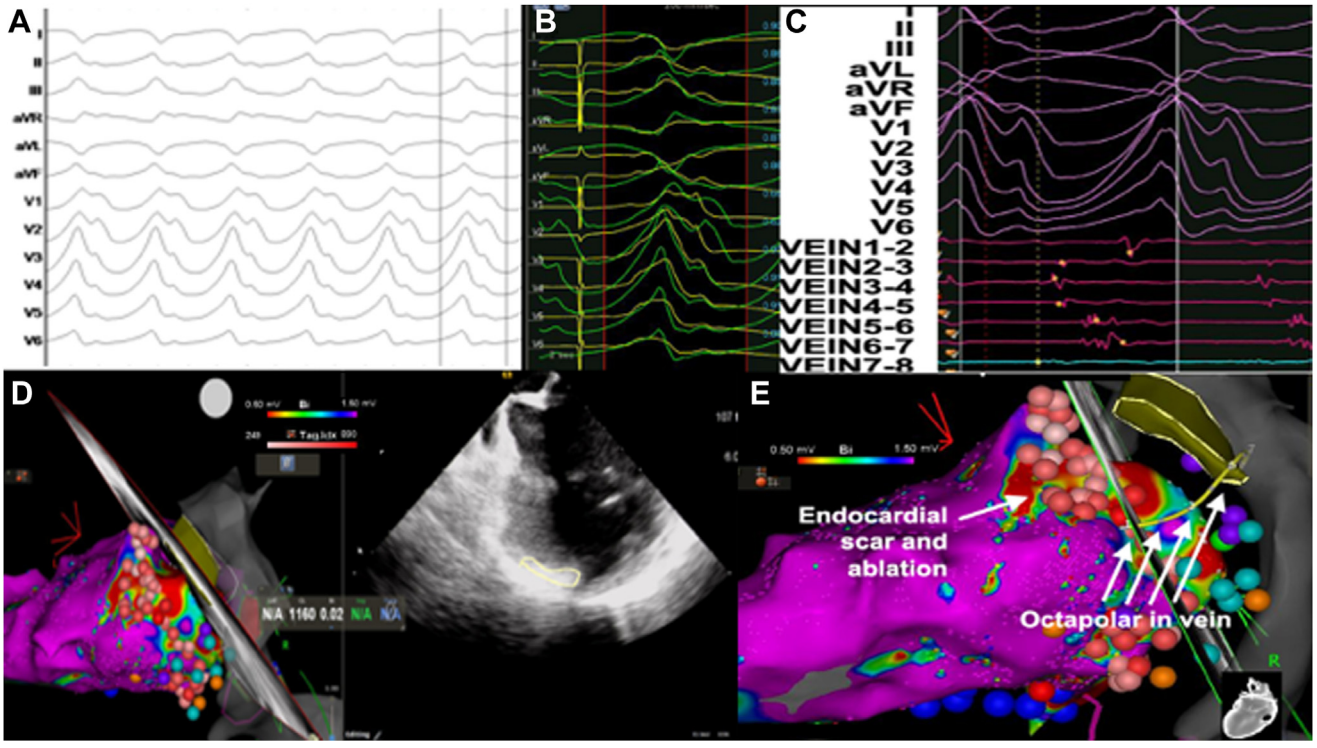

Figure 3 Electrocardiogram (ECG), pace map, and electroanatomic mapping (EAM) during venous mapping and post ethanol administration. **A:** ECG of the anterolateral ventricular tachycardia (VT) induced during electrophysiology study. **B:** Post–endocardial ablation pace map from the EPstar (Baylis Medical) with a 92% match to the VT. **C:** Diastolic signals on the EPstar during VT. **D:** Final post-ethanol EAM showing intracardiac echo images with integrated CartoSound (Biosense Webster). Olive rectangle: CartoSound integrated image tracing the ethanol-induced epicardial/midmyocardial scar seen on ICE. **E:** Final post-ethanol EAM revealing ethanol scar on the basal lateral left ventricle near EPstar poles 5,6,7.

