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CASE REPORT

INTERMEDIATE

CLINICAL CASE/TECHNICAL CORNER

Ablation of Incessant Premature Ventricular Complex Through Retrograde Transvenous Ethanol Infusion

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ABSTRACT

Ethanol infusion has been used for the treatment of ventricular arrhythmia. We describe a case of ethanol infusion through the coronary sinus venous network to treat refractory epicardial premature ventricular complexes. The premature ventricular complexes were initially successfully suppressed but recurred after resolution of the myocardial edema. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2020;2:973–8) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Frequent premature ventricular complexes (PVC) may cause left ventricular (LV) dysfunction. Treating PVCs and LV dysfunction is especially challenging in cases of intramural and epicardial foci. Epicardial ablation is an appealing alternative but bears some potential complications (1). Transcoronary ethanol infusion has been successfully used for ablation of arrhythmia (2), but complications such as collateral myocardial damage and complete atrioventricular block (3) limit the use of ethanol infusion. Recently, a new technique has emerged based on a retrograde venous ethanol infusion using the coronary sinus (CS) vein network. This approach has been used as an adjunct therapy for ablation of atrial fibrillation (4,5) and for ventricular tachycardia (VT) ablation in 2 short series of 7 patients (6). This paper reports the case of a 24-year-old male patient with LV dysfunction and frequent PVCs treated with retrograde venous ethanol infusion.

LEARNING OBJECTIVE

- To recognize the role of alternative techniques in refractory cases of ventricular arrhythmia.
- To recognize the role of cardiac magnetic resonance to monitor the effect of ablation.

PRESENTATION

A 24-year-old male patient was referred to the authors' department in January 2019 for ablation of incessant PVCs associated with LV dysfunction. The patient's symptoms were marked exercise capacity reduction with no overt clinical signs of heart failure.

MEDICAL HISTORY

A high incidence of monomorphic PVCs (30%) was first discovered in 2012. Cardiac magnetic resonance

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ABBREVIATIONS AND ACRONYMS

CMR = cardiac magnetic resonance

CS = coronary sinus

LGE = late gadolinium enhancement

LV = left ventricle

MVO = microvascular obstruction

NSVT = nonsustained ventricular tachycardia

PVC = premature ventricular complex

VT = ventricular tachycardia

(CMR) showed no structural anomaly and normal LV function and the absence of fibrosis. Treatments with beta-blockers and amiodarone were unsuccessful. In 2013, the first retroaortic ablation attempt was unsuccessful, resulting in a low local prematurity, raising the suspicion of an epicardial focus.

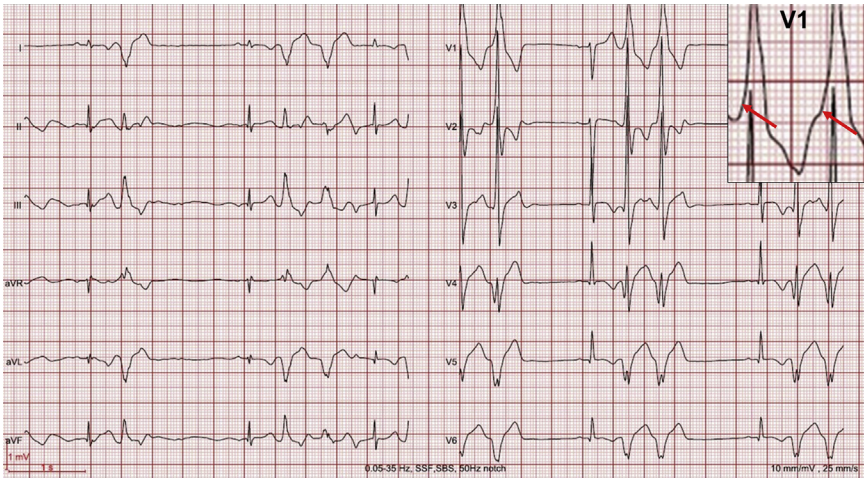
INVESTIGATIONS

Repeated CMR performed in May 2018 revealed LV dilation and reduced LV ejection fraction (40%). Electrocardiography (ECG) showed PVCs with a morphology that suggested an origin from the mid-lateral LV wall (Figure 1). The patient was then referred to the authors' center for a new endocardial attempt through transseptal access, with additional retrograde venous ethanol infusion in case of repeated failure.

