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Review Article

New Trends in High Risk Ventricular Tachycardia Catheter Ablation

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ABSTRACT

Ventricular tachycardia (VT) is one of the major causes of sudden cardiac death (SCD). In general, VT could be managed with antiarrhythmic drugs (AADs) therapy, catheter ablation and implantable cardioverter defibrillators (ICD). While the AADs therapy and catheter ablation have been shown to reduce the recurrence of VT, only the ICD therapy is effective in aborting SCD. The recently published VANISH trial reveals that VT catheter ablation significantly decreases the rate of death, VT storm and appropriate ICD shock comparing with an escalation of AADs therapy for ischemic cardiomyopathy (ICM). However, the mapping strategies and feasibility of VT catheter ablation are often limited by the hemodynamically intolerant VT. Substrate modification strategy and percutaneous left ventricular assist device (pLVAD) are often used to overcome the hemodynamic intolerance. So far there are no large-scale randomized clinical trials comparing different mapping strategies in the setting of hemodynamically unstable VT, specifically when it comes to risk stratification for patients with hemodynamic instability. The aim of the present article is to systemically review different VT mapping strategies, the role of pLVAD in hemodynamically intolerant VT ablation with a special consideration of high risk VT.

Key words: Ventricular tachycardia, Left ventricle thrombus, Catheter ablation, Hemodynamically intolerant, Mapping, Percutaneous left ventricular assist device.

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INTRODUCTION

VT is one of the major causes of SCD, which leads to about 356,500 deaths annually in the United States.[1] In general, there are three options in VT management, AADs therapy, VT catheter ablation and ICD implantation.

ICDs are still the first line choice for primary and secondary prevention of SCD, especially in population with structural heart disease.[2] The caveat is that ICDs play no role in VT recurrence prevention. ICD shocks decrease life quality with psychological stress.[3,4] In addition, recently a link has been demonstrated between repeated ICD shocks and heart failure admission and higher mortality. VT recurrence rate is about 40% to 60% in patients who receive an ICD for secondary SCD prevention after a spontaneous sustained VT.[5] 20% patients who receive an ICD for primary SCD prevention will have at least one VT within 3-5 year following the device implantation.[6]

In addition to the adverse effects and narrow therapeutic window, lack of survival benefit and suboptimal outcomes on ICD shock reduction have been observed in AADs therapy.[7,8] VANISH trial reveals inferior mortality reduction, VT storm and appropriate ICD shock prevention of ADDs comparing with VT catheter ablation in ICM.[9]

With the capacity to significantly reduce VT burden, VT catheter ablation has become a promising treatment modality of VT over the past decade.[10-13] The VT free rate within the first 2 years of VT ablation ranges between 50% and 75% in population with structurally abnormal heart, either from ischemic cardiomyopathy (ICM) or non-ischemic cardiomyopathy (NICM).[14]

Hemodynamically intolerant VTs occur in a majority of patient population with structurally abnormal heart diseases.[15] Depressed left ventricular systolic function in either ICM or NICM patients, along with potential hypotension induced by general anesthesia, makes this population more vulnerable to hemodynamic collapse, fluid overload and end-organ hypoperfusion.[15] As a result, more than two thirds of patients cannot tolerate extensive activation and entrainment mapping for the purpose of VT ablation.[16] The hemodynamically intolerant VT catheter ablation is usually performed under general anesthesia and with substrate and pace mapping combination in sinus or paced rhythm.[17-19]

Mechanical circulatory support (MCS) devices have been used in advanced heart failure, cardiogenic shock and refractory VT storm management,[20,21] however, a paucity of data on these devices during hemodynamically intolerant VT mapping and ablation makes the utilization of these devices, especially pLVAD, less widely accepted.

This review will focus on the current status of different VT mapping strategies, new ablation techniques, risk stratification of peri-procedural acute hemodynamic decompensation (AHD), pLVAD safety and outcome, pLVAD comparison and other noninvasive choices in hemodynamically intolerant VT ablation. We will also discuss the high-risk VT ablation experience at our institute at the end of this article.

Mapping Strategies

Various mapping strategies can be used separately or in combination to achieve optimal outcomes and safety of VT ablation. Activation and entrainment mapping are ideal for stable VT as they can precisely identify the critical circuit, however are fraught with limitations in hemodynamically unstable or non-inducible VT.[16] For these special scenarios, substrate and scar mapping is the only way to localize the arrhythmogenic substrates in sinus or paced rhythm[17,18,22] and address the VT. The caveat, however, is that substrate mapping does not always identify the fractionated and/or late potentials responsible for the clinical VT, particularly in NICM[23] hence being inefficient and potentially leading to excessive ablation otherwise unnecessary. In those situations, one has to consider conventional mapping approaches while employing pLVAD in order to mitigate the hemodynamic compromise.