unipolar mapping revealed increased scarring in the basal lateral LV compared to bipolar mapping; however, owing to safety concerns from the patient's body habitus, a subxiphoid approach was abandoned and venous mapping was performed first. The deflectable sheath was engaged in the coronary sinus and venograms identified a lateral vein in the region of the mapped endocardial scar (Figure 2a and 2b). A 4F Glidecath was advanced into the coronary sinus to selectively engage the vein (Figure 2c). A 2F octopolar EPstar (Baylis Medical Company) catheter was advanced into the lateral vein (Figure 2d). Late signals and the ability to pace (92% pace match) on the epicardial aspect of the scar revealed the lack of a transmural endocardial ablation (Figure 3b).The same VT was reinduced and multiple diastolic signals were appreciated (Figure 3c). The 4F Glidecath was removed and an 8F FR-4 was advanced into the lateral vein. Using the double balloon technique described by DaWariboko et al, [9] a 3.5 × 6 mm balloon was inserted into this lateral vein over a BMW wire. Next, a 3.0 × 8 mm balloon was inserted into the lateral vein. The distal and proximal aspects of the lateral vein branch were occluded using a 3.5 × 6 mm and 3.0 × 8 mm balloon in the proximal and distal portions of the lateral vein, respectively (Figure 2e). Two 1 cc injections of 98% ethanol were injected into the proximal balloon with obliteration of a collateral vein overlying the endocardial scar. This created an epicardial and midmyocardial lesion seen on intracardiac echocardiography (Figure 3d and 3e). VT was no longer inducible at 400 ms with triple extrastimuli. Postprocedure, the patient was followed on inpatient telemetry and underwent upgrade of her device to an intracardiac defibrillator.

Biopsies of the LV were negative for any acute or chronic etiology of the scarring. However, on further questioning, the patient revealed she was raised in rural Colombia, where Chagas was endemic. Serology was positive for chronic Chagas disease. Twelve months postprocedure, the patient has had no recurrences of VT.

Discussion

To our knowledge, this is the first documented case where ethanol administration in the coronary venous system was used to complete an epicardial ablation in a patient with VT secondary to chronic Chagas disease. Besides the technical novelty of using venous ethanol for Chagas disease, the case illustrates our own overlooking of Chagas as a cause of VT in the United States.

The cardiac conduction system and myocardium can be affected in up to 45% of patients with chronic Chagas disease.[10] Initial conduction abnormalities include bifascicular block, while late manifestations include sinus node dysfunction leading to severe bradycardia, high-degree atrioventricular blocks, nonsustained or sustained VT, complex ventricular extrasystoles, progressive dilated cardiomyopathy with congestive

heart failure, apical aneurysms (usually of the LV), and emboli due to thrombus formation in the dilated LV or aneurysm.[11] Scarring occurs most commonly on the basal lateral left ventricular wall, creating a substrate for VT and increased risk of sudden cardiac death. The VT frequently has epicardial circuits requiring epicardial mapping and ablation.

Case reports have been published reporting the treatment of Chagas VT with transcoronary chemoablation in the distal left anterior descending and distal left circumflex artery.[12–14] Described complications include incomplete lesions creating a nidus for reentrant tachycardia circuits, complete atrioventricular block when targeting basal septal VA substrate, nontarget coronary vessel occlusion, contrast nephropathy, systemic embolization, coronary vasospasm, arterial perforation, myocardial dissection, and potential damage outside the targeted area by unintended ethanol reflux into nontargeted arterial branches.[9,15]

Chagas VT often has epicardial circuits requiring mapping and ablation in the epicardium. An endocardial/epicardial (endo/epi) approach has proven to be an effective strategy, as seen in the Efficacy and safety of combined endocardial/epicardial catheter ablation for VT in Chagas disease randomized trial that was published in 2020.[5]

In our patient, endocardial scar ablation was performed with elimination of LAVA, late potentials, and the ability to capture inside of the scar. However, owing to the basal lateral substrate location causing difficult catheter manipulation, there was concern that transmural lesions were not being created. Unipolar electroanatomic mapping was consistent with epicardial scar requiring epicardial ablation; however, with her body habitus, subxiphoid epicardial puncture was felt too high of a risk. Venous mapping confirmed late potentials and VT inducibility. The targeted posterior lateral vein appeared too small for cannulation during initial venogram, but as we commonly see, the vein was much larger during balloon occlusion venogram. Using the double balloon technique, ethanol venous ablation was successful in eliminating VT and preventing recurrence on follow-up.

Conclusion

Chronic Chagas disease can cause many cardiac arrhythmias, including sinus node dysfunction, interventricular conduction delays, high-degree AV blocks, and VT. Chagas cardiomyopathy should be considered in the differential diagnosis of nonischemic VT in the United States. The treatment of VT in these patients is commonly in the basal, inferolateral aspect of the LV, often requiring epicardial access. Ethanol ablation via the coronary venous anatomy can offer electrophysiologists another option in their armamentarium of treatments against VT.

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