Epicardial Ablation of Hemodynamically Unstable Ventricular Tachycardia Using CT Imaging, Three-Dimensional Electro-Anatomic Mapping of the Coronary Arteries and the Impella Left Ventricular Assist Device

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ABSTRACT. This case report describes successful epicardial ablation of a hemodynamically unstable ventricular tachycardia (VT) in a 19-year old man with a prior unsuccessful attempt using endocardial approach. A pre-procedure contrast-enhanced cardiac computer tomography showed hypokinesis and thinning of the apical lateral wall with loss of epicardial myocardial layer replaced by hypodense tissue consistent with fat. This finding strongly suggested epicardial origin of the VT and, thus, epicardial access was obtained at the beginning of the case. The use of a percutaneous left ventricular assist device (Impella 2.5) allowed detailed mapping of otherwise hypotensive VT. Electro-anatomic reconstruction of the coronary arteries was used to avoid applications of radio-frequency energy in close proximity to these vessels.

KEYWORDS. ventricular tachycardia, epicardial catheter ablation, left ventricular assist device, electro-anatomic mapping, cardiac computer tomography.

Introduction

Hemodynamically unstable ventricular tachycardia (VT) presents a clinical challenge. Mapping is best performed during the arrhythmia of interest, but unfortunately this may result in deterioration of the patient, require multiple defibrillation, and may ultimately result in abandonment of the procedure itself. Scar mapping and ablation can be performed during normal sinus rhythm, but this approach may not be always feasible if the VT is epicardial in origin because of the risk of damage to the coronary arteries or to the phrenic nerve. The following case illustrates how successful ablation of hemodynamically unstable epicardial VT was facilitated by application of novel modalities, namely computed tomography (CT) of the heart, use of a temporary left ventricular assist device (LVAD), and electro-anatomic mapping of the coronary arteries.

Background

A 19-year-old man presented to his local medical facility after experiencing an unwitnessed “seizure” and was found to have monomorphic VT at a rate of 220 bpm, which required electrical cardioversion (Figure 1). He was subsequently transferred to our hospital. An echocardiogram and cardiac magnetic resonance imaging (MRI) showed moderately reduced left ventricular (LV) systolic function (LV ejection fraction (EF) 40–45% by echo, 36% by MRI), with no other significant structural abnormalities or evidence of delayed enhancement. He next underwent an electrophysiology (EP) study and
attempted VT ablation using conscious sedation. Monomorphic VT (right bundle branch block superior axis)—identical to his clinical VT morphology and cycle length (CL)—was reproducibly induced with programmed stimulation. The VT was accompanied by an abrupt decline in systolic blood pressure (BP) to 60–80 mmHg. Endocardial LV voltage mapping in sinus rhythm revealed no areas with abnormal electrograms. However, pace mapping in the apical lateral wall closely matched the VT QRS pattern. Detailed VT activation and entrainment mapping was not feasible due to hemodynamic instability. Limited local activation mapping in the area where pace mapping best reproduced the VT QRS pattern showed local activation preceding the onset of the QRS complex by 20–30 ms. Radiofrequency (RF) ablation was attempted in this area but could not be continued because of recurrent acceleration of VT and its degeneration to VF requiring multiple defibrillations. It was felt that further RF ablation would be unsafe, and the procedure was aborted. He subsequently underwent implantable cardioverter-defibrillator (ICD) implantation prior to discharge.

Two years later, after a period of relative quiescence, the patient presented with recurrent VT requiring multiple ICD shocks despite therapy with sotalol. A second VT ablation attempt was thus pursued. A pre-procedure contrast-enhanced cardiac CT showed hypokinesis and thinning of the apical lateral wall with loss of epicardial myocardial layer replaced by hypodense tissue consistent with fat (–60 Hounsfield units) (Figure 2). The right ventricle was normal. This finding as well as the prior unfruitful experience with endocardial mapping and ablation strongly suggested an epicardial origin of his arrhythmia.

**VT ablation**

The procedure was performed under general anesthesia. Following epicardial and atrial transseptal access, coronary angiography of the left coronary system was performed (Figure 3). The left anterior descending artery (LAD) and diagonal branches were determined to be overlying the area of suspected epicardial VT substrate in the apical/lateral wall. Using an angioplasty guidewire (BMW Universal, Abbot, Santa Clara, CA) and a...
1.5 mm × 20 mm over-the-wire angioplasty balloon (Apex OTW, Boston Scientific (St. Paul, MN)), three-dimensional (3D) reconstruction of the LAD and diagonal branches was obtained (Figure 3). For this purpose, the guidewire was advanced via the balloon to expose 3–4 mm of the distal tip, which served as an electrode. The proximal end of the guidewire was connected to an electrophysiology (EP) recording system (CardioLab v6.5, GE Medical System, Waukesha, WI) and an electro-anatomic mapping system (EnSite Velocity, St Jude Medical (St. Paul, MN)) using a unipolar configuration. A 6F quadripolar diagnostic EP catheter placed in the inferior vena cava (IVC) was used as an indifferent electrode. The guidewire was initially advanced to the most distal part of each vessel and then slowly dragged back to create the corresponding geometry. A total of 40 ml of contrast was used for coronary angiography and electro-anatomic mapping of the coronary arteries.