Standard Mapping techniques

Activation mapping is the ideal strategy to localize focal VTs in hemodynamically stable focal VT. It identifies either the earliest possible signal or signal progression around the macro-reentrant circuit during VT by

comparing the intracardiac electrograms recorded with a roving multipolar catheter.[24]

Entrainment mapping is used to localize macro-reentrant circuits with an excitable gap, which is confirmed by the ability to entrain VT with pacing.[24,25] By continuously resetting the reentry circuit, the QRS morphology and postpacing intervals are analyzed to localize the macro-reentrant circuits.

Similar to activation mapping, pace mapping is ideal to identify either a focal VT or the VT exit site in a reentrant VT while seeking a paced QRS complex that is identical to the spontaneous ventricular tachycardia.

Substrate Mapping

Substrate mapping focuses on labeling scar as well as the fractionated abnormal potentials recorded in normal sinus rhythm. While these techniques are predominantly used in hemodynamically unstable VTs, there seems to be a surge of interest in utilizing this approach in all patients.

There are several targets during substrate mapping, including late potentials (LPs), scar de-channeling, local abnormal ventricular activities (LAVAs), core isolation (CI) and homogenization of the scar.[26]

First concept, LPs are thought to help to localize the slow conducting channels that cause VT. LPs are defined as any type of electrograms with a duration that extended beyond the end of the surface QRS.[27] Cassidy *et al.* and Miller *et al.* originally demonstrated that ablation of LPs was an effective strategy for VT ablation.[27-29] Conversely, Nakahara *et al.* showed that LPs ablation was less successful in patient with NICM compared to ICM, with 82% VT free 10 ± 12 months following ablation in ICM group versus 50% VT free 10 ± 13 months following ablation in NICM group.[13]

The second concept of scar de-channeling, is designed to eliminate all identified conducting channels (CCs) by ablation at the CC entrance and was proposed by Berruezo and colleagues.[30,31] It was found by Arenal *et al.* that conducting channels (CC) represented areas of slow conduction that could be identified in 75% of patients with sustained monomorphic VT.[32] In a study of 101 patients with LV scar–related VT, Berruezo *et al.* demonstrated that scar de-channeling alone resulted in low recurrence and mortality rates in more than half of patients despite the limited ablation extent being required. Residual inducible VT ablation improved acute results, but patients who required it had worse outcomes. Recurrences were mainly related to incomplete CC electrogram elimination.[30] The same group also proved that combined endocardial and epicardial ablation in Arrhythmogenic right ventricular dysplasia (ARVD) by incorporating scar de-channeling achieved a very good short and midterm success rate.[31]

The third concept introduced by Jais and colleagues defines Local abnormal ventricular activities (LAVAs) as sharp high-frequency ventricular potentials, possibly of low amplitude.[33] In their prospective study of 70 patients with VT and structurally abnormal ventricle, conventional mapping was performed in sinus rhythm in all and a high-density mapping catheter was used in the endocardium and epicardium. LAVAs were recorded in 67 patients (95.7%). Catheter ablation was performed targeting LAVAs, which successfully abolished or dissociated in 47 of 67 patients (70.1%). In multivariate analysis, LAVA elimination was independently associated with a reduction in recurrent VT or death during long-term follow-up.

Core isolation (CI) focuses on a circumferential ablation around all critical VT circuit elements, which was described by Tzou and colleagues in 2015 as a novel strategy with a discrete and measurable end point beyond VT inducibility to treat patients with multiple or un-mappable VTs.[34] In their original study, CI was performed incorporating putative isthmus and early exit site based on standard criteria. If VT was noninducible, the dense scar (<0.5 mV) region was isolated. 44 patients were included, among which CI was achieved in 37 (84%) and led to better VT-free survival. Additional substrate modification was performed in 27 (61%) and epicardial radiofrequency ablation was performed in 4 (9%) patients.[34]

Lastly, homogenization of scar was introduced by the Di Biase *et al.* with a non-randomized prospective study enrolled 92 consecutive patients with ICM and electrical storm. The goal of this ablation strategy is to cover the entire scar with ablation lesions targeting abnormal electrograms, including the dense scar and border zone. By homogenizing the scar, one extensively ablates the triggers from within the scar areas that are assumed to be inert during traditional mapping. During a mean follow-up of 25 ± 10 months, the recurrence rate of any VT was 47% in patients undergoing traditional ablation and 19% (8 of 43 patients) in patients undergoing scar homogenization.[35] Subsequently, in 2015, in VISTA trial, a randomized controlled trial, these authors concluded that an extensive substrate-based ablation approach was superior to conventional ablation targeting only clinical and stable VTs in patients with ICM presenting with tolerated VT.[36] Combined incidence of rehospitalization and mortality was significantly lower with substrate modification while periprocedural complications were similar in both groups.