MANAGEMENT

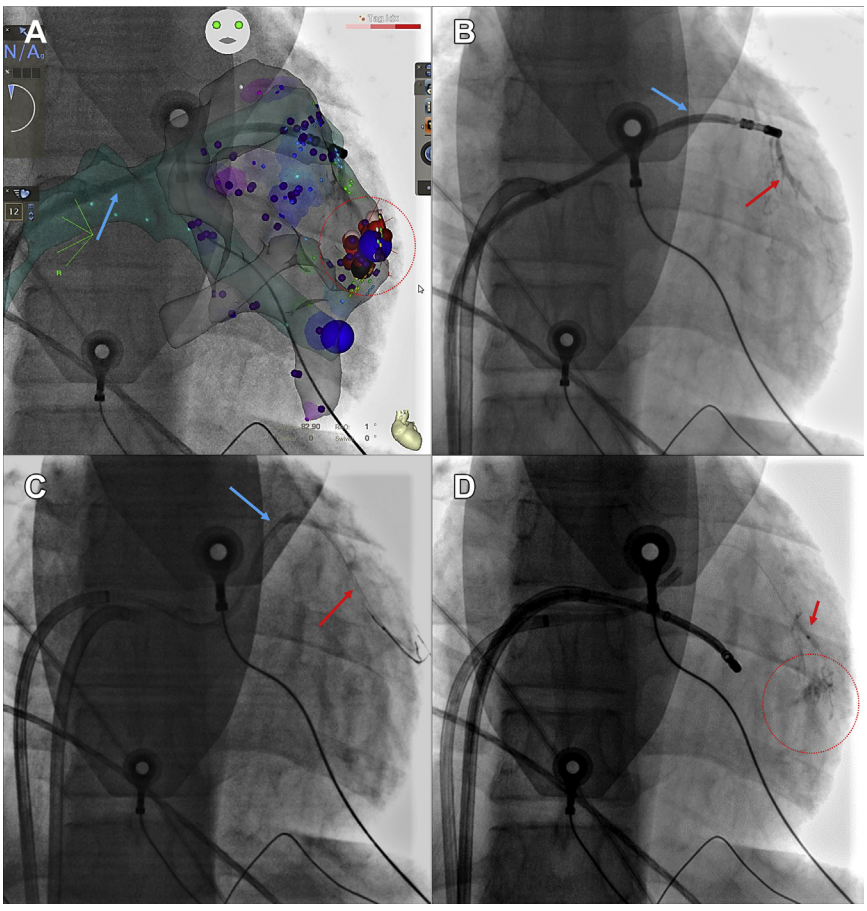
With the patient under general anesthesia, a decapolar catheter (Decanav, Biosense Webster, Irvine, California) was advanced into the CS. An ablation catheter (SmartTouch SF, Biosense Webster) was inserted into the LV using a transseptal access. The site of earliest endocardial activation during the PVC was at best at 0 ms compared to the QRS onset and localized at the junction between the mid and basal thirds of the inferolateral LV. The endocardial approach failed despite prolonged ablation with significant force and power application (20 g, 40 W, with impedance monitoring showing a significant drop). The CS and 2 lateral side branches directed toward the arrhythmic focus were reconstructed using the ablation catheter (Figure 2A). The anterolateral vein appeared to drain the site of PVC origin as shown by both selective contrast infusion (Figure 2B) and 5°C saline infusion that briefly suppressed the PVCs (Figure 3). One milliliter of 96% ethanol injected through the ablation catheter triggered a nonsustained VT that perfectly matched the morphology of the clinical PVC followed by its disappearance (Figure 4, top panel). However, the PVCs resumed within minutes. A coronary guiding catheter (Voda Left, SciMed, Durham, North Carolina) was advanced into the CS allowing the insertion of 2 guidewires (Sion blue, Asahi, Tokyo, Japan) into a downstream vein (Figure 2C). A 1.5-mm balloon (Sprinter Legend, Medtronic, Dublin, Ireland) was then inflated for 5 min to prevent backflow during the infusion of 1 ml of ethanol (Figure 2D). A nonsustained VT occurred again, followed by PVC termination without procedural recurrence. Monitoring of the patient on the ward showed an initial high incidence of PVCs and nonsustained VTs, followed by gradual disappearance with lengthening coupling intervals over the next 24 h, until complete disappearance