Following removal of the guidewire, balloon, and angiography catheter, a LVAD (Impella 2.5, Abiomed, Danvers, MA) was placed retrogradely across the aortic valve to enable mapping of hemodynamically unstable VT (Figure 4). This device is a motor-driven micro-axial pump mounted on the distal portion of a 12F catheter. It pumps blood from the left ventricle directly to the aorta with forward flow of up to 2.5 l/min.

Using an irrigated tip catheter (Thermocool, Biosense Webster (Diamond Bar, CA)) and the electro-anatomic mapping system, geometry and endocardial voltage mapping of the LV were performed. Similar to his initial VT ablation 2 years previously, the endocardium of the left ventricle showed no areas of abnormal electrograms (e.g. low amplitude <1.5 mV, fractionated, or isolated high frequency late potentials). His clinical VT with a cycle length (C) of 360 ms was reproducibly induced with programmed stimulation from the right ventricle. The patient remained stable with a mean BP of 70–80 mm Hg with the Impella running. The earliest bipolar electrograms noted in the lateral/apical wall during VT showed manifest entrainment with the post-pacing interval close to the VT CL suggesting an outer loop. Ablation at this location was attempted with no change in the VT.

Epigardial mapping was then performed. The ablation catheter was placed into the pericardium via a steerable sheath used for better maneuverability and support (40 cm, Agilis, St. Jude Medical, St. Paul, MN). Sinus rhythm voltage mapping demonstrated a large area of fractionated low-amplitude electrograms and isolated high-frequency late potentials overlying the lateral/apical wall corresponding to the area of abnormal substrate on the CT images. Pace-mapping in this region matched the clinical VT (11/12 leads matching) at a number of pacing sites, although consistent capture was not feasible over...
most of the attempted pacing sites. The VT described above was easily induced again. With the Impella device in place and running, the patient’s mean arterial pressure despite remaining in VT was ~60–70 mmHg. Entrainment was not feasible due to lack of stable capture at maximum output.

Ablation (Figure 5) was guided by mid-diastolic potentials (Figure 6) during the tachycardia, using the aforementioned electro-anatomic map of the coronary arteries to avoid ablating directly near them (Figure 3). Before each RF application, high output pacing was performed to ensure lack of phrenic nerve capture (and thus avoid ablating this structure). The VT was terminated on a number of occasions during ablation and subsequently reinduced at progressively longer CLs (from 360 ms to 450 ms). A total of 26 lesions were required to render the arrhythmia non-inducible. Four additional ablations were performed targeting isolated high-frequency late potentials in sinus rhythm. Follow-up coronary angiography done at the end of the procedure showed no visible damage to coronary arteries.

Discussion

This case report highlights three innovative technologies used to facilitate successful ablation of a hemodynamically unstable VT. First, pre-ablation contrast-enhanced cardiac CT was used to identify abnormal myocardium prior to ablation. Although delayed-enhanced MRI remains the gold standard for characterizing myocardial pathology, it is generally contraindicated in patients with an ICD. In addition, metal artifacts caused by the ICD may significantly affect quality of cardiac MRI images and therefore preclude their meaningful interpretation. Contrast-enhanced cardiac CT has recently emerged as a possible alternative imaging technology to gadolinium-enhanced MRI in ICD patients. In our case, contrast-enhanced cardiac CT identified an area of abnormal epicardial substrate in the apical-lateral LV wall, which ultimately was the region where the VT was successfully ablated. The epicardial origin of the VT was thus strongly considered prior to the case because of this finding and epicardial access was obtained at the beginning of the procedure.

The exact etiology for the abnormal epicardial substrate is unclear, but tissue characterization by CT suggests a loss of myocardium with fat tissue replacement which would be consistent with arrhythmogenic right ventricular cardiomyopathy (ARVC), prior myocarditis or infarction. Our patient had normal RV size, function, and appearance on CT and no prior history of myocarditis or infarction. However, a multicenter autopsy study of ARVC showed LV involvement in up to 76% of cases, and isolated LV arrhythmogenic cardiomyopathy has been described. Myocardial biopsy was not performed in this case.