These non-randomized studies presented in this substrate mapping section suggest that a substrate based ablation is superior to the traditional ablation of clinical and hemodynamically stable VT.[33,35,37,38] Based on the above published data, it appears that VT induction and mapping before substrate ablation prolongs the procedure, radiation exposure and the need for electrical cardioversion without improving acute results and long- term ablation outcomes. The concern for "too much" ablation during substrate mapping while lacking the absolute confirmation of effectiveness - cessation of tachycardia while ablating, raises the question of hemodynamic support for selected cases.

New Ablation Techniques

Unipolar radiofrequency (RF) is the most widely used technique for VT ablation. However, the success rate is limited if the VT substrate is deep intramurally, where neither endocardial nor epicardial access can reach. Several novel techniques have been developed to complement the traditional RF ablation, including transcoronary ethanol ablation, coronary coil embolization, bipolar RF ablation and stereotactic ablative radiosurgery.[39]

Transcoronary Ethanol Ablation (TCEA)

Alcohol is injected directly into the coronary artery to cause both primary chemical necrotic injury and secondary ischemic injury by vascular damage. The injured myocardium later becomes permanent scar and replaces the VT substrates.[40] It is reserved for deep intramural VT substrates that cannot be accessed with either an endocardial or epicardial approach. The efficacy of this technique may be limited by the difficulties to identify the target coronary artery, the collaterals of the coronary artery. Serious complication might happen, including complete heart block, early ventricular arrhythmia, distant myocardial infarction from coronary complication and even death.[39]

Coronary Coil Embolization

Instead of injection alcohol, coils are deployed to occlude the coronary artery and subsequently cause myocardium necrosis after identifying the target artery supplying the VT substrate area.[41] This technique causes relatively controlled myocardium injury compared to the unpredictable injury by TCAE, thus relative lower complication rate.

Needle Ablation

The RF energy can be delivered via a specialized catheter that incorporates an extendable/retractable injection needle.[42] Due to the extendable/

retractable injection needle, the catheter can reach the deep intramural myocardium that otherwise inaccessible with conventional RF ablation. However, despite the promising effectiveness, a precise mapping is necessary for the procedure. Life threatening complications, particular cardiac perforation and tamponade, is not trivial. In addition to RF delivered by these needle catheters, a direct intramural alcohol injection into the myocardium is also feasible to cause deep intramural lesion, as demonstrated by animal models.[43]

Bipolar Radiofrequency Ablation

In conventional unipolar RF, there is large current density at the catheter tip, which causes tissue heating at the catheter-myocardium interface and conductive heating of deeper tissues. However, the conductive heating to deep tissue is limited by the amount of energy feasible. Bipolar RF ablation can be used to achieve deep tissue ablation. In a bipolar RF ablation system, RF current flows between the 2 ablation catheters in close proximity, which are usually placed on the opposite of the myocardium. In one animal study, it was found that bipolar RF ablation was more likely to achieve transmural lesions.[44] Good acute but mixed long term outcomes have be reported in refractory septal VT (ischemic and nonischemic), free-wall VT (ischemic) and outflow tract VT (idiopathic) ablation, with bipolar ablation system consisting of irrigated 3.5 mm ablation catheters and up to 40 W of energy.[45-47] However, no bipolar RF ablation cables or switches are yet commercially available on market.

Stereotactic Ablative Radiosurgery (SABR)

SABR, widely used in cancer radiotherapy, can deliver high dose ionizing radiation to a small localized tissue and cause radiation induced necrosis in the target tissue.[48] The nature of non-invasiveness of SABR makes it an ideal mode of ablation in patients with comorbidities who cannot tolerate invasive treatment.[49] In 2015, Loo *et al.* reported the first clinical application of SABR in VT treatment.[49] No major complications have been reported yet, but considering the very early stage of this technology, more investigation is needed to ascertain its safety.

