Management of refractory ventricular tachycardia by direct intramyocardial injection of alcohol: A novel method

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Introduction

Epicardial surgical ablation of ventricular tachycardia (VT) is preferred when percutaneous access to epicardial space is restricted by adhesions or ablation cannot be performed owing to overlying coronary artery. Even after surgical exposure, ablation of VT originating beneath a major coronary artery can be challenging.[1] We describe a case of epicardial VT presenting as a VT storm in which tachycardia was originating beneath the left circumflex artery (LCX). Successful ablation was performed in this case with direct intramural alcohol injection after surgical exposure.

Case report

A 40-year-old man, a known case of nonischemic cardiomyopathy was referred to our institute for management of VT storm. Two years earlier, he had undergone dual-chamber implantable cardioverter defibrillator implantation when he presented with VT and underlying high-grade atrioventricular block. Presently, he had multiple episodes of monomorphic VT, which were refractory to antiarrhythmic drugs (AADs). The morphology of the VT was a right bundle branch block with inferior axis (Figure 1a). He underwent endo-epicardial mapping and ablation guided by a 3-dimensional electroanatomic mapping system (CARTO 3; Biosense Webster Inc, Diamond Bar, CA). As the clinical VT was hemodynamically unstable, substrate mapping was performed after VT termination. Left ventricular endocardial mapping did not reveal any abnormal substrate for ablation. Following this, pericardial space was accessed through subxiphoid puncture. Low-voltage substrates (bipolar <1 mV) representing scar and abnormal signals including fractionated potentials and isolated late potentials were found in the basal anterolateral region of the left ventricle (LV). These abnormal epicardial substrates were ablated with a 3.5 mm open irrigated-tip electrode catheter (7.5F, Navistar ThermoCool; Biosense Webster) with flow rate of 2 mL/min, temperature cut-off of 43°C, and maximum power of 30–35 W. Pace-mapping in the anterolateral region of the LV showed good pace-map morphology match (pace-map score 22/24) to the clinical VT (Figure 1b). However, this site could not be ablated, as angiogram in multiple views showed large LCX at the proposed site of ablation (Figure 1c and d). Ablation from the diametrically opposite site from the endocardium also failed to abolish this focus. Post ablation, there was only ill-sustained clinical VT induced with 3 ventricular extrastimuli. Further, the patient was managed with oral AADs including amiodarone.

Two months later, he presented with incessant slow VT with the same morphology refractory to multiple AADs. As the target site for VT ablation was adjacent to a major epicardial coronary artery, the patient was offered surgical ablation.

KEY TEACHING POINTS

- Intramural alcohol injection can be considered for ablation of ventricular tachycardia arising beneath the major coronary artery during open surgical procedure.
- Intramural alcohol injection at the intended site produces small infarcts owing to direct cytotoxic effect of alcohol.
- Compared to transcoronary alcohol ablation, direct chemical ablation has a lesser chance of lesion extending beyond the required site of ablation owing to absence of spillover effect.

KEYWORDS Chemical ablation; Direct intramural alcohol ablation; Epicardial ablation; Surgical ventricular tachycardia ablation; Ventricular tachycardia ablation

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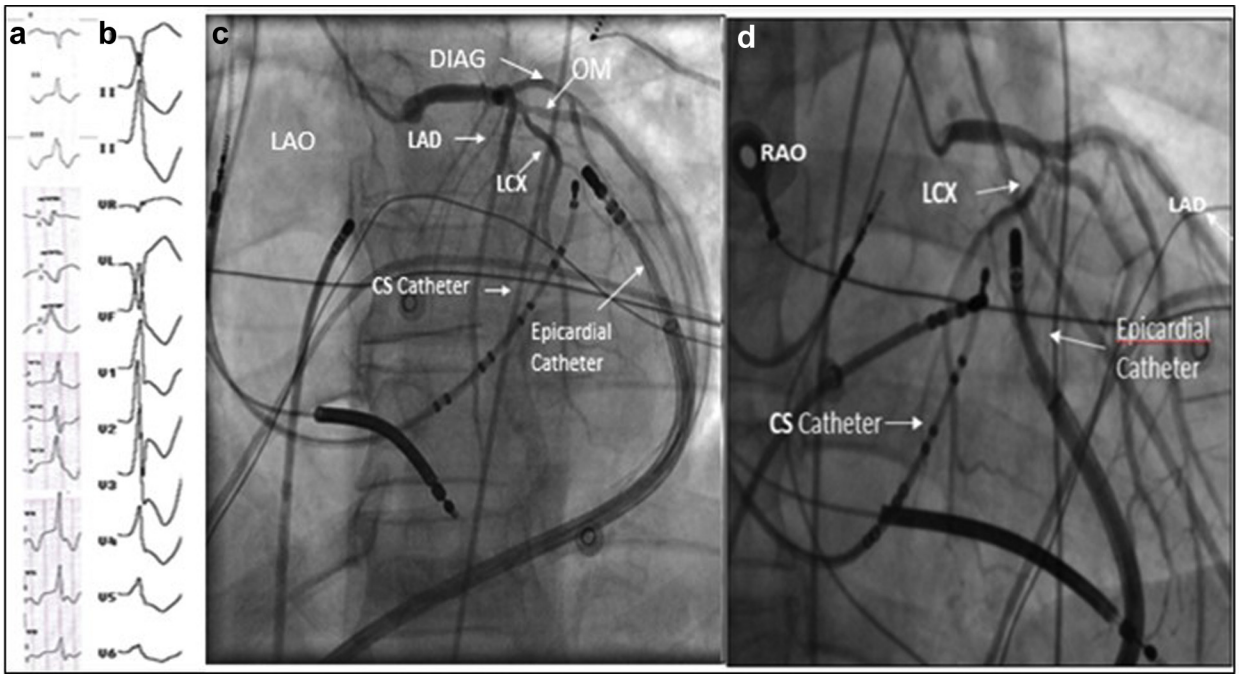