FIGURE 1 12-Lead ECG of the PVC



The PVC displays a complete RBBB morphology in V_1, suggestive of an LV origin. The QS pattern in V_5 and V_6, and in aVL and D_I localizes the focus on the LV lateral wall, and the pseudo-delta wave in V_1 suggests an epicardial focus (see **inset**). ECG = electrocardiography; LV = left ventricle; PVC = premature ventricular complex; RBBB = right bundle branch block.

FIGURE 2 Fluoroscopic and Electroanatomical Mapping Views of the CS Cannulation



(A) Anteroposterior combined fluoroscopic and electroanatomical mapping views show the ablation catheter **(blue arrow)** in the target vein of the CS (**transparent green**). The site of earliest endocardial activation is encircled in **red**. **(B)** Anteroposterior fluoroscopic view of the angiogram of the target CS vein **(red arrow)** performed through the ablation catheter **(blue arrow)**. **(C)** Two guidewires **(red arrow)** were inserted through a guide catheter **(blue arrow)**. **(D)** Myocardial staining **(circle)** after ethanol infusion. CS = coronary sinus.

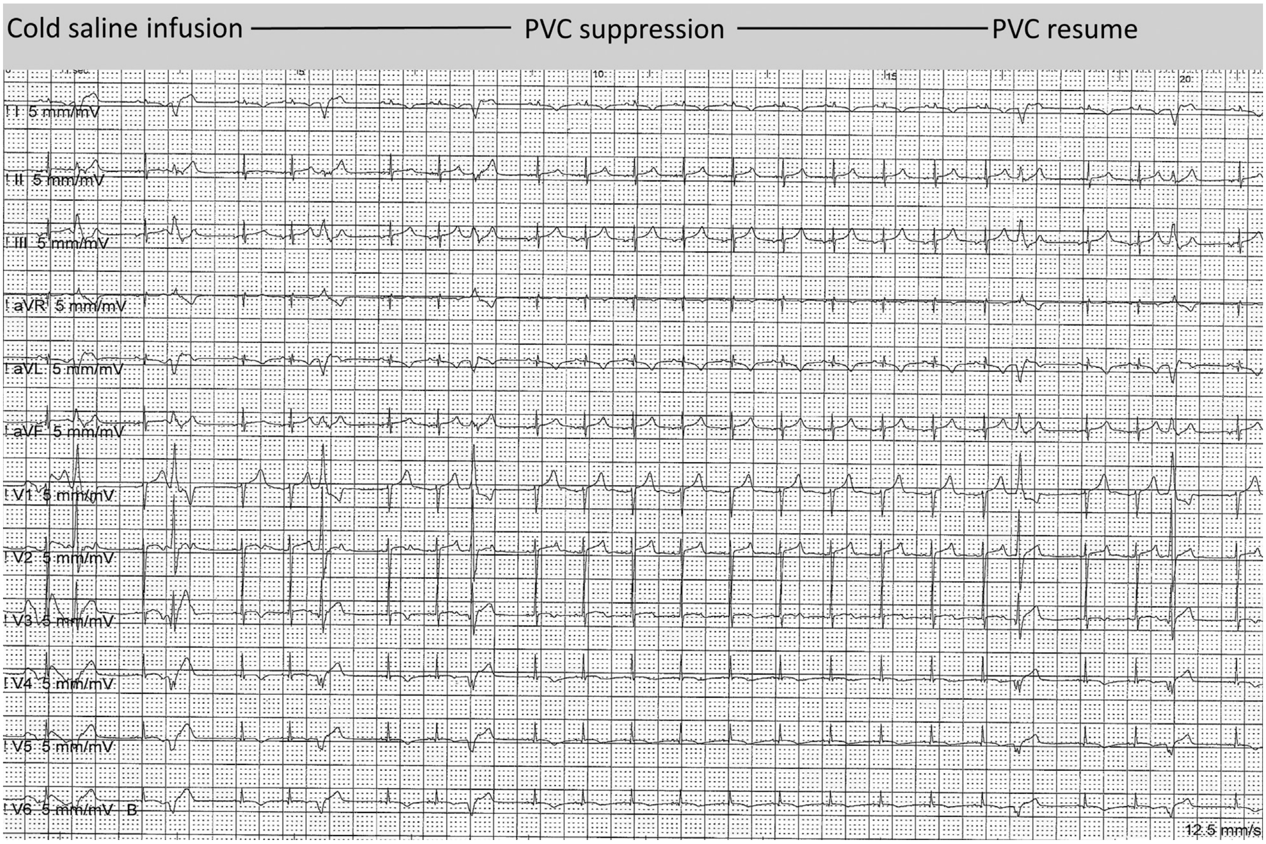
(Figure 4, middle and bottom panels). A CMR performed 48 h after the procedure showed late gadolinium enhancement (LGE) affecting 4 segments at the junction between the basal and median thirds of the LV lateral wall with small areas of microvascular obstruction (Figure 5A). At 3 months, a new CMR showed slight improvement of LV ejection fraction (from 32% right after the ablation to 40% to 45%) and marked reduction of the LGE from 4 to 2 segments with a subendocardial distribution involving 50% of the myocardial thickness (Figure 5B, arrow). Nevertheless, the referring cardiologist reported PVC recurrence, although at a lower incidence, 4 weeks after ablation.

DISCUSSION

This case reports the ablation of PVCs by using retrograde CS ethanol infusion with acute suppression followed by recurrence after 4 weeks. This approach offers the advantage of feasibility using the same femoral venous access rather than the transseptal approach, allowing the operator to reach the epicardium without the need for a dry pericardial puncture and its potential complications. Three observations can be drawn.

ARRHYTHMIC FOCUS LOCALIZATION. With regard to ethanol toxicity, it is highly recommended that the

FIGURE 3 Effect of Cold Saline on PVC



Transient suppression of PVCs by cold saline infusion into the target vein. Abbreviation as in Figure 1.

target myocardium be targeted precisely. Cold saline injection is confirmed as an effective tool that can be used to map the myocardium to be ablated (7). Interestingly, venous mapping with contrast and saline injection did not require specific guiding catheters but were performed using the ablation catheter.