Hemodynamically Intolerant VT Ablation

Risk Stratification of Periprocedural Acute Hemodynamic Decompensation (AHD)

As mentioned earlier, more than two thirds patients cannot tolerate VT catheter ablation with activation and entrainment mapping only. The hemodynamically intolerant VT catheter ablation is usually performed with substrate modification approach. However, even with a substrate modification approach, some patients are still vulnerable to periprocedural AHD.[50] Moreover, depressed left ventricular systolic dysfunction, found in the majority of VT patients, along with potential hypotension induced by general anesthesia, makes this population more vulnerable to AHD. With these scenarios, pLVAD might be of a valuable tool to achieve successful mapping, ablation and decrease procedural complications. It is neither reasonable nor practical to insert pLVAD for all VT ablation regardless of the hemodynamic status, given the cost and inherent complications. So far no widely accepted algorithm is available to identify patients with high risk of AHD.

Santangeli *et al.* proposed the PAAINESD pilot risk score (P: pulmonary disease- COPD, 5 points; A: age > 60, 3 points; A: Anesthesia, general, 4 points; I: ischemic cardiomyopathy, 6 points; N: NYHA class III or IV, 6 points; E: Ejection fraction <25%, 3 points; S: storm VT, 5 points; D: diabetes mellitus, 3 points) to predict the occurrence of periprocedural AHD during scar-related VT RF ablation.[51]

The PAAINESD score ranges from a minimum of 0 points to a maximum of 36 points. The risk of AHD proportionally increased per increasing risk score tertile (1st tertile: 1% risk of AHD; 2nd tertile: 6% risk of AHD; 3rd tertile: 24% risk of AHD).

The Benefits of pLVAD

So far there is only limited data to address the role of pLVAD in hemodynamically intolerant VT ablation. All available data is from either case reports, or observational studies in a single or multiple centers.[52-57]

The first case of VT ablation with pLVAD was reported by Friedman *et al.* in 2007.[54] A patient with hemodynamically intolerant VT induced during mapping was stabilized with Tandem Heart and underwent both epicardial and endocardial mapping (1h and 45 min) and had a successful ablation.

Aryana *et al.* evaluated the pLVAD effects on unstable VT ablation procedure and clinical outcomes.[58] While pLVAD support did not affect VT recurrence, it was associated with a lower composite endpoint of 30-day rehospitalization, redo-VT ablation, recurrent ICD therapies and 3-month mortality. In this nonrandomized retrospective study, 68 consecutive unstable, scar-mediated endocardial and/or epicardial VT ablation procedures performed in 63 patients were evaluated. During VT mapping and ablation, hemodynamic support was provided by intravenous inotropes with a pLVAD (n = 34) or without a pLVAD (n = 34). VT was sustained longer with a pLVAD (27.4 ± 18.7 mins) than without a pLVAD (5.3 ± 3.6 mins). A higher number of VTs were terminated during ablation with a pLVAD (1.2 ± 0.9 per procedure) than without a pLVAD (0.4 ± 0.6 per procedure). Total radiofrequency ablation time was shorter with a pLVAD (53 ± 30 mins) than without a pLVAD (68 ± 33 mins), but with similar procedural success rates (71% for both pLVAD and control groups). Although during 19 ± 12 months of follow-up VT recurrence did not differ between pLVAD (26%) and control (41%), the composite endpoint of 30-day rehospitalization, redo-VT ablation, recurrent ICD therapies and 3-month mortality was lower with a pLVAD (12%) than without a pLVAD (35%).

Shigeki Kusa *et al.* investigated whether the acute hemodynamic benefits from pLVAD during unstable VT ablation could translate into favorable clinical outcome.[59] The single center retrospective study consisted of 194 patients (109 pLVAD and 85 non-pLVAD). The pLVAD group more often had dilated cardiomyopathy (33% versus 13%), NYHA HF class ≥III (51% versus 25%), lower left ventricular ejection fractions (26±10% versus 39±16%) and electrical storm (49% versus 34%). Procedure times (422±112 versus 330±92 mins), post ablation VT inducibility (20% versus 7%) and length of subsequent hospitalization (median 6 versus 4 days) were all higher in the pLVAD group. During median follow-up of 215 days, the primary end point (recurrent VT, heart transplantation or death) occurred in 36% of the pLVAD versus 26% of the non-pLVAD groups. After propensity matching for differences between groups, no differences were seen between groups for both acute procedural outcomes and the primary end point. Despite the worse clinical status of the patients selected for pLVAD support, clinical outcomes were better than expected and were similar to healthier patients not receiving hemodynamic support.