Figure 1 **a:** The 12-lead electrocardiogram of clinical ventricular tachycardia (VT). **b:** Good pace-map (22/24) of clinical VT from epicardial anterolateral wall of left ventricle. **c,d:** Fluoroscopic left anterior oblique (LAO) and right anterior oblique (RAO) view of epicardial mapping showing epicardial ablation catheter near left circumflex coronary artery (LCX), at the good pace-map site. CS = coronary sinus; DIAG = diagonal branch; LAD = left anterior descending coronary artery; OM = obtuse marginal branch.

Under general anesthesia, median sternotomy was performed to expose the proposed site of ablation in the LV. Mapping and ablation in the operating room was performed with the help of the mapping catheter taped to the surgeon's index finger.[2] During mapping, tachycardia consistently terminated during mapping owing to catheter pressure adjacent to the LCX. At this location, bipolar electrogram during VT showed presystolic fractionated signal preceding QRS by 52 ms with QS morphology in unipolar recording (Figure 2b). Multiple attempts to entrain the VT resulted in termination of VT, so we decided to pace-map the VT. Paced QRS morphology at this site showed a good pace-map match (Figure 2d). Owing to the risk of vessel damage during ablation, the coronary artery segment was mobilized and lifted off the epicardium with the help of a tape loop (Figure 3a). Following this, cryoablation was attempted beneath the vessel. It resulted in repeated severe coronary vasospasm leading to ST-segment elevation owing to myocardial ischemia. Coronary vasospasm was relieved by topical application of papaverine and release of the mobilized vessel. To avoid further vasospasm and myocardial injury, we decided to perform chemical ablation with injection of absolute alcohol. Under direct visualization, 0.1 mL of alcohol was injected twice at an approximate depth of 2–3 mm directly into the myocardium beneath the LCX with the help of an insulin delivery needle (6 mm × 26 gauge needle)