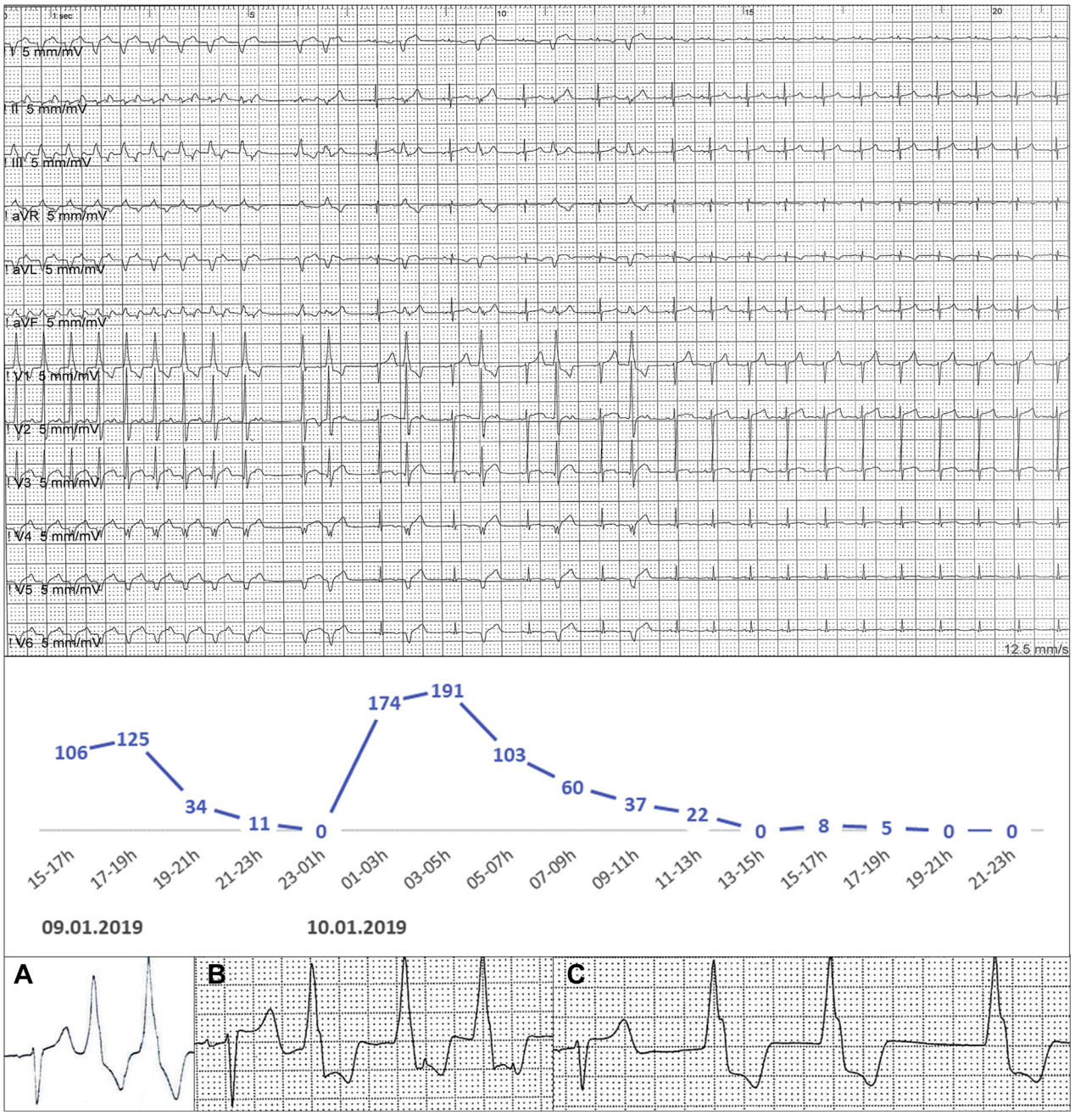
ETHANOL VOLUME. Here, 2 ml of ethanol was used. The optimal dose remains to be determined, given the limited experience reported so far (6). This issue is important because an excessive volume of ethanol may cause collateral myocardial damage, whereas an insufficient dose may favor PVC recurrence. Based on the PVC recurrence and on the CMR lesion assessment reported here, one or more repeated injections are most probably warranted in such cases as proposed by others, who tended to repeat the 1-ml injection (up to 4 ml) (6).

CMR IS USEFUL TO MONITOR ABLATION DAMAGES AND MYOCARDIAL RECOVERY. Even if the spatial and temporal resolution of CMR does not allow precise differentiation between the effect of the endocardial and epicardial ablation, the epicardial ablation, the extended edema associated with PVC disappearance 2 days after the current procedure speaks for a combined effect of both approaches.