Nilesh Mathuria *et al.* assessed the outcomes of preemptive and rescue use of pLVAD during VT ablation in patients with ICM and NICM. They reported that there was no significant difference in 30-day mortality or long-term freedom of VT between the pre-emptive and non-pLVAD groups. 93 patients underwent VT ablation. Three groups were compared: (1) Rescue group (n = 12), patients who required emergent pLVAD insertion due to hemodynamic collapse during VT ablation, (2) Preemptive group (n = 24), patients who had pre-ablation pLVAD insertion and (3) Non-pLVAD group (n = 57), patients who did not undergo pLVAD insertion. Thirty-day mortality was higher in the rescue group compared to the preemptive group (58 vs. 4 %) and non-pLVAD (58 vs. 3 %) group.[60]

The Comparison of Different pLVADs

The pLVADs that are most commonly used in hemodynamically intolerant VT ablation are as following, the intra-aortic balloon pump (IABP), the Impella microcirculatory axial blood flow pump (Abiomed, Inc., Danvers, MA, USA), the percutaneous ventricular assist device (pVAD) (Tandem-Heart, Cardiac Assist, Inc., Pittsburg, PA, USA), the cardiopulmonary support (CPS) with bypass pump and venoarterial extracorporeal membrane oxygenation (VA-ECMO).[61]

Direct comparison of these pLAVDs for the treatment of cardiogenic shock or refractory hemodynamically intolerant VT in a porcine model was performed by Ostadal *et al.* in 2012.[62] The major finding revealed the significant difference in the hemodynamic efficacy of the currently available pLVADs, favoring the RA-Ao system (ECMO), followed by the LA-Ao system (TandemHeart). The least efficacious appeared to be LV-Ao system (Impella 2.5). However, even the LV-Ao system allowed short time blood pressure support during VFib when norepinephrine at 0.1 μg/kg/minute was added.

Lü *et al.* first compared, in a clinical trial, the hemodynamic support effects among the Impella 2.5, peripheral cardiopulmonary bypass (CPB) and durable continuous-flow left ventricular assist device (CF-LVAD) (HeartMate II, Thoratec, Pleasanton, California, USA) in unstable VT ablation and found that peripheral CPB and implantable LVAD provided adequate hemodynamic support for successful ablation. Impella® 2.5, on the other hand, was associated with increased risk of complications and may not provide sufficient hemodynamic support in some cases.[63] 16 consecutive patients who underwent ablation of hemodynamically unstable VT were included in this study. In the Impella and CPB groups, mean time under hemodynamic support was 185 ± 86 min and time in VT was 78 ± 36 min. Clinical VT could be terminated at least once by ablation in all patients except 1 case with Impella due to hemodynamic instability. Periprocedural complications included hemolysis in 1 patient with Impella and surgical intervention for percutaneous Impella placement problems in another 2.

Reddy *et al.* compared IABP with the other pLVAD and concluded that Impella and TandemHeart use in VT ablation facilitated extensive activation mapping of several unstable VTs and required fewer rescue shocks during the procedure when compared with using IABP.[64] This was a multicenter, observational study from a prospective registry including all consecutive patients (N=66) undergoing VT ablation with a pLVAD in 6 centers in the United States. Patients with IABP (IABP group; N = 22) were compared with patients with either an Impella or a TandemHeart device (non-IABP group; N = 44). In non-IABP group (1) more patients could undergo entrainment/activation mapping (82% versus 59%), (2) more number of unstable VTs could be mapped and ablated per patient (1.05 ± 0.78 versus 0.32 ± 0.48), (3) more number of VTs could be terminated by ablation (1.59 ± 1.0 versus 0.91 ± 0.81) and (4) fewer VTs were terminated with rescue shocks (1.9 ± 2.2 versus 3.0 ± 1.5) when compared with IABP group. Complications of the procedure trended to be more in the non-IABP group when compared with those in the IABP group (32% versus 14%). Intermediate term outcomes (mortality and VT recurrence) during 12 ± 5 month follow- up were not different between both groups.

The latest retrospective result of ECMO hemodynamic support during high risk VT ablation was reported by Baratto *et al.* in 2016.[65] The authors concluded that unstable VT ablation could be safely performed with ECMO support, with a cohort of 64 patients undergoing 74 unstable VT ablation with ECMO. At least one VT was terminated in 81% of procedures with baseline inducible VT and VT noninducibility was achieved in 69%. Acute heart failure occurred in 5 patients: 3 underwent emergency heart transplantation, 1 had left ventricular assist device (LVAD) implantation and 1 patient eventually died because of subsequent mesenteric ischemia. After a median follow-up of 21 months (13-28 months), VT recurrence was 33%; overall survival was 56 out of 64 patients (88%). As previously shown, this study also confirmed that preemptive placement of patients on hemodynamic support is a superior approach compared to initiating pLVAD as an emergency.