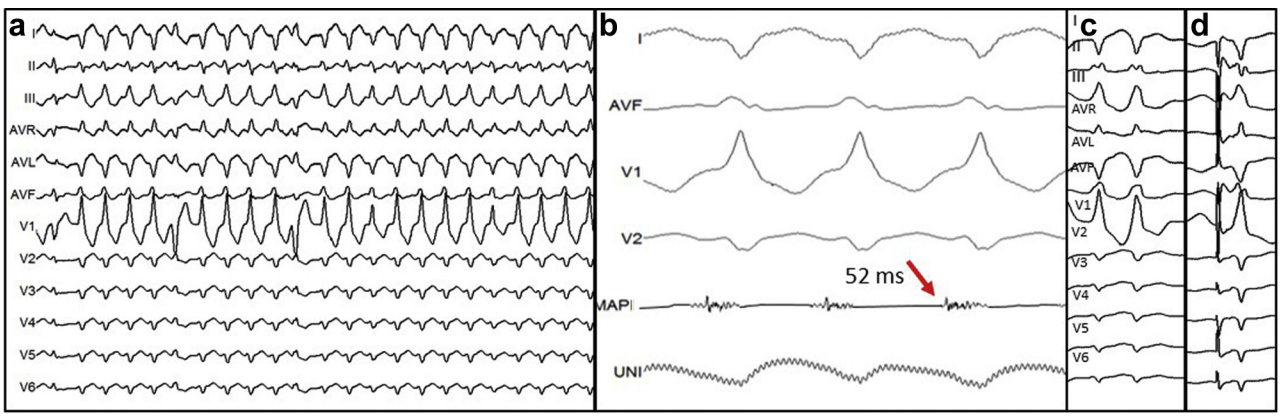

Figure 2 **a:** The 12-lead electrocardiogram (ECG) of clinical ventricular tachycardia (VT) recorded in the operating room (cycle length 440 ms). **b:** Surface ECG of VT showing leads I, AVF, V_1, and V_2 and bipolar electrocardiogram recorded from the mapping catheter during VT. Fractionated late diastolic potentials were recorded preceding QRS onset of VT by 52 ms at the site of successful ablation (*red arrow*). Simultaneous QS morphology was observed in UNI (unipolar) recording. **c:** VT and **d:** pace-map showed good match (22/24) at the site of early signal. MAP = mapping catheter; UNI = unipolar catheter recording.

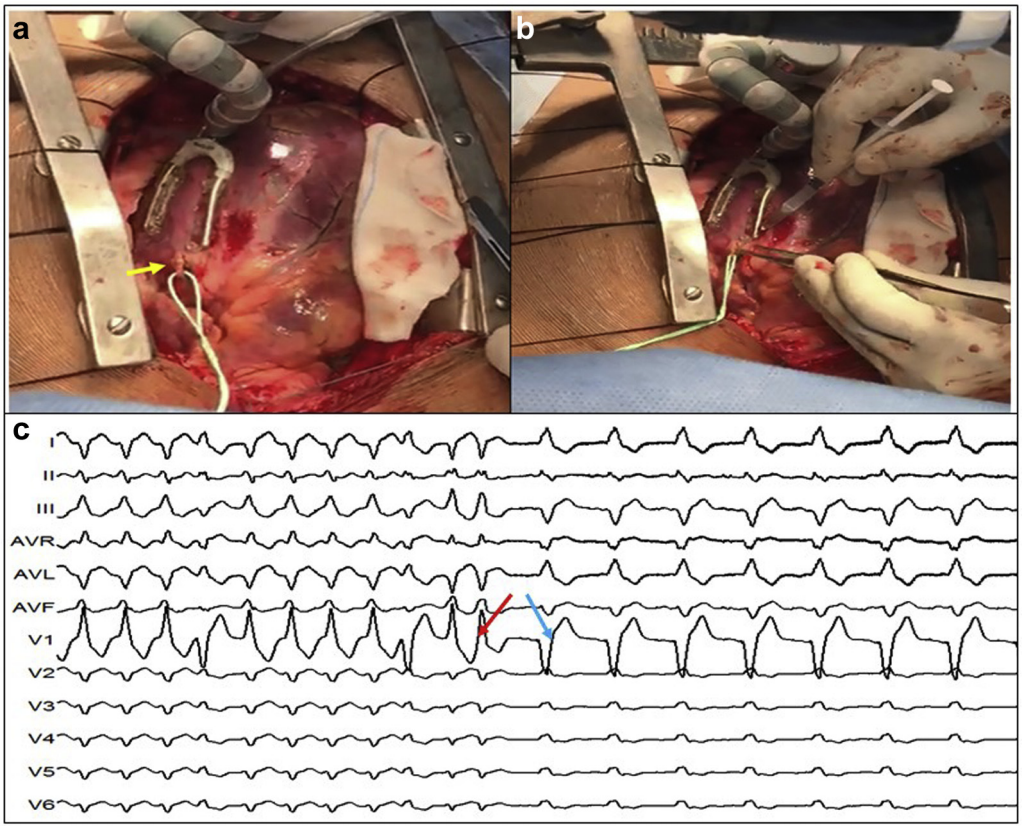

Figure 3 Surgical mapping of ventricular tachycardia (VT) via median sternotomy: **a:** Yellow arrow showing left circumflex coronary artery (LCX) being lifted off the epicardium with the help of tape loop. **b:** Intramyocardial injection of alcohol beneath the LCX with the help of insulin syringe (6 mm × 26 gauge needle). **c:** The 12-lead ECG showing termination of VT following alcohol injection (*red arrow*), followed by paced rhythm (*blue arrow*).