Residual LGE seen at 1 month spanning 2 thirds of the myocardium toward the epicardium correlates well with the site of the endocardial approach, whereas the reappearance of viable epicardial tissue after edema resolution, in parallel with PVC recurrence, militates for an insufficient epicardial ablation. Interestingly, the first CMR performed before the current procedure did not show LGE at the site of the first ablation attempt in 2013.

Treatment of the present case bears some limitations, such as the lack of definite proof that the focus was indeed epicardial, which might have been confirmed and successfully ablated based on a detailed epicardial mapping using a dry pericardial puncture. Alternatively, a more focused identification

FIGURE 4 PVC Evolution After the Ablation

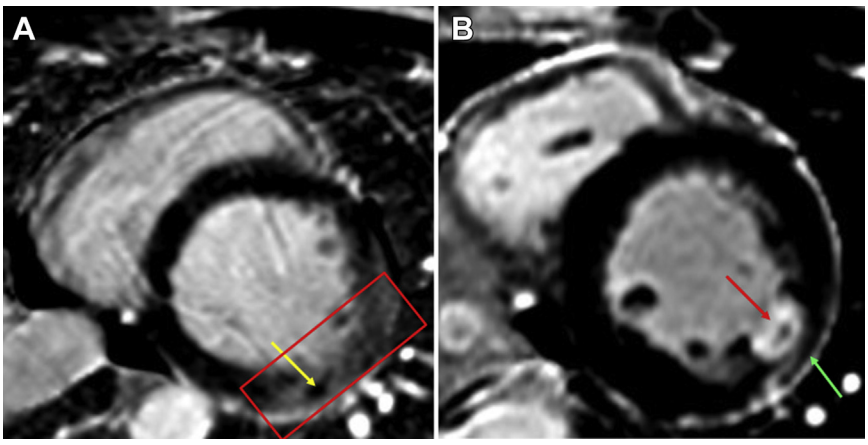


(Top) Nonsustained VT reproducing the clinical PVC morphology after ethanol infusion. **(Middle)** Evolution of PVC count during monitoring. **Numbers** indicate PVC incidence per 2-h periods. **(Bottom)** Progressive lengthening of PVC coupling intervals after ablation. **(A)** Extract from the original PVC before ablation. **(B)** Telemetry strip during the first 12 h. **(C)** Telemetry strip 24 h after the ablation. Abbreviations are as in Figure 1.

of the target site could be obtained through detailed electrical mapping of the CS network using dedicated unipolar guidewires.

Finally, hypo-osmolar irrigation of the ablation catheter could have been used as it has been shown, first in animal models and then in humans (8,9), to allow deeper penetration of ablation energy into the muscle, which seems very promising when targeting intramural or epicardial focus.

FIGURE 5 CMR Views of the Ablation Zone



(A) T1-weighted inversion-recovery gradient echo sequence shows diffuse and transmural LGE on the lateral wall **(red square)** with a small area of MVO **(yellow arrow)** 48 h after ablation. **(B)** T1-weighted inversion-recovery gradient echo sequence of the same slice shows reduction in the extension of the subendocardial LGE **(red arrow)** and reappearance of a thin rim of viable epicardial tissue **(green arrow)** at 3 months. LGE = late gadolinium enhancement; LV = left ventricle; MVO = microvascular obstruction.

FOLLOW-UP

A combined attempt using retrograde venous infusion and the percutaneous epicardial approach through pericardial puncture was programmed, but the patient did not show up to follow-up visits.

CONCLUSIONS

Retrograde venous infusion of ethanol seems technically feasible for refractory cases of PVC/VT. To optimize efficacy-to-damage ratio, local epicardial mapping may be achieved using dedicated unipolar mapping guide wires. The optimal volume of ethanol needs to be determined as well.

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