Other Measures in Hemodynamic Intolerant VT Ablation

Ventricular Synchronized Triggered Atrial Pacing (VSTAP)

VSTAP was initially described by Hamer *et al.* in 8 patients with VT induced during electrophysiology testing.[66] During the induced VTs, these patients were put on synchronized 1:1 triggered atrial pacing (atrium paced, ventricle sensed and triggered mode). The ventriculoatrial coupling interval was adjusted to produce a maximal blood pressure response and the optimal interval was observed to be between 60% and 73% of the RR interval. The mean arterial blood pressure in their series increased from 79 ± 14 mmHg during VTs to 98 ± 12 mmHg during VT plus VSTAP. Evidence from pressure recordings suggested that optimal atrial pacing resulted in atrial contraction in early left ventricular diastole. Thus, appropriately timed atrial pacing during VTs can result in significant increases in blood pressure and a consistent increase in cardiac index.

VT RF ablation in the presence of LV thrombus

One of the common complications of myocardial infarction is LV thrombus, especially among those with large anterior wall STEMI leading to apical akinesis or aneurysm. If these thrombi get dislodged, thromboembolic events, including stroke, may cause significant clinical consequences. In general, a fresh thrombus is less stable and more prone to dislodge to cause embolic events compared with chronic thrombus. It is important to distinguish these two types of thrombus given the higher likelihood of thrombus distribution during VT RF ablation in presence of fresh thrombus. The echographic characteristics of a fresh thrombus include echo-lucency, high mobility and protrusion into the center of the ventricular cavity, while a chronic thrombus is usually higher echogenic, laminar with a smooth border and less mobile. Echo contrast may be used to improve the sensitivity and specificity of LV thrombus detection by revealing a filling defect caused by the thrombus. Peichl *et al.* demonstrate that ICE seems to be more sensitive for the detection of LV thrombi compared to TTE and is helpful in real-time navigation of mapping/ablation catheter. Besides the potential thromboembolic risk, large thrombus may prevent accessibility to the "critical" portion of arrhythmia circuit and epicardial ablation might be required in selected cases.[67]

Case reports and case series have been published on VT RF ablation in the presence of LV thrombus.[67-70] In addition, transcoronary ethanol ablation of VT has been reported, instead of RF ablation, as a salvage technique in the presence of LV thrombus.[71] Based on these limited clinical experiences, Rao *et al.* conclude that ablation of VT in the presence of intracavitary thrombus is feasible, is associated with a similar success rate to historical studies in patients without a thrombus and has an acceptable risk of complications given the high-risk nature of patients with electrical storm.[70]

Our Institute Experience of High-Risk VT Ablation

In our institute, we employ a multispecialty approach, involving cardiac imaging, interventional cardiology, heart failure/heart transplant team, cardiac anesthesia and electrophysiology, specifically on patients with high risk of hemodynamic decompensation. Comprehensive diagnostic tests are performed prior to the procedure to determine the nature of the cardiomyopathy (ischemic, non-ischemic, or infiltrative disease). Patients with high risks will undergo cardiac anesthesia evaluation.

Cardiac anesthesiologist and two electrophysiologists are present for the procedure.

While considering PAAINESD score proposed by Santangeli *et al.* we use straight forward criteria in deciding on pLVADs support. These indications include left ventricular ejection fraction less than 40%, NYHA class III to IV heart failure and unstable clinical VT associated with severely hypotension and syncope.

In our experience Impella seems to provide adequate hemodynamic support compared with IABP and is easier to insert with less bleeding complications compared with ECMO. The goal of mean blood pressure is determined by the patient's preoperative vital signs and evidence of end organ perfusion. Cerebral oximetry based on Near-infrared spectroscopy (NIRS) is used during the VT ablation to monitor brain perfusion.

Usually Impella is placed via the left femoral artery after confirmation of the absence of significant vascular calcification by CT scan as part of routine preprocedural workup. The right femoral vein is reserved for other access during VT ablation. In addition, aortic valve function is assessed carefully to rule out severe aortic stenosis (AS) and aortic regurgitation (AR), which are contraindicated for Impella placement. We prefer trans-septal access to LV for ablation to avoid simultaneous multiple retrograde accesses via aortic valve.

During the procedure, we usually maintain the activated clotting time (ACT) higher than 300 seconds with Impella, although the HRS/ EHRA VT consensus report recommends keeping ACT higher than 250 secs during VT ablation in LV. We note that lower ACT target might lead to higher likelihood of thrombus in Impella circuit, while an ACT target much higher than 300 secs increase the bleeding risks.

During VT induction phase, only relative low flow Impella support is delivered as the patients are clinically relatively stable. Moreover, we noted a more difficult inducibility of VT with maximum Impella function, conceivably due to LV pressure decompression. The Impella flow is increased to delivered full hemodynamic support as soon as sustained VT is induced and sustained – naturally the most critical part of the procedure requiring hemodynamic support.

The pLVADs might be kept in place overnight after VT ablation if clinically appropriate, especially in patients with very low EF, with extensive ablation leading to myocardial stunning, hence allowing adequate mechanical recovery. We are certainly meticulously monitoring the anticoagulation status in the indwelling Impella devices post ablation balancing the risk of thromboembolic events and the risk of bleeding with anticoagulation.

Fluid management is difficult in hemodynamically intolerant VT ablation, because most of these patients have reduced ejection fraction, inferior response to diuretics and thus are prone to develop fluid overload and/ or acute kidney injury secondary to acute tubular necrosis. We perform a meticulous intake/output tracking, monitor urine color changes and choose ablation catheters with minimal flow in order to avoid fluid overload. Moreover, aggressive diuresis might be used at the end of VT ablation if clinically appropriate. It is our policy that dialysis patients should not have an ablation after a weekend on dialysis break as fluid overload is more pronounced at that time.

In our institute, we try to avoid VT ablation in patient with fresh LV thrombus unless no other therapeutic options exist. When the ablation is needed, a thorough evaluation is performed. The most important screening tool is the 12 lead surface ECG – based on the VT morphology one can determine if the VT focus is located away anatomically from the clot location which is usually apical. While capturing the clinical VT on a 12 lead ECG could be difficult, the critical presentation of these patients, usually in incessant VT or VT storm almost always allow capturing enough ECG data. During VT RF ablation procedure, ICE is employed to achieve continuous visual contact of the LV thrombus. As the risk of embolic events is high, we ensure an interventional neuro-radiology team on standby for a possible thrombectomy. It is worthwhile to mention that Embolic protection devices (EPD), usually used in carotid stent procedure, are not a valid option when considering the increased risk of bilateral employment. We present two representative cases selected from our own experience.

A 63-year-old male with past medical history of coronary artery disease status post CABGs, HFrEF with LVEF 10% status post ICD, VT on amiodarone was admitted for syncope with incessant VT requiring anti-tachycardia pacing (ATP) and multiple ICD discharges. The clinical VT was easily inducible during a preliminary EP study, had a cycle length of 350 ms and had an inferior axis suggesting an apical origin. As soon as the VT was induced the arterial pressure dropped to undetectable and the patient was cardioverted.

Subsequently the patient was brought back for a VT ablation employing general anesthesia and hemodynamic support using Impella. Contraindications of Impella, such as severe AR, AS and intracardiac thrombus, were ruled out by echocardiogram. CT scan was performed to rule out severe left femoral artery calcification. A comprehensive map in sinus rhythm using a multipolar pentaray catheter identified areas of scar and fractionated potentials. We subsequently induced VT and mapped the areas of interest using a Biosense Webster DF Curve STSF catheter. While in VT we found the presumed isthmus of the circuit showing concealed entrainment and an equivalent stim to QRS to fractionated potential to QRS. Ablation in that location terminated the arrhythmia almost instantaneously. A second ventricular tachycardia was triggered about 30 mins later by RV pacing. An ablation was performed in the area of the earliest activation, which was on the mid septum between the apex and the mitral annulus. Following this, a third VT was induced. This VT had a cycle length of 420 ms and after extensive mapping and entrainment maneuvers, we were able to document that this ventricular tachycardia was revolving around a large septal scar, between the mitral annulus and apical scar. Entrainments from the lateral wall of the LV showed bystander entrainment, while entrainments from the RV septum showed fusion, however, the morphology of the QRS was very similar to the morphology of the VT. The VT was terminated by connecting the apical scar to the septal scar and subsequently mitral annulus through a line of continuous ablation on the superior part of the LV. As this line was performed, a ventricular tachycardia cycle length slowed gradually and eventually terminated. The patient tolerated the procedure well. Figure 1A shows the presence of Impella device under X ray. Figure 1B demonstrates the line we created to terminate one of the VTs. Figure 1C shows the surface ECG when the VT was terminated. At the end of the procedure we were unable to induce any VT despite aggressive stimulation. The patient was monitored closely in CVICU with Impella and remained intubated overnight due to the concern for myocardial stunning with prolonged VT.