(Figure 3b). VT terminated after 10 seconds of alcohol injection (Figure 3c). Following VT termination, substrate modification was performed around the LCX. Post ablation, no VT was inducible with programmed ventricular stimulation facilitated by isoprenaline. Post procedure, there was no deterioration in LV function or any new regional wall motion abnormality. Coronary angiogram performed after chemical ablation did not reveal any abnormality in the LCX. The patient's overall clinical status improved without any arrhythmic episodes. The patient was event free for 2 years during the follow-up.

Discussion

Surgery was an early mode of treatment for refractory VT. The advent of catheter ablation, and later description of percutaneous epicardial access by Sosa and colleagues,[3] led to the decline in VT surgeries. In an occasional patient, pericardial adhesions owing to prior surgery or inflammation poses difficulty in accessing epicardial space that may call for surgical ablation. More important, the presence of an overlying major coronary artery, phrenic nerve, and thick epicardial fat prevents safe or effective energy delivery from the epicardial surface. In these conditions, surgical access is required to directly visualize the major coronaries and deliver ablation energy. There are also many ancillary methods available in addition to conventional radiofrequency catheter ablation, including bipolar ablation, infusion needle ablation, and alcohol ablation, to manage difficult-to-ablate VTs.[4] Despite these techniques, some patients require surgical VT ablation as a treatment of last resort.

Surgical VT ablation has the advantage of direct visualization of the myocardium and good catheter contact. Contact and stability of the catheter can be further improved by taping the mapping catheter to the surgeon's finger. However, operators may face certain difficulties during surgical VT mapping and ablation. Cryoablation has a risk of coronary artery spasm even after adequate surgical exposure, as illustrated in our case. In a study by Choi and colleagues,[1] cryoablation near the coronaries resulted in transient ST elevation and wall motion abnormality in 50% of the cases. Apart from this, there is also a risk of rapid progression of pre-existing coronary artery disease owing to ablation.[1] Other complications such as steam pops have been reported to occur during radiofrequency ablation.[5] Rarely, accumulation of air in the pericardial cavity during limited-access surgical ablation can cause failure of external shocks to terminate VT owing to increased impedance. This can be overcome by delivery of internal shocks with an implantable cardioverter-defibrillator.[5,6] Importantly, electrophysiologists must be aware of alteration in VT morphology owing to the electrode displacement caused by chest wall retraction and mobilization of the heart during surgery.

Chemical ablation by alcohol is preferred when there is failure of both epicardial and endocardial ablation. In clinical practice, 2 modes of alcohol delivery practiced are transcoronary ethanol ablation (TCEA) and transvenous ethanol ablation. TCEA causes direct chemical injury to cells and ischemic damage to myocardium by causing intravascular thrombosis.[7] This approach can sometimes lead to unintended large myocardial infarct or unrelated injury owing to collaterals or leak of alcohol. In instances of reduced ventricular function, this large-area necrosis may lead to further deterioration in LV function. Furthermore, LV scar produced by chemical ablation may contain surviving myocardium, giving rise to late-onset ventricular arrhythmia. Overall data suggest long-term transcoronary ethanol ablation is suboptimal and should be used judiciously, as in refractory cases of VT.[8] Transvenous ethanol ablation has additional advantages such as avoidance of arterial injury owing to cannulation and less susceptibility to injury in case of alcohol leak.[9]

Direct injection of alcohol into the myocardium for controlling VT was first reported in dogs by Kurita and colleagues.[10] They demonstrated successful treatment of aconite-induced VT. Later, Callans and colleagues[11] reported direct intramural alcohol injection with a 27 gauge needle delivered by a deflectable catheter in the left ventricle of swine. They found that lesion formation following intramural alcohol injection was immediate, and it was confined adjacent to the injection port. Histologic analysis of these lesions revealed homogenous lesions with intramural hemorrhage and contraction band necrosis.[11] In our patient, we opted for direct chemical ablation with alcohol during VT surgery as a bail-out technique to prevent severe coronary spasm. Alcohol, when administered in high concentrations in tissues, dissolves the cell membrane and disrupts the tertiary protein structure, leading to immediate cell lysis.[7] This property explains the immediate termination of VT in our case. Many complications associated with TCEA and transvenous ethanol ablation can be overcome by direct myocardial injection of alcohol. This method can spare the healthy tissue and avoid extensive injury to the myocardium. Addition of iodinated contrast to absolute alcohol or echocardiography will help in assessing the extent of extravasation in the myocardium during alcohol injection.

Conclusion

Direct instillation of alcohol into the myocardium appears to be safe and efficacious in eliminating VT foci in the LV. Cryoablation-induced arterial vasoconstriction resulting in ischemia and hemodynamic instability can be avoided with intramural injection of alcohol during surgical ablation.

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