Second, the use of the percutaneous LVAD (Impella) enabled activation mapping of the hemodynamically unstable VT. Friedman et al previously reported feasibility of using percutaneous circulatory support in the EP laboratory for endocardial and epicardial mapping of poorly tolerated VT. A more recent report described the use of the Impella device for successful mapping and ablation of hemodynamically unstable VT.

Because of symptomatic hypotensive response (systolic BP of 60–80 mmHg), only brief VT mapping (for a few minutes) could be performed during the first attempt at ablation. Of note, the first procedure was done under conscious sedation while general anesthesia was utilized during the second procedure. In our experience, even well-tolerated VT in a conscious patient frequently becomes hemodynamically unstable with the use of general anesthesia. Therefore, more severe hemodynamic derangements would have been expected during VT if the circulatory support had not been used during the second ablation attempt.

Mean BP measurements were used as a surrogate of adequacy of circulatory support provided by the Impella device. Because of continuous flow patterns, cyclic variations in the BP are attenuated. Although the optimal BP range remains unknown, our limited experience with the pump in VT patients (4 cases, unpublished data) suggests that a mean BP of 60–80 mm Hg does not appear to cause clinically evident end organ damage. However, these BP measurements may not be applicable to all populations of patients, particularly elderly patients with extensive vascular disease or pre-existing organ insufficiency. Also, optimal tissue perfusion markers warrant further study.

Potential complications that may occur with the use of the Impella device include aortic regurgitation, severe hemolysis, or vascular injury. None was observed in the present case.

VT mapping and focused ablation is critical during epicardial procedures. In contrast to the endocardial approach, an extensive substrate-guided ablation strategy with deployment of linear lesions in sinus rhythm

Figure 5: Endocardial electro-anatomic left ventricular (LV) geometry with color-coded voltage map, coronary artery map, and epicardial ablation sites. The purple color on endocardial LV geometry represents voltage areas above 1.5 mV. The overlying coronary anatomy was reconstructed with electro-anatomic mapping and is represented by orange, teal, and salmon spheres. Epicardial ablation sites are shown as white spheres.
may not be feasible in the epicardium due to multiple structures, such as the coronary arteries, phrenic nerve, and epicardial fat, that may be damaged or impede adequate ablation. In addition, the use of pace- or entrainment mapping techniques is commonly limited in the epicardium due to difficulty in achieving a stable catheter position and myocardial capture.

Finally, 3D electro-anatomic reconstruction of the coronary arteries was used to avoid applications of RF energy in close proximity to these vessels. Although, coronary angiography is a standard approach to identify spatial relation of the ablation sites to the coronary vessels during epicardial ablation procedures, it is well recognized that two-dimensional fluoroscopic images are suboptimal for accurate navigation in the 3D space. Furthermore, when multiple epicardial areas are targeted for ablation, repeated coronary angiograms may be impractical and can significantly increase radiation exposure. Although registration of CT images of the coronary arteries into 3D electro-anatomic mapping systems is currently available and is a potential alternative to repeat coronary angiograms to guide epicardial ablation, repeated and reliable image registration remains a significant challenge. Electro-anatomic mapping has been shown to be a reliable tool for precise catheter placement in 3D space. A potential limitation of all available 3D mapping systems which may affect the accuracy of lesion placement is catheter movement related to the respiration or cardiac contraction. The 3D mapping system used in this case (EnSite Velocity, St Jude Medical) allows to compensate for respiratory catheter motions by a specifically designed “respiration compensation” feature which is based on serial transthoracic impedance measurements. General anesthesia facilitates effective use of this feature by providing a uniform respiration pattern. The system uses a high-frequency sampling rate of anatomical points which minimizes the effect of cardiac motion on endocardial geometry and obviate the need for cardiac gating during endocardial mapping. It is not clear whether a cardiac gating feature would be useful for epicardial ablation since the system is not designed for epicardial procedures. The impact of cardiac motion on catheter position in the pericardial space appears to be less prominent than the endocardial surface. Nevertheless, catheter position in relation to the geometry of the coronary arteries was carefully observed before each application of RF energy to ensure that there was a safe distance (>0.5 cm) between them at any point during the cardiac or respiratory cycles.

This case suggests that the use of electro-anatomic reconstruction of the coronary arteries for epicardial ablation is feasible. However, the clinical utility and safety of this technique requires further study.

In summary, our report demonstrates the symbiosis of non-conventional technologies including cardiac CT, LVAD, and electro-anatomic mapping of coronary

**Figure 6:** An example of mid-diastolic potentials recorded on the epicardium (arrows) during ventricular tachycardia.
arteries, coupled with interspecialty collaboration to ultimately result in successful ablation of an otherwise unmappable epicardial VT.

References