A 68-year-old male with past medical history of CAD status post CABGs, ICM with EF 10 to 15% s/p ICD placement, A fib on warfarin, VT s/p ablation one year prior, known LV apex thrombus (Figure 2B) was admitted for incessant VT with failed AADs therapy of amiodarone, mexiletine and quinidine. Surface ECG indicated the VT originated from the basal LV (Figure 2A) while echocardiography revealed an apical LV clot. After an extensive discussion with the patient of treatment options, VT ablation was performed despite the presence of LV apical thrombus. The LV apex and thrombus was delineated by ICE. LV access was obtained by trans-septal route. We performed a comprehensive mapping of the base and mid cavity of LV, trying to avoid the apex as much as possible. A monomorphic hemodynamically unstable VT with

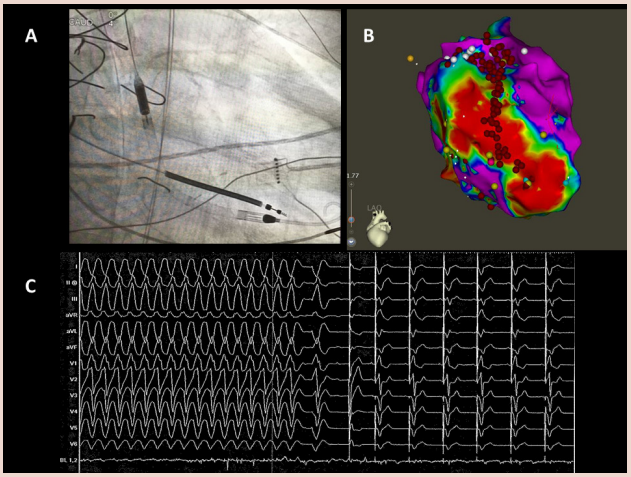

Figure 1: High risk VT ablation with Impella support.
Panel A: Impella under X ray.
Panel B: the continuous ablation line (presented by red dots) which terminated the third VT with cycle length of 420 ms.
Panel C: the Surface ECG when the third VT was terminated.

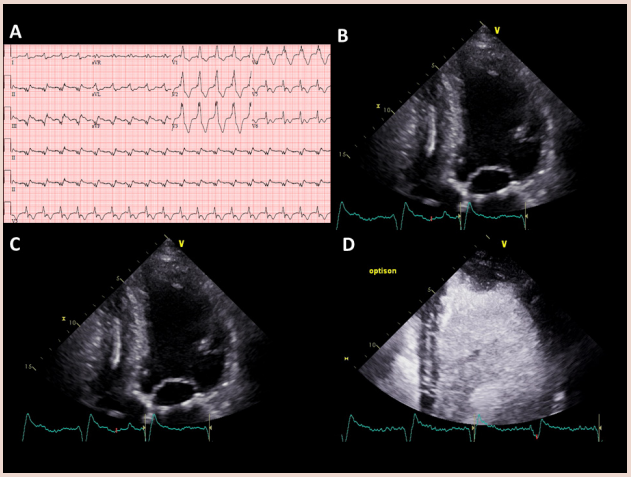

Figure 2: VT ablation with LV apical thrombus.
Panel A: ECG before VT ablation indicating the LV basal origine.
Panel B: Echocardigram three months priot to the VT ablation showing a chronic LV apical thrombus.
Panel C and D: Echocardigram one day after the VT ablation showing no changes of the chronic LV apical thrombus.

morphology similar to the presenting VT was induced and terminated with burst pacing. Using a pace mapping approach, the presumed VT focus was localized and ablated in the infero-septal wall. Additional ablation was performed for scar homogenization. At the end of the procedure, no inducible VT was found. Throughout the case the LV clot was monitored with ICE images, while the next day follow up echocardiogram showed no changes of the LV apical thrombus (Figure 2 C, D).

CONCLUSION

Although hemodynamically unstable VT ablation is usually performed in highly experienced tertiary medical centers, there is a clear trend for more patients being treated outside tertiary institutions, largely due to improved overall mortality in cardiovascular disease leading to increased patient volume and gravity. Clear protocols and safety procedures need to be instituted in centers embarking on high risk VT ablations in order to maintain patient safety and improve outcomes. While complete randomized data may be lacking in some areas, the advancement in technology and increased experience in the EP community will likely allow a consistent progress